Discussion Paper
Breathing pattern disorders and physiotherapy: inspiration for our profession

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Background: Breathing pattern disorders (BPDs), historically known as hyperventilation syndrome, are being increasingly recognized as an entity of their own. Breathing patterns reflect the functioning of the respiratory system and the biomechanical system as well as the cognitive state.

Clinical relevance: It is essential, therefore, that physiotherapists from all areas of specialty consider the assessment and treatment of a patient’s breathing pattern. New literature is emerging which underpins the relevance of BPD in patients with lung disease, anxiety, and also in the comparatively new area of sport performance. Physiotherapists are well placed to treat people with disordered breathing because of their clinical skills and comprehensive knowledge base. Current treatment is briefly reviewed in this paper, and trends for future treatment are also addressed.

Conclusion: The potential for improving the patient’s state, by optimizing their breathing pattern in all their activities, is an important development in physiotherapy. It is a developing area of knowledge which is pertinent to physiotherapy practice as it develops in a biopsychosocial model.

Keywords: Breathing dysfunction, Breathing exercises, Breathing pattern disorders, Breathing retraining, Hyperventilation syndrome

Introduction
Breathing is a central aspect of our whole being and is one of our most vital functions. A disordered breathing pattern can be the first sign that all is not well, whether it be a mechanical, physiological or psychological dysfunction. It is essential, therefore, that breathing is considered in all physiotherapy assessments.

Breathing practices historically span many centuries, philosophies and cultures. Since the turn of the century, Western medicine has been acknowledging the role of the breath in wellbeing,1–3 and more recently research has been critically evaluating the role of the breath in both wellness and illness.4–6 The concept of dysfunctional breathing, or breathing pattern disorders (BPDs) has developed, to describe the presentation of a poor breathing pattern that produces symptoms.7 Defining BPD is an evolving process, and various disciplines are providing unique perspectives which give a multi-dimensional understanding of the multi-faceted function that is breathing.4,8,9 Research is providing new knowledge which underpins the comprehensive role physiotherapy can provide in optimizing the breathing pattern, reducing/eliminating symptoms and facilitating wellbeing.10–12 To date the physiotherapy literature on the topic of breathing pattern disorders and breathing re-education is sparse. Breathing pattern disorders are fast becoming recognized within the speciality area of musculoskeletal and sports physiotherapy11 and private practice,13 whilst still having a significant role in the more likely areas of lung disease5,6 and of anxiety.9,14

A Developing Understanding of Breathing Pattern Disorders
The symptoms of BPD first appeared in medical literature in 1871 when DaCosta,1 noted a set of symptoms predominately in American civil war soldiers that were similar to those of heart disease: fatigue upon exertion, palpitations, sweating, chest pain and a disabling shortness of breath. DaCosta’s syndrome became known as Soldier’s Heart (chest pain).15 As early as 1876 the suggestion of a mechanical origin was considered. Surgeon Arthur Davy attributed the symptoms to military drill where ‘over-expanding’ the chest caused dilatation of the heart, and so induced irritability.16

Haldane and Poultons2 produced a paper linking the symptoms to overbreathing. This gained further support when Solely and Shock3 reported that symptoms could be relieved by increasing partial pressure of carbon dioxide (CO2), reinforcing an underlying respiratory disorder as the cause. It was the discovery of the role of hypocapnia in hyperventilation syndrome (HVS), which placed it firmly in the
Breathing pattern disorders and physiotherapy

The term ‘hyperventilation’ was first used by Kerr et al. and has been frequently used since this time, and more recently defined as, ‘breathing in excess of metabolic demands, resulting in hypocapnia’. Although the syndrome was given various names, the term inferred an anxiety state concurrent with cardiovascular and emotional symptoms, hence patients were considered neurotic and their condition not appropriate for serious medical consideration. Gardner questions whether HVS is an appropriate term when it is the underlying cause of the symptoms may occur without hypocapnia, suggesting there are other mechanisms involved. Vickery refers to breathing patterns disorders as long term abnormal respiratory mechanics. Also, BPD is a distinct syndrome, that is, BPDs are not an inevitable result of pathologic changes due to illness/disease. Discussion at an international level as well as a local level has failed to provide a succinct definition which all parties support.

