REVIEW
ASTHMA

Breathing training for dysfunctional breathing in asthma: taking a multidimensional approach

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ABSTRACT Various breathing training programmes may be helpful for adults with asthma. The main therapeutic aim for many of these programmes is the correction of dysfunctional breathing. Dysfunctional breathing can be viewed practically as a multidimensional entity with the three key dimensions being biochemical, biomechanical and psychophysiological. The objectives of this review are to explore how each of these dimensions might impact on asthma sufferers, to review how various breathing therapy protocols target these dimensions and to determine if there is evidence suggesting how breathing therapy protocols might be optimised.

Databases and reference lists of articles were searched for peer-reviewed English language studies that discussed asthma or dysfunctional breathing and various breathing therapies.

Biochemical, biomechanical and psychophysiological aspects of dysfunctional breathing can all potentially impact on asthma symptoms and breathing control. There is significant variation in breathing training protocols and the extent to which they evaluate and improve function in these three dimensions.

The various dimensions of dysfunctional breathing may be of greater or lesser importance in different cases and the effectiveness of breathing training protocols is likely to be improved when all three dimensions are considered. Outcomes for breathing training for dysfunctional breathing in asthma may be most successful when the three key dimensions of dysfunctional breathing are evaluated at the start of treatment and monitored during treatment. This allows breathing training protocols to be adjusted as appropriate to ensure that treatment is sufficiently comprehensive and intensive to produce measurable improvements where necessary.

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Introduction

Research into breathing therapy for asthma shows that a range of breathing retraining programmes may be helpful, but that the diversity of breathing therapies and insufficiency of research assessing ideal protocols and patient selection make it difficult to determine how breathing therapy can best be applied clinically for optimal patient outcomes [1, 2].

Dysfunctional breathing in asthma: significance and prevalence

Dysfunctional breathing can complicate asthma treatment because it leads to disproportionate dyspnoea and medically unexplained symptoms that do not respond to standard asthma medication [3, 4]. Patients with asthma or asthma-like symptoms who also have dysfunctional breathing are reported to have significantly lower quality of life, greater anxiety, lower sense of coherence and decreased asthma control [5, 6].

Breathing training may be most suitable for asthma sufferers who also have dysfunctional breathing. Prevalence of dysfunctional breathing in asthmatic subjects is reported as ranging from 29% to 64%, with a higher incidence in patients with difficult-to-treat asthma and poor asthma control [7, 8]. The presence of dysfunctional breathing is identified in various ways, including through high scores on the Nijmegen Questionnaire [7, 9], by presence of hypocapnia at rest [9], from clinical observation of unusual breathing patterns with disproportionate breathlessness [10] and through observing breathing patterns during exercise challenge [8]. These methods of identifying dysfunctional breathing all have limitations. The Nijmegen Questionnaire, which contains items related to dyspnoea, has not been validated for use in asthma patients. It should also be noted that hypocapnia during acute asthma cannot be assumed to be indicative of dysfunctional breathing as it can be a normal response to acute airway obstruction.

Definitions of dysfunctional breathing

While dysfunctional breathing can be an important consideration in the treatment of asthma, there has been a lack of consensus on its definition. The use of the term "dysfunctional breathing" only appeared in the literature after the 1990s and seems to have come about in response to controversy about hyperventilation syndrome (HVS). This controversy was largely due to research showing inconsistent relationships between hypocapnia and non-neurovascular symptoms in patients who had been diagnosed with HVS [11]. Several recent discussions of dysfunctional breathing propose that hyperventilation and the biochemical dimension comprise only one aspect of breathing dysfunction, and that biomechanical and psychophysiological dimensions of dysfunctional breathing also play a role in symptom production and perpetuation of abnormal breathing behaviours [3, 5, 6, 12–15].

Multiple dimensions of dysfunctional breathing in assessment and treatment

There is no consensus regarding assessment of dysfunctional breathing or indeed its subsequent treatment. This is not surprising given its multidimensional nature and the insufficiency of research clarifying the relative importance of targeting biochemical, biomechanical and psychophysiological dimensions for treatment success.

The biochemical dimension refers to hyperventilation, the biomechanical dimension refers to breathing pattern disorders, and the psychophysiological dimension refers to interactions of physiology with cognitive and emotional factors. While these three dimensions are often related, they can also be distinct, with some individuals showing signs of dysfunction in one dimension but not in others [16]. It is possible that all three dimensions, either individually or through interaction with each other, may play a role in producing the unexplained symptoms and reduced quality of life associated with dysfunctional breathing.