A working definition by Rowley based on the above perspectives, defines BPD as ‘Inappropriate breathing which is persistent enough to cause symptoms, with no apparent organic cause’. Symptoms may not interrupt daily life but may impact on specific tasks, e.g. elite athletes and their performance, singers and voice production, or the child playing Saturday morning sport.

Mechanisms Underlying Breathing Pattern Disorders

The mechanisms underlying disordered breathing involve physiological, psychological and biomechanical components, and these cannot be completely separated. At a physiological level, hyperventilation has been thought to be driven by central and peripheral chemoreceptors, and cortical drive. Physiologically every cell in the body requires oxygen to survive yet the body’s need to rid itself of carbon dioxide is the most important stimulus for breathing in a healthy person. CO₂ is the most potent chemical affecting respiration.

Hyperventilation results in altered (CO₂) levels, and this is most commonly seen as lowered end tidal CO₂ (PET CO₂), or fluctuating CO₂ levels, and a slower return to normal CO₂ levels. The exact mechanism by which CO₂ influences BPD symptoms remains under debate. Research into levels of CO₂ in the HVS/BPD population has produced disparate results, therefore it may be that the effect of hypocapnia appears highly dependent on the individual.

Common understanding is that the resulting respiratory alkalosis creates a state of sympathetic dominance, which invokes a ‘fright-flight’ response throughout the body. This includes heightened psychological and neuronal arousal, which leads to increased muscle tone, parasthesia and altered rate and depth of breathing. Respiratory alkalosis also affects hemoglobin uptake of oxygen (O₂), coronary artery constriction and cerebral blood flow. These changes in physiological, psychological, and neuronal states affect the musculo-skeletal system.

Musculo-skeletal imbalances may exist, as a result or as a pre-existing contributing factor, and this can be seen in areas such as loss of thoracic cage compliance, constant overuse and tension in the accessory respiratory muscles, and dysfunctional postures. These may impede normal movement of the chest wall, and exacerbate poor diaphragmatic descent. The inefficient respiratory pattern and the increased sympathetic drive contribute further to muscle pain and fatigue, as well as psychological traits such as anxiety.

Psychological factors both influence and are influenced by breathing patterns. Ley states breathing should be examined as an independent variable affecting the psychological process. For example Ley calls dyspnoea a ‘harbinger of suffocation’ and believes that it is the fear of the dyspnoea that...
plays a major factor in panic attacks. Anxiety is the commonest factor thought to influence breathing, and it has been noted to cause increased inspiratory flow rate, breathing to become faster and shallower, and/or involve breath holding.\textsuperscript{23,42} Subjects with BPD have been observed to have higher anxiety levels than the normal population.\textsuperscript{43} Tasks involving prolonged or intense concentration have also been shown to alter breathing patterns.\textsuperscript{44}

### Aetiological Factors in Breathing Pattern Disorders

There is an extensive, perhaps exhaustive list of factors thought to trigger disordered breathing. The broad range of triggers is due to both the variable nature of BPD, and the variation in an individual’s response to environmental and psychological factors. Factors that initially cause a BPD may be different from the factors that perpetuate it.\textsuperscript{38} Once a pattern is established, however,\textsuperscript{31,42} the breathing pattern disorder becomes habituated, and thus a disorder of its own.\textsuperscript{19}

Table 1 shows a list produced from a range of sources.\textsuperscript{8,18–21,23,32,38,46–49}

### Common Symptoms of Breathing Pattern Disorders

The symptoms most commonly reported are respiratory. These include dyspnoea, frequent yawning and sighing, unable to get a deep enough breath, and ‘air hunger’.\textsuperscript{50} The irregularity of the breathing pattern is a common feature, and ironically breathing may appear normal at times, which makes diagnosis and observation difficult.\textsuperscript{51} Other common symptoms are dizziness, chest pain, altered vision, feelings of depersonalization and panic attacks, nausea and reflux, general fatigue and difficulty concentrating. A large range of neurological, psychological, gastrointestinal and musculoskeletal changes can occur, and over 30 possible symptoms have been described.\textsuperscript{52} Assessment of BPD needs to consider this range of manifestations.

### Breathing Patterns

Faulty breathing patterns present differently, depending on the individual. Some patients are more inclined to mental distress, fear anxiety and co-existing loss of self-confidence. Others may exhibit musculoskeletal and more physical symptoms such as neck and shoulder problems, chronic pain and fatigue. Many are a combination of both mental and physical factors.\textsuperscript{53} The key focus of this paper is the musculo-skeletal aspect of BPD. Lung disease and anxiety will be covered, but to a lesser degree as these have been covered in previous physiotherapy literature reviews.

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**Breathing patterns and the musculo-skeletal implications**

‘If breathing is not normalized no other movement pattern can be’.\textsuperscript{54,55}

### Respiration and stability

Respiratory mechanics play a key role in both posture and spinal stability. Research by Hodges et al.\textsuperscript{56–58} examines the relationship between trunk stability and low back pain. It supports the vital role the diaphragm plays with respect to trunk stability and locomotor control. The diaphragm has the ability to perform the dual role of respiration and postural stability. When all systems are challenged, however, breathing will remain as the final driving force.\textsuperscript{59}

In other words ‘Breathing always wins’.\textsuperscript{60}

Respiration is integral to movement as well as stability.\textsuperscript{56,57} The diaphragm, transversus abdominus, multifidius and the pelvic floor muscles work in

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**Table 1 Aetiological factors in breathing pattern disorders**

<table>
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<tr>
<th>Biomechanical factors</th>
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<td>Postural maladaptations</td>
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<td>Upper limb movement</td>
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<td>Chronic mouth breathing</td>
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<td>Cultural, for example, ‘tummy in, chest out’, tight waisted clothing</td>
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<td>Congenital</td>
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<td>Overuse, misuse or abuse of musculo-skeletal system</td>
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<td>Abnormal movement patterns</td>
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<td>Braced posture, for example, post-operative</td>
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<td>Occupational, for example, divers, singers, swimmers, dancers, musicians, equestrians</td>
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<th>Physiological/biochemical factors</th>
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<td>Lung disease</td>
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<td>Metabolic disorders</td>
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<td>Allergies – post-nasal drip, rhinitis, sinusitis</td>
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<td>Diet</td>
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<td>Exaggerated response to decreased CO₂</td>
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<td>Drugs, including recreational drugs, caffeine, aspirin, alcohol</td>
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<td>Hormonal, including progesterone</td>
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<td>Exercise</td>
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<td>Speech/laughter</td>
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<td>Chronic low grade fever</td>
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<td>Heat</td>
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<td>Humidity/hot</td>
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<td>Altitude</td>
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<th>Psychological factors</th>
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<td>Anxiety</td>
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<td>Stress</td>
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<td>Panic disorders</td>
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<td>Personality traits, including perfectionist, high achiever, obsessive</td>
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<td>Suppressed emotions, for example anger</td>
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<td>Conditioning/learnt response</td>
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<td>Action projection/anticipation</td>
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<td>History of abuse</td>
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<td>Mental tasks involving sustained concentration</td>
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<td>Sustained boredom</td>
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<td>Pain</td>
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<td>Depression</td>
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<td>Phobic avoidance</td>
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<td>Fear of symptoms/misattribution of symptoms</td>
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unison to establish intra-abdominal pressure. All structures add to stability and allow efficient respiration, movement and continence control. Should there be a deviation away from a normal recruitment pattern, then pressure, ventilation volumes and ultimately work of breathing is affected. Research by O’Sullivan and Falla et al further supports Chaitow’s claims with respect to position/postures and activation of muscle groups.