Aims

Here, the aim was to review literature to explore the following questions. 1) Significance/rationale: How do biochemical, biomechanical and psychophysiological aspects of dysfunctional breathing impact on asthma, contribute to symptom production or perpetuate dysfunctional breathing behaviours? 2) Breathing training protocols: How do different styles of breathing training address biochemical, biomechanical and/or psychophysiological aspects of dysfunctional breathing? Is there evidence that they create measurable change in these dimensions? 3) Ideal protocols: Does the literature suggest how breathing training protocols might be refined to improve clinical outcomes?

Methods

MEDLINE, CINAHL and the Cochrane Database of Systematic Reviews were searched for articles using the search terms ("asthma" or "dysfunctional breathing" or "hyperventilation" or "breathing pattern disorder") AND ("breathing therapy" or "breathing training" or "biofeedback" or "Buteyko" or "yoga" or "relaxation therapy"). A total of 329 articles were found using these search terms for articles written from

1982 to 2017. Peer-reviewed articles, including randomised controlled trials and uncontrolled trials related to the use of breathing techniques as the primary modification for asthma or dysfunctional breathing, as well as articles that seemed to be pertinent to the questions explored in this review were examined (n=60). Additional articles discussing hyperventilation, breathing pattern disorders and psychophysiological aspects of dysfunctional breathing in asthma were sourced from reference lists of articles retrieved through these database searches, the author's personal reference library and discussion with colleagues. Older as well as more recent peer-reviewed articles, including a small number of observational studies as well as randomised controlled trials, are included in this narrative review where they provide relevant background information and context for the reader.

Results and discussion

Biochemical dimension: hyperventilation disorders

Description of the biochemical dimension

Hyperventilation, defined as breathing in excess of metabolic needs with resulting depletion of carbon dioxide (CO_2), can be intermittent or chronic, with lowered chemoreceptor set-point, abnormal breathing control and pH disturbance [17].

Significance and rationale

Hyperventilation is more prevalent in individuals with asthma than in healthy individuals, and clinically significant low arterial carbon dioxide tension (P_{aCO_2}) and respiratory alkalosis may exist in acute and chronic symptomatic asthma [18, 19], as well as in mild asymptomatic asthma [20]. Compared with nonasthmatic subjects, asthmatic subjects are more likely to hyperventilate in response to a range of challenges, including muscle tension, dynamic exercise, increased airway resistance, psychological stress and from conditioned response to an innocuous inhaler [21–23].

Given that hyperventilation is a relatively common occurrence in asthma, it has been proposed that it might be a natural response to increased airway resistance, particularly if the response is transitory [18]. Nevertheless, in susceptible individuals hyperventilation can have a severely destabilising effect on breathing control and biochemical homeostasis [24]. Also during hyperventilation, both hypocapnia and associated hyperpnoea may contribute to bronchospasm and lung pathology [25].

Hyperventilation may not always result in symptoms [20]. However, in symptomatic individuals hyperventilation has been associated with both respiratory and nonrespiratory symptoms as represented by the Nijmegen Questionnaire [26]. Nonrespiratory symptoms of hyperventilation disorders, particularly central and peripheral neurovascular symptoms such as dizziness, numbness and tingling, have the strongest relationship to CO_2 [27]. These neurovascular symptoms of hyperventilation do not tend to respond to asthma medication and asthmatic subjects who complain of these types of symptoms have been found to have lower general perceived health, possibly associated with a poor sense of control [28]. Respiratory symptoms such as inability to take a deep breath, chest tightness and shortness of breath are more likely to be related to psychophysiological and neuromechanical factors than to hypocapnia [11, 16].

Response of the biochemical dimension to breathing training protocols

Many breathing therapies used for the treatment of asthma, including therapeutic capnometry biofeedback, the Buteyko method and physiotherapy-based breathing retraining such as the Papworth method, might explicitly or implicitly target hyperventilation. While it has been proposed that correction of hyperventilation is probably not an important mechanism of breathing retraining [25], some recent research suggests that improvements in CO_2 are important for reduction of asthma symptoms [9, 29]. Indeed, a systematic review exploring comparative effectiveness of breathing exercises for asthma proposed that breathing retraining protocols that targeted hyperventilation were more effective than those that had a more general approach [2]. However, the conclusions of this systematic review need closer appraisal as many of the breathing retraining studies reviewed in this study that targeted hyperventilation had not measured CO_2 levels to verify that hyperventilation had been brought under control.