When considering total body pressure control, the vocal folds and the surrounding musculature control the top of the system, the diaphragm which sits in the middle plays a key role in pressure generation, and the pelvic muscle group support at the base. The primary purpose of the human larynx is to function as an exchange valve, controlling the flow of air in and out of the lungs. This system adds to not only to structural support but also contributes to motility of fluid based systems within the body, i.e. gastrointestinal, lymphatic drainage, arterial and venous circulation. It also creates phonation and voice production. When a system is under load respiration will dominate at the expense of voice and locomotion and postural control.

It is important to consider how these diverse functions are inter-related and can be co-ordinated into physiotherapy treatment regimes, for example, treatment regimes utilizing all systems, breath, movement and voice.

Length–tension relationship
Pressure determines the length–tension relationship. If a BPD is present respiratory accessory muscles shorten, and the diaphragm is unable to return to its optimal resting position, thus potentially contributing to dynamic hyperinflation, causing pressure changes and further compounding the disorder. Not only is accessory muscle load increased, but the muscles are also working from a shortened disadvantaged position. Shortened muscles create less force, hence the muscle length–tension relationship is altered. Patients with neck pain commonly have faulty breathing patterns.

It is advantageous to keep this in mind, musculo-skeletal techniques will not address an altered length tension ratio unless the driving BPD is addressed. It is also important to note that sustained muscular contraction may occlude local vasculature, momentarily impeding blood flow to activated muscle; this can lead to trigger point development in these muscles.

Dynamic hyperinflation
Dynamic hyperinflation can occur due to a phenomenon known as breath stacking. Traditionally thought to occur in asthma, this also occurs significantly during exercise, when incomplete exhalation can result in residual air adding to the volume of the next inhalation with eventual over-inflation of the lungs. Airflow can become limited and the amount of O₂ reaching the alveoli decreases as dead space volume increases. Inefficient ventilation and dyspnoea are the end result. The supporting musculature also work in less than optimal positions.

The concept of addressing dynamic hyperinflation is not new in the physiotherapy literature: this has been identified and clearly addressed regarding the asthma patient. The idea of decreasing the dynamic hyperinflation of the rib cage is based on the assumption that this intervention will decrease the elastic work of breathing and allow the inspiratory muscles to work over a more advantageous part of their length–tension relationship. There are several treatment strategies that aim to reduce chest wall hyperinflation. Similar strategies could be considered when treating dynamic inflation with no organic lung disorder present.

Motor pattern changes
Dynamic hyperinflation can result due to habitual motor patterns; e.g. increased resting tone of the abdominal muscles in particular the oblique muscles at rest. This can have a ‘corset’ effect preventing diaphragm distension, resulting in the breathing pattern changing to one of upper chest (apical); this leads to over use of the respiratory accessory muscles, pectoralis minor tightens lifting the chest apically, their action opposed by the trapezius which work harder. Forward head posture occurs, and temporomandibular joint compression may occur, and potentially mouth breathing. The tension relationship is altered, and consequently the diaphragm cannot return to optimal resting point, so dynamic hyperinflation occurs. At rest the work of breathing has exceeded the normal values. Unbeknown to the fashion conscious or ‘fab ab’ seeker, there is a host of serious physiological and mechanical, as well as psychological changes taking place. This process challenges the deep motor patterns that control trunk stability. The expiratory reserve volume is increased where tidal volume may remain the same but inspiratory reserve volume decreases, suggesting a dynamic hyperinflated pattern. If hypocapnia is present, this can further alter the resting muscle tone and ultimately motor pattern changes via the increased excitability in the nervous system and muscular system.

Sport/the Athlete
Vital capacity and oxygen delivery
Little attention has been paid to the breathing pattern of the athlete until recently. Historically this area of research has been dominated by sports physiologists.
who have focused on ventilation and the delivery of oxygen. Research is now beyond the capacity of ventilation and starting to look at the muscles of respiration and breathing patterns. The fundamental goal of our system is the protection of oxygen delivery to the respiratory muscles, thus ensuring the ability to maintain pulmonary ventilation, proper regulation of arterial blood gases and pH and overall homeostasis.

Harms et al. identified that the work of breathing during maximal exercise resulted in marked changes in locomotor muscle blood flow, cardiac output and

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**Figure 1** Breathing and the musculoskeletal connection (Tania Clifton-Smith).
both whole-body and active limb O$_2$ uptake. They identified the compromised locomotor blood flow was associated with noradrenaline (norepinephrine) suggesting enhanced sympathetic vasoconstriction. This concept has been referred to as blood stealing, a novel idea that literally the muscles of respiration steal O$_2$ rich blood from the lower limbs. Further work by Sheel$^{75}$ and St Croix$^{76}$ provide evidence for the existence of a metaboreflex, with its origin in the respiratory muscles. They believe this reflex can modulate limb perfusion via stimulation of sympathetic nervous system vasoconstrictor neurones.

**Breathing pattern retraining**

Vickery$^{17}$ conducted ground breaking research assessing the effect of breathing pattern retraining on performance in competitive cyclists. Results supported that four weeks of specific breathing pattern retraining enhanced endurance performance and incremental peak power and positively affected breathing pattern and perceived exertion. It appears that our system has the potential to become sensitized in its protective role and fire too early resulting in bronchospasm? Exercise-induced bronchoconstriction has a high prevalence in athletes and in particular elite athletes, predominately affecting endurance athletes, winter athletes and swimmers.$^{17}$ However, exercise-induced bronchoconstriction also occurs in up to 10% of subjects who are not known to be atopic or asthmatic.$^{78}$

**Breathing Pattern Disorders and Lung Disease**

The altered breathing pattern that occurs with acute asthma is similar to the hyperinflated, rapid upper chest, shallow pattern common in BPD, and therefore it appears reasonable that chronic asthma may contribute to a habitual disordered breathing pattern, as well as a habitual poor breathing pattern exacerbating the symptoms of asthma.$^{52,79}$ Thomas et al.$^{80}$ noted an incidence of hyperventilation of 29% in a sample of 219 known asthmatics in their clinic. Martinez-Moragon et al.$^{81}$ similarly observed 36% (n=171/157) of asthmatics at a pulmonary outpatient clinic had a BPD. A higher correlation is seen in studies assessing patients with known hyperventilation. Saisch et al.$^{82}$ noted asthma was certain or probable in 78% (17) of patients attending an emergency department with acute hyperventilation, including asymptomatic asthma. Similarly, Demeter and Cordasco$^{83}$ recorded 80% (38/47) of patients with hyperventilation, at a private pulmonary clinic, also had asthma. More accurate assessment and including mild/asymptomatic asthma is the likely reason the studies retrospectively assessing for asthma show a higher correlation.

**BPD, asthma and exercise**

Exercise is commonly thought to be a trigger for asthma, and whilst it is true for some, for others the anxiety-inducing breathlessness they attribute to asthma may be due to hyperinflation and excessive respiratory effect due to faulty breathing patterns. Kinnula and Sovijarvi$^{84}$ using cycle ergometry, noted consistent hyperventilation in all the female asthmatics, despite no evidence of bronchospasm at one minute after exercise or differences in exercise capacity. The findings are similar to a study by Hammo and Wienburger$^{85}$ which assessed 32 patients diagnosed with exercise-induced asthma, for hyperventilation. Of the 21 patients who experienced asthma symptoms, 11 had no significant decrease in FEV1, but demonstrated the lowest PETCO$_2$, suggesting hyperventilation, rather than asthma, was responsible for their symptoms. Hibbit and Pilsbury$^{86}$ observed their asthmatic subject began hyperventilating prior to exercise, with slightly lowered peak flow (470 L min$^{-1}$ versus 500–660 expected norm). A marked decrease in PCO$_2$ occurred during exercise and following exercise peak flow dropped to 385 L min$^{-1}$, with the subject feeling anxious and distressed. After two months of breathing retraining and increased physical activities, the exercise test was repeated, with the same initial peak flow, but with considerably less PCO$_2$ changes during exercise, no decrease in PEFR afterwards, and no need for treatment.