Breathing retraining protocols for asthmatic subjects that do report on CO_2 tension (P_{CO_2}) measurements before and after breathing retraining demonstrate variable results, and factors such as initial levels of hypocapnia in participants, type of breathing training and CO_2 monitoring might affect outcomes. It cannot be assumed that all breathing retraining protocols raise CO_2 levels. At least three randomised controlled trials, in which CO_2 levels were measured in asthma patients undergoing breathing retraining, did not find any statistically significant changes in resting P_{CO_2} despite all studies reporting improvement in either symptoms or quality of life, or reduced use of medication [30–32]. The first of these studies by BOWLER *et al.* [30] did find a significant reduction in minute ventilation after breathing retraining using the Buteyko method, but surprisingly this was not reflected in changes of mean P_{CO_2} level. In a study by

Holloway and West [31] on the Papworth method, a physiotherapy breathing retaining protocol, patients and controls began with normal CO_2 levels and these did not change in either group over the course of breathing training. Finally, in a randomised controlled trial by Thomas *et al.* [32], utilising a breathing protocol similar to the Papworth method, baseline P_{CO_2} levels were in the zone that would be classified as hypocapnia at the beginning of the research trial (31.9 mmHg) and while levels did increase (33.0 mmHg) in the breathing retraining group, the change was not statistically significantly by the end of the trial.

Several studies in which P_{CO_2} was monitored repeatedly over the course of breathing training were able to demonstrate that progressive increases in this variable were achieved, and that CO_2 correlated with gradual symptom improvement and in some cases also with improved lung function [29, 33, 34]. In a small uncontrolled study of therapeutic capnometry biofeedback, Ritz *et al.* [33] demonstrated reduced symptoms and reduced hyperventilation in a group of eight asthmatic subjects. In this pilot trial the changes in P_{CO_2} showed a statistically significant linear increase across the weeks of home exercises training (F(1,7)=13.19; p=0.008). The trend for increasing CO_2 over the 4 weeks of this training programme correlated well with gradual reduction of asthma symptoms. In a subsequent randomised controlled trial, 120 patients with asthma were randomly assigned to either a therapeutic capnometry biofeedback protocol or controlled slow breathing without capnometry. Both groups improved in 17 out of 21 clinical indices measured. The capnometry biofeedback group showed statistically significant greater increases in P_{CO_2} than those using slow breathing, and this was accompanied by greater reductions in respiratory impedance and respiratory symptoms. Results were sustained at 1- and 6-month follow-up [29]. Another study of physiotherapy-based breathing retraining also showed that increases in CO_2 over the course of breathing training correlated with reduced symptoms scores, improved quality of life and also improved lung function. In this study by Grammatopoulou *et al.* [34], time effect on end-tidal CO_2 was significant (F=61.33; p<0.001).

Without adequate testing of CO_2 levels it is difficult to be certain that breathing training techniques aiming to correct hyperventilation are achieving their goal. It has been reported that an individual's resting CO_2 set-point is difficult to change [35]. Treatment needs to be sufficiently intensive and breathing techniques need to be practised adequately and correctly to ensure P_{CO_2} levels normalise. Breathing techniques such as slow abdominal breathing as taught by several breathing retraining protocols can lead to increased hyperventilation in susceptible individuals, as can the Buteyko method instruction to hypoventilate by reducing breathing volume [36, 37].

Optimising breathing training for the biochemical dimension

Capnometry at the start of therapy would be useful to identify patients with chronic hyperventilation as changes in CO_2 are also more likely to be necessary for positive outcomes from breathing training in those individuals. As CO_2 levels appear to rise more predictably when breathing training includes monitoring of CO_2 it seems advisable to monitor CO_2 over the course of therapy, particularly in those individuals who are hypocapnic at the start of breathing. However, the fact that patients with asthma undergoing breathing retraining can improve without any evidence of normalised CO_2 suggests that other mechanisms, possibly related to biomechanical, neuromuscular or psychophysiological factors, are also involved.

Biomechanical dimension: neuromuscular and breathing pattern dysfunctions

Description of the biomechanical dimension

The biomechanical dimension refers mostly to respiratory muscles, their resting tone, strength, efficiency and coordination as evidenced by breathing patterns. In this instance it also relates to breathing behaviours such as mouth breathing.

Significance of neuromuscular and breathing pattern dysfunctions in asthma

Breathing pattern disorders such as thoracic breathing, paradoxical breathing, excessive sighing and irregular breathing are commonly found in asthma patients as well as in individuals with medically unexplained dyspnoea and hyperventilation. These neuromechanical and biomechanical dysfunctions can also be found in individuals with medically unexplained dyspnoea who are not hyperventilating and have normal end-tidal CO_2 at rest, and it has been proposed that dyspnoea in these patients is a result of neuromuscular factors rather than chemoreceptor response or sensitivity [38].

This seems likely given that sensory feedback from respiratory muscles is an important contributor to quality and extent of dyspnoea [39]. Breath holding time reflects dyspnoea threshold; while it is significantly shorter in individuals with chronic hypocapnia [40], it is strongly affected by biomechanical elements of breathing such as respiratory muscle tone and tension [41].

Hypertonicity of respiratory muscles can contribute to dyspnoea [42], and aggravate the tendency for asthma sufferers to develop hyperinflation of the lungs [43] and abnormal breathing patterns such as thoracic breathing and paradoxical breathing [44]. Increased tone and activity of inspiratory muscles at the end of expiration leads to higher than normal levels of lung inflation. Hyperinflation changes the operating conditions of respiratory muscles, making them shorter and weaker. In the case of the diaphragm, hyperinflation and abnormally elevated resting tone lead to loss of the zone of apposition, reducing the ability of the diaphragm to generate inspiratory force. This results in greater recruitment of accessory muscles of respiration in the neck and upper thoracic region as evidenced by thoracic and asynchronous breathing patterns [44]. Shortened, tense and functionally weak respiratory muscles are less able to respond appropriately to central motor command from respiratory centres, potentially contributing to the disparity between ventilation commanded and ventilation achieved. This type of disparity, sometimes called afferent re-efferent dissociation or neuromechanical uncoupling, is thought to be a key factor in determining the extent and quality of dyspnoea in patients with chronic obstructive pulmonary disease [45].

Mouth breathing has been shown to decrease lung function and in some cases to initiate asthma symptoms [46]. It also increases tendencies to hyperventilation [47], and has negative effects on respiratory biomechanics and posture [48]. Improving nasal function to support nasal breathing is associated with improvements in asthma [49].

Response of the biomechanical dimension to breathing training protocols

Many breathing retraining packages aim to teach patients to breathe using more optimal breathing patterns in daily life and also when experiencing dyspnoea [31, 34, 50, 51]. While detailed descriptions of training techniques are often not given, it can be observed that the quality, intensity and type of training are variable. More comprehensive training of breathing includes attention to minute and tidal volume to ensure these are appropriate, and encourages lower rib cage and abdominal breathing, relaxation, and nasal mode of breathing [31, 52].

Studies of breathing training often report that patients are taught “diaphragmatic breathing”, but do not specify instructional techniques or measure whether diaphragm function has indeed improved. While a systematic review of “diaphragmatic breathing” in asthma concluded that it is beneficial for improving both short- and long-term quality of life [53], it should be noted that incorrectly performed “diaphragmatic breathing”, *e.g.* using excessive relaxation of abdominal muscles with high tidal volumes, can potentially worsen rather than improve biomechanical aspects of breathing functionality [54]. In some cases, particularly in patients with more severe chronic respiratory disease, attempts to perform “diaphragmatic breathing” can actually increase hyperinflation, decrease functional strength of the diaphragm and increase dyspnoea [55]. Conversely, techniques such as the Buteyko method, which teaches patients to voluntarily reduce the volume of breathing by relaxing and tolerating slight “lack of air” sensations, might help by reducing hyperinflation and improving length and force generating capacity of the diaphragm [56]. However, no research studies on the Buteyko method have evaluated breathing muscle function or breathing pattern as outcome measures so this remains speculative.

Hypertonicity and weakness of respiratory muscles might contribute to dysfunctional breathing patterns, neuromechanical uncoupling and increased dyspnoea. Hypertonic respiratory muscles tend to be weaker due to the laws of the length–tension relationship. Strengthening of respiratory muscles can be increased by direct training using devices that provide inspiratory resistance and there is some evidence that this can reduce dyspnoea [57]. However, relaxation of hypertonic respiratory muscles is also important for increasing functional strength of respiratory muscles [54]. Manual therapy that aims to relax respiratory muscles and improve mobility of the rib cage has been found to increase peak flow and improve dyspnoea symptoms associated with a sense of unsatisfied respiration, and might be a useful addition to breathing retraining [58, 59], although one study found that addition of manual therapy did not improve outcomes of breathing training [60].

Most studies do not report on breathing pattern changes after breathing training, making it difficult to compare effectiveness of differing approaches to breathing training. Lack of clinically accessible validated tools could be one of the impediments to monitoring breathing patterns during the course of breathing training. One uncontrolled study of patients with medically unexplained dyspnoea that did evaluate diaphragm function and breathing pattern before and after breathing training used a validated clinical assessment tool called the Manual Assessment of Respiratory Motion [61]. In this study, 62 patients with dysfunctional breathing and medically unexplained dyspnoea were trained in a relaxation and breathing protocol. Four weeks of breathing and relaxation training normalised the breathing pattern and substantially reduced dysfunctional breathing symptoms as measured by the Nijmegen Questionnaire, particularly the subset of Nijmegen Questionnaire dyspnoea items (*i.e.* “short of breath”, “faster and deeper breathing”, “unable to breathe deeply”, “tight chest”). This study found that improved breathing pattern was a necessary condition for reduction of dyspnoea symptoms.