A Cochrane review by Holloway and Ram$^{87}$ reported a trend for improvement in asthma symptoms after breathing retraining. More consistent improvements related to quality of life markers rather than changes in lung physiology.$^{10,88}$ The authors$^{87}$ conclude that it is the lack of consistent, robust data with a clear description of the retraining method that limits the conclusions that can be made, rather than necessarily the effectiveness of the breathing retraining itself.

People with chronic asthma may also have lower resting PeCO$_2$ making them more vulnerable to the sympathetic arousal hypocapnia can induce – which they will feel as anxiety.$^{82,89}$

**Breathing pattern disorders, anxiety and COPD**

A review by Brenes$^{90}$ indicates a higher rate of anxiety in people with COPD than the general population. Other studies have linked anxiety in this population to negative quality of life status and lower functional status.$^{91,92}$ Supporting this, Livermore et al.$^{93}$ observed a correlation between higher anxiety in COPD patients and lower threshold for perceived dyspnoea when breathing against a set resistance.
increasing the exertion of breathing, compared to perceived dyspnoea in matched subjects with COPD and a normal control group.

For these populations, correcting the breathing pattern to an efficient steady diaphragmatic pattern can help reduce perceived dyspnoea by reducing the inspiratory effort and anxiety, helping clarify symptoms attributable to actual lung disease rather than functional factors.

**Breathing pattern disorders and anxiety**

Anxiety may be driven by negative thoughts, but also by physiology, for example autonomic disregulation, and/or abnormal lung biomechanics causing a sensation of dyspnoea, not related to actual insufficiencies. The factors surrounding anxiety are too complex and interconnected to suggest there can be a simple causal effect. 102

Studies report greater changes in respiratory patterns in subjects reporting high anxiety levels, when completing a stressful task, with marked increases in tidal volume and respiratory rate and decreased inspiratory time with significant drop in FETCO2 in the high trait anxiety group. 95,96 Similar changes are seen with anticipatory anxiety. 97 Conditioned respiratory responses have also been shown to occur prior to starting a computer task. 98,99

In people with a confirmed diagnosis of an anxiety disorder, such as panic disorder or post traumatic stress disorder, there appears to be a loss of homeostasis, in particular regarding persistent hyperarousal of the sympathetic control. 100,101 Interestingly, Blechert et al. 102 noted the changes in firing of vasoconstrictor fibers in panic disorder patients were similar to those in subjects with increased muscle sympathetic outflow induced by inhaled breath hold and obstructive sleep apnoea, again reinforcing the overlap between breathing pattern disorders and a wide range of causes and symptoms.

**Treatment of BPD in Physiotherapy**

**Assessment**

Physiotherapy treatment of BPD begins with assessment. The lack of a definitive assessment tool for BPD does make diagnosis difficult and sometimes it is achieved only by a process of elimination. 103 Assessment includes gaining an accurate clinical history, observation of the person’s breathing and musculo-skeletal status, and ‘hands on’ assessment of breathing and muscle tension. 4,104,105 Assessment tools commonly used include the Nijmegen Questionnaire, breath hold test, peak expiratory flow rate, and pulse oximetry. 4 Spirometry and capnography may be used, depending on the clinic resources. 6 Treatment can then focus on areas of dysfunction identified during assessment.