The Nijmegen Questionnaire was at one time used to identify individuals with HVS under the assumption that hyperventilation and hypocapnia were the cause of symptoms [26]. However, it is now recognised that the Nijmegen Questionnaire reflects a more generalised disturbance of breathing functionality related to breathing pattern, anxiety and interoception [12].

Optimising breathing training for the biomechanical dimension

Breathing pattern disorders can exist separate to hyperventilation and can independently contribute to dyspnoea in asthma sufferers. Therefore, it seems important to address this dimension by evaluating the breathing pattern during the course of breathing training. Not all attempts at "diaphragmic breathing" successfully improve the function of the diaphragm, particularly in asthma patients who might have hyperinflation or hypertonic respiratory muscles. Control of breathing volume and relaxation of hypertonic respiratory muscles are likely to be important in these patients. Asthma patients should be taught to minimise mouth breathing and use a predominately nasal route of breathing.

Psychophysiological dimension: cognitive and emotional factors

Description of the psychophysiological dimension

The psychophysiological dimension of dysfunctional breathing refers to the various relationships between mental/emotional factors and breathing physiology, breathing behaviours and dysfunctional breathing symptoms.

Prevalence and significance of respiratory-related psychophysiological disturbance in asthma

Psychophysiological factors, including fear, stress and anxiety, along with the hyperarousal and the increased ventilatory drive that accompanies them, are important causes and contributors to dysfunctional breathing symptoms and behaviours in individuals with and without asthma [40]. Individuals with asthma may be particularly susceptible to negative effects of these psychophysiological factors because of the higher than normal prevalence of anxiety disorders in the asthma population, and the unpleasant and sometimes fear inducing effects of dyspnoea [62].

A complex interplay between cognitive and emotional factors with biochemical and biomechanical aspects of breathing control contributes to and perpetuates dysfunctional breathing behaviours and symptoms. Fearful beliefs about respiratory symptoms and anticipation of not being able to satisfy one's ventilatory needs can play a part in conditioning inappropriate and excessive increases in ventilation and sensitisation to bodily symptoms [63]. In some asthma sufferers, mental recall of previous asthma episodes is sufficient to induce hyperventilation and asthma symptoms [64]. Asthma sufferers who are anxious are also more likely to become conditioned and sensitised to experience a greater number and increased severity of respiratory and nonrespiratory symptoms regardless of the extent of airway obstruction [65].

Asthmatic subjects with dysfunctional breathing have been found to have a decrease in subjective sense of control as measured by the perceived control questionnaire, as well as a decreased sense of coherence [10, 28]. Sense of control and sense of coherence have been extensively studied for their relationships to health. Reduced sense of control has been shown to increase anxiety, fear and emotional distress, and to adversely affect a range of health outcomes [66]. Sense of coherence also predicts health-related quality of life and in asthma lower scores on the sense of coherence scale have been shown to correlate with negative coping patterns [67].

Hyperventilation and breathing pattern disorders might contribute to decreased perceived control and sense of coherence in asthmatic subjects. Asthma patients with classic neurovascular hyperventilation symptoms such as numbness and tingling of extremities have lower scores on perceived control scales [28]. The decrease in sense of control may arise when symptoms are unresponsive or less responsive to asthma medication. Weakness and hypertonicity of respiratory muscles, as found in hyperinflation and breathing pattern disorders, leads to inadequate and inefficient response from respiratory muscles to voluntary and involuntary breathing efforts. This incongruence between ventilation attempted and ventilation achieved is known to contribute to increased dyspnoea, particularly the sense of unsatisfied respiration [68], and in the author's view might conceivably reduce the sense of coherence and sense of control in some patients.

Dysregulation of autonomic nervous system function, a common response to psychological and other types of stress, has been observed in asthma. Autonomic nervous system dysregulation is proposed to alter neural regulation of immune function and explain the paradoxical bronchoconstriction in response to sympathetic nervous system activation observed in asthma sufferers [69, 70].

Addressing the psychophysiological dimension in breathing training protocols

Many breathing training protocols for asthma include training in relaxation and self-regulation of stress and arousal. Research has not clearly established the importance of these strategies. However, it can probably be assumed that reduction of psychologically driven hyperarousal is a necessary factor for normalising hyperventilation and a dysfunctional breathing pattern given that behavioural factors are major influencers of respiratory drive, CO_2 set-point and respiratory symptoms [40]. In the psychosomatic patient with unexplained dyspnoea, breathing training combined with relaxation leads to a measurably significant reduction of dyspnoea and stress-related symptoms [61, 63]. Breathing techniques combined with heart rate variability biofeedback to increase vagal tone and improve regulation of the autonomic nervous system have been found to be effective in reducing asthma symptoms, reducing medication needs and improving lung function [71]. Functional relaxation techniques that combine movement and relaxation with conscious exhalation have also been found to improve respiratory resistance and forced expiratory volume in 1 s [72].

Optimising breathing training for the psychophysiological dimension

Relaxation techniques and mental and emotional self-regulation tools that reduce anxiety and hyperarousal are probably important aspects of breathing retraining. Breathing training might also include instructions or processes that help to reduce a patient's fear of dyspnoea and increase their sense of being in control of their breathing. Increasing a patient's understanding of how respiratory and nonrespiratory symptoms can arise from hyperventilation and hyperinflation could help to decrease the patient's fears and negative cognitions about their dysfunctional breathing symptoms. Exploring sensations of breathlessness and breathing discomfort mindfully while being in a relaxed state could also be useful in this regard, and could improve their sense of coherence and control.

Conclusions

While the mechanisms of breathing training in asthma are unclear, there is evidence that biochemical, biomechanical and psychophysiological aspects of dysfunctional breathing can all potentially impact on asthma symptoms and breathing control. Aggravation of respiratory and nonrespiratory symptoms can occur due to hyperventilation, inefficient and aberrant breathing patterns as well as cognitive and emotional factors.

The various dimensions of dysfunctional breathing may be of greater or lesser importance in different cases and the effectiveness of breathing training protocols may need to address an individual's specific type of breathing dysfunction, *e.g.* hyperventilation, breathing pattern disorder or anxiety and fearful cognitions related to dyspnoea and ability to control breathing.

Outcomes for breathing training for dysfunctional breathing in asthma may be most successful when the three key dimensions of dysfunctional breathing are evaluated at the start of treatment and monitored during treatment. This allows breathing training protocols to be adjusted as necessary to ensure that treatment is sufficiently comprehensive and intensive to produce measurable improvements in specific disruptions found to exist in the biochemical, biomechanical and psychophysiological dimensions of dysfunctional breathing.

References

1 Burgess J, Ekanayake B, Lowe A, *et al.* Systematic review of the effectiveness of breathing retraining in asthma management. *Expert Rev Respir Med* 2011; 5: 789–807.
2 O'Connor E, Patnode CD, Burda BU, *et al.* Breathing Exercises and/or Retraining Techniques in the Treatment of Asthma: Comparative Effectiveness. Rockville, Agency for Healthcare Research and Quality, 2012.
3 Barker N, Everard ML. Getting to grips with 'dysfunctional breathing'. *Paediatr Respir Rev* 2015; 16: 53–61.
4 Porsbjerg C, Menzies-Gow A. Co-morbidities in severe asthma: clinical impact and management. *Respirology* 2017; 22: 651–661.
5 Hagman C, Janson C, Emtner M. A comparison between patients with dysfunctional breathing and patients with asthma. *Clin Respir J* 2008; 2: 86–91.
6 Agache I, Ciobanu C, Paul G, *et al.* Dysfunctional breathing phenotype in adults with asthma – incidence and risk factors. *Clin Transl Allergy* 2012; 2: 18.
7 Thomas M, McKinley RK, Freeman E, *et al.* Prevalence of dysfunctional breathing in patients treated for asthma in primary care: cross sectional survey. *BMJ* 2001; 322: 1098–1100.
8 Stanton AE, Vaughn P, Carter R, *et al.* An observational investigation of dysfunctional breathing and breathing control therapy in a problem asthma clinic. *J Asthma* 2008; 45: 758–765.
9 Grammatopoulou EP, Skordilis EK, Georgoudis G, *et al.* Hyperventilation in asthma: a validation study of the Nijmegen Questionnaire – NQ. *J Asthma* 2014; 51: 839–846.
10 Prys-Picard C, Niven RM. Dysfunctional breathing in patients with asthma. *Thorax* 2008; 63: 568.
11 Hornsveld HK, Garsson B. Hyperventilation syndrome: an elegant but scientifically untenable concept. *Neth J Med* 1997; 50: 13–20.
12 van Dixhoorn J, Folgering H. The Nijmegen Questionnaire and dysfunctional breathing. *ERJ Open Res* 2015; 1: 00001-2015.
13 Courtney R. Dysfunctional Breathing: Its Parameters, Measurement and Relevance [PhD Thesis]. Melbourne, RMIT University, 2011.