**Treatment**

The role of breathing exercises in patients with pulmonary disorders was documented as early as 1915. 106,107 By 1919 it was recommended that many medical and surgical patients be given breathing and physical exercise as accessories to medical and surgical treatment. 108

The first literature referring to BPDs and breathing re-education within the physiotherapy profession was in the 1960s in cardiorespiratory physiotherapy. At this time physiotherapists advocated breathing retraining for BPD. 109 The Papworth method of breathing retraining evolved from the collaboration of chest physician Claude Lum and physiotherapists Diana Innocenti and Rosemary Cluff. This focused on education, and a nose/abdominal breathing pattern. 45,105,109 Other key aspects in the physiotherapy literature treatment are education, reassurance, and breathing retraining. 104,110–115

Most physiotherapy treatment protocols appear to have the following basic principles in common. 4,6,105,112,115

1. Education on the pathophysiology of the disorder
2. Self-observation of one’s own breathing pattern
3. Restoration to a basic physiological breathing pattern: relaxed, rhythmical nose-abdominal breathing.
4. Appropriate tidal volume
5. Education of stress and tension in the body
6. Posture
7. Breathing with movement and activity
8. Clothing Awareness
9. Breathing and speech
10. Breathing and nutrition
11. Breathing and sleep
12. Breathing through an acute episode

**Education**

Education is broader than breathing pattern alone. Education includes the effects of abnormal versus diaphragmatic breathing, and reassurance that HVS/BPD symptoms have a physiological basis, and are treatable. Education also involves identifying the factors that initially caused the BPD, and/or may trigger the poor breathing pattern in the future. 4 Lifestyle issues are addressed, such as level of activity, relaxation (both as a technique and as a recreational activity), and sleep. Work issues, such as sustained computer work, extended periods of intense concentration and speech are also addressed, as these areas have been shown to impact on breathing patterns. 116–118

For the public domain, physiotherapists have written and co-written books on hyperventilation/BPD. ‘Asthma and Your Child’ by Thompson 119 highlights many techniques to assist with breathing pattern disorders and asthma treatment. In 1991 Bradley 120 wrote the first patient handbook on the subject of hyperventilation syndrome/breathing pattern disorders. More recently, in collaboration with...
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Breathing retraining

The terms breathing exercises, breathing retraining and breathing pattern training are used interchangeably in the physiotherapy literature. There is variation, even within the physiotherapy discipline, of what parameters of normal breathing are. Cluff states the rate should be 8–12 average sized breaths per minute at rest, with gentle, silent, rhythmical diaphragmatic (tummy) breathing, with little upper chest movement. West reports breathing rate for an adult at rest is 10–14 breaths per minute.

The treatment of BPD is under recognized. Guidelines for the physiotherapy management of the adult, medical, spontaneously breathing patient have been recently published. These guidelines represent an extensive amount of work collating and analyzing research to support current physiotherapeutic management in the area of cardiorespiratory, neuromuscular diseases and musculoskeletal. Breathing pattern disorders were not mentioned during the review, except used in the context as a historical reference when referring to the treatment by physiotherapists in the management of disordered breathing. Breathing retraining was only used in reference to asthma and secondary disordered breathing.

The BradCliff Method looks at breathing dysfunction as an indicator of physiological and mechanical imbalances and psychological stress in the human body. It is structured on current physiotherapeutic management in the area of cardiorespiratory, neuromuscular diseases and musculoskeletal. Breathing pattern disorders were not mentioned during the review, except used in the context as a historical reference when referring to the treatment by physiotherapists in the management of disordered breathing. Breathing retraining was only used in reference to asthma and secondary disordered breathing.

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Research addressing treatment efficacy for BPD

The variability of treatment regimes and poor description of the regime details have made it difficult to gain a cohesive understanding of what the research to date has shown. Despite this variation, the authors report improvements are achieved, suggesting key elements are covered within the treatment programme. The Papworth method has shown favourable outcomes, significantly reducing respiratory symptoms and improving health-related quality of life in a group of patients with asthma.