14 Boulding R, Stacey R, Niven R, *et al.* Dysfunctional breathing: a review of the literature and proposal for classification. *Eur Respir Rev* 2016; 25: 287–294.

15 Courtney R. Multi-dimensional model of dysfunctional breathing and integrative breathing therapy – commentary on the functions of breathing and its dysfunctions and their relationship to breathing therapy. *J Yoga Phys Ther* 2016; 6: 1000257.

16 Courtney R, Greenwood K, Cohen M. Relationships between measures of dysfunctional breathing in a population with concerns about their breathing. *J Bodyw Mov Ther* 2011; 15: 24–34.

17 Jack S, Rossiter HB, Warburton CJ, *et al.* Behavioral influences and physiological indices of ventilatory control in subjects with idiopathic hyperventilation. *Behav Modif* 2003; 27: 637–652.

18 Tobin M, Chadha TS, Jenouri G, *et al.* Breathing patterns 2. Diseased subjects. *Chest* 1983; 84: 286–294.

19 McFadden ER Jr, Lyons HA. Arterial blood gas tension in asthma. *N Engl J Med* 1968; 278: 1027–1032.

20 Osbourne C. Hyperventilation and asymptomatic chronic asthma. *Thorax* 2000; 55: 1016–1022.

21 Kelsen SG, Fleeger B, Altose MD. The respiratory neuromuscular response to hypoxia hypercapnia and obstruction to airflow in asthma. *Am Rev Respir Dis* 1979; 120: 517–527.

22 Grossman P, Wientjes C. Respiratory disorders: asthma and hyperventilation syndrome. *In*: Turpin G, ed. Handbook of Clinical Psychophysiology. Chichester, Wiley, 1989; pp. 519–554.

23 Meuret A, Ritz T. Hyperventilation in panic disorder and asthma: empirical evidence and clinical strategies. *Int J Psychophysiol* 2010; 78: 68–79.

24 Folgering H, Colla P. Some anomalies in the control of $PACO_2$ in patients with a hyperventilation syndrome. *Bull Eur Physiopathol Respir* 1978; 14: 503–512.

25 Bruton A, Holgate ST. Hypocapnia and asthma: a mechanism for breathing retraining? *Chest* 2005; 127: 1808–1818.

26 Dixhoorn J, Duivenvoorden H. Efficacy of the Nijmegen Questionnaire in recognition of the hyperventilation syndrome. *J Psychsom Res* 1985; 29: 199–205.

27 Hornsveld HK, Garssen B, Dop MJ, *et al.* Double-bind placebo-controlled study of the hyperventilation provocation test and the validity of the hyperventilation syndrome. *Lancet* 1996; 348: 154–158.

28 Ritz T, Rosenfield D, Meuret AE, *et al.* Hyperventilation symptoms are linked to a lower perceived health in asthma patients. *Ann Behav Med* 2008; 35: 97–104.

29 Ritz T, Rosenfield D, Steele AM, *et al.* Controlling asthma by training of Capnometry-Assisted Hypoventilation (CATCH) vs slow breathing: a randomized controlled trial. *Chest* 2014; 146: 1237–1247.

30 Bowler SD, Green A, Mitchell A. Buteyko breathing technique in asthma: a blinded randomised controlled trial. *Med J Aust* 1998; 169: 575–578.

31 Holloway E, West RJ. Integrated breathing and relaxation training (Papworth Method) for adults with asthma in primary care: a randomised controlled trial. *Thorax* 2007; 62: 1039–1042.

32 Thomas M, McKinley RK, Mellor S, *et al.* Breathing exercises for asthma: a randomised controlled trial. *Thorax* 2009; 64: 55–61.

33 Ritz T, Meuret AE, Wilhelm FH, *et al.* Changes in pCO_2 symptoms, and lung function of asthma patients during capnometry-assisted breathing training. *Appl Psychophysiol Biofeedback* 2009; 34: 1–6.

34 Grammatopoulou EP, Skordilis EK, Stavrou N, *et al.* The effect of physiotherapy-based breathing retraining on asthma control. *J Asthma* 2011; 48: 593–601.

35 Jack S, Rossiter HB, Pearson MG. Ventilatory responses to inhaled carbon dioxide, hypoxia, and exercise in idiopathic hyperventilation. *Am J Respir Crit Care Med* 2004; 170: 118–125.

36 Meuret AE, Wilhelm FH, Ritz T, *et al.* Breathing training for treating panic disorder. Useful intervention or impediment? *Behav Modif* 2003; 27: 731–754.

37 Conrad A, Müller A, Doberenz S, *et al.* Psychophysiological effects of breathing instructions for stress management. *Appl Psychophysiol Biofeedback* 2007; 32: 89–98.

38 Howell J. Behavioural breathlessness. *Thorax* 1990; 45: 287–292.

39 Banzett RB, Dempsey JA, O'Donnell DE, *et al.* Symptom perception and respiratory sensation in asthma. *Am J Respir Crit Care Med* 2000; 162: 1178–1182.

40 Jack S, Rossiter HB, Warburton CJ, *et al.* Relationship between behavioral influences and ventilatory control mechanisms in patients with idiopathic hyperventilation. *Behav Modif* 2003; 27: 637–652.

41 Parks MJ. The limits of breath holding. *Sci Am* 2012; 306: 74–79.

42 Ritz T, Meuret AE, Bhaskara L, *et al.* Respiratory muscle tension as symptom generator in individuals with high anxiety sensitivity. *Psychosom Med* 2013; 75: 187–195.

43 Muller N, Bryan A, Zamel N. Tonic inspiratory muscle activity as a cause of hyperinflation in asthma. *J Appl Physiol* 1981; 50: 279–282.

44 Decram ML. Hyperinflation and respiratory muscle interaction. *Eur Respir J* 1997; 10: 934–941.

45 O'Donnell DE, Banzett RB, Carrieri-Kohlman V, *et al.* Pathophysiology of dyspnea in chronic obstructive pulmonary disease. *Proc Am Thorac Soc* 2007; 4: 145–168.

46 Hallani M, Wheatley JR, Amis TC. Enforced mouth breathing decreases lung function in mild asthmatics. *Respirology* 2008; 13: 553–558.

47 Tanaka Y, Morikawa T, Honda Y. An assessment of nasal functions in control of breathing. *J Appl Physiol* 1988; 65: 1520–1524.

48 Okuro RT, Morcillo AM, Ribeiro MÃ, *et al.* Mouth breathing and forward head posture: effects on respiratory biomechanics and exercise capacity in children. *J Bras Pneumol* 2011; 37: 471–479.

49 Corren J. Allergic rhinitis and asthma: how important is the link. *J Allergy Clin Immunol* 1997; 99: S781–S786.

50 Hagman C, Janson C, Emtner M. Breathing retraining – a five-year follow-up of patients with dysfunctional breathing. *Respir Med* 2011; 105: 1153–1159.

51 Bruton A, Kirby S, Arden-Close E, *et al.* The BREATHE study: Breathing REtraining for Asthma – Trial of Home Exercises. a protocol summary of a randomised controlled trial. *Prim Care Respir J* 2013; 22: PS1–PS7.

52 Arden-Close E, Teasdale E, Tonkin-Crine S, *et al.* Patients' perceptions of the potential of breathing training for asthma: a qualitative study. *Prim Care Respir J* 2013; 22: 449–453.

53 Prem V, Sahoo RC, Adhikari P. Effect of diaphragmatic breathing exercise on quality of life in subjects with asthma: a systematic review. *Physiother Theory Pract* 2013; 29: 271–277.

54 De Troyer A, Estenne M. Functional anatomy of the respiratory muscles. *Clin Chest Med* 1988; 9: 175–193.
55 Cahalin LP, Braga M, Matsuo Y, *et al.* Efficacy of diaphragmatic breathing in persons with chronic obstructive pulmonary disease: a review of the literature. *J Cardiopulm Rehabil* 2002; 22: 7–21.
56 Courtney R. Strengths, weaknesses and possibilities of the Buteyko method. *Biofeedback* 2008; 36: 59–63.
57 Turner LA, Mickleborough TD, McConnell AK, *et al.* Effect of inspiratory muscle training on exercise tolerance in asthmatic individuals. *Med Sci Sports Exerc* 2011; 43: 2031–2038.
58 Lowhagen O, Bergqvist P. Physiotherapy in asthma using the new Lotorp method. *Complement Ther Clin Pract* 2014; 20: 276–279.
59 Bockenhauer SE, Julliard KN, Lo KS, *et al.* Quantifiable effects of osteopathic manipulative techniques on patients with chronic asthma. *J Am Osteopath Assoc* 2002; 102: 371–375.
60 Jones M, Troup F, Nugus J, *et al.* Does manual therapy provide additional benefit to breathing retraining in the management of dysfunctional breathing? A