Other papers from the UK also support breathing reeducation/training within physiotherapy practice. Singh reviewed the literature with respect to physiotherapy treatment and hyperventilation. The review concluded that the definition and diagnosis of hyperventilation is difficult; however, once identified physiotherapy intervention can provide an effective intervention to significantly reduce the symptoms and improve quality of life. The query over diagnosis was the hyperventilation versus breathing pattern disorder debate. It has been shown clearly in studies that breathing retraining has a positive effect on improving symptoms where the subject does not exhibit low levels of CO2—highlighting that not only do we see people with chronic hyperventilation (lowered CO2) but perhaps a bigger group who present with symptoms due to mechanisms directly related to other pathways.

A 2004 Cochrane review of breathing exercises for asthma concluded that, due to the diversity of breathing exercises and outcomes used, it was impossible to draw conclusions from the available evidence.
evidence. Thomas et al. randomized participants into a group to receive the Papworth breathing re-education method or to see an experienced respiratory nurse providing asthma education. There were significant improvements in asthma-related quality of life in both groups after 1 month, but at 6 months a large difference between groups was found, in favour of the breathing retraining group, in asthma quality of life, anxiety and depression, Nijmegen score and a trend for an improvement in asthma control.

Vickery investigated the effect of breathing pattern retraining on 20-km time trial performance and respiratory and metabolic measures in competitive cyclists. The results supported the performance enhancing effect of four weeks of breathing pattern retraining in cyclists. They suggested breathing pattern can be retrained to exhibit a controlled pattern, without a tachypnoeic shift (increased respiratory rate leading potentially to breath stacking and an irregular pattern that may impair alveolar ventilation) during high intensity cycling. Results also showed that respiratory and peripheral perceived effort was diminished. This research could open avenues of practice not yet proven before within the field of sports physiotherapy, emphasizing the importance of breathing patterns and ultimate performance.

**Future trends for physiotherapy treatment**

Currently in western medicine, a fundamental push is to encourage healthy lifestyle skills. Education in one of the most fundamental tools, and yet breathing has not been emphasized enough as part of this healthy lifestyle package.

Looking to the future the consensus of health in the twenty-first century in the public domain, there appears to be a move away from the twentieth century biomedical model to a more global initiative, promoting projects and programmes that reach all human beings in a worldwide commitment to health as a global public good. Keeping this in mind, there is a push from within our professions to run with this idea of ‘health for all’ and in particular involvement in the management, rehabilitation education and prevention of the epidemic of lifestyles diseases we are currently seeing, such as obesity, ischemic heart disease, cancer, smoking related conditions and pulmonary conditions. There is scope within this framework to explore the concepts of breathing re-education within the profession. Breathing re-education is drug free, appealing to the new paradigm of health for all, and a practice that requires little or no machinery so a low running cost, and initial set-up is minimal for the therapist.

**Conclusions**

For the clinician the observation of breathing can provide insight into many systems, including biomechanics, biochemistry/physiology, and psychology reflecting the consideration of a multisystem approach.

Everyone is a complex integration of musculo/neurological/respiratory systems, which combined with individual personalities and lifestyles, reminds us that these are never distinct groups, and everyone we meet or treat works best when all systems are in homeostasis as supported by an appropriate and efficient breathing pattern.

There is a lack of robust evidence surrounding breathing pattern disorders. Ongoing research is needed that clearly describes treatment regimes and assesses outcomes that are compatible with other research and remains clinically relevant.

As a profession our diversity is an asset. The key points of breathing pattern disorders are common to whomever we treat. Our expertise is in our unique assessment and treatment skills, which enable us to develop specific programmes relevant to the individual cases whether it is the child with asthma or the elite athlete. The diversity of our profession enables us to approach breathing pattern disorders from different perspectives, yet allows us a cohesive informed approach, as physiotherapy aims to treat the whole person not just the system.

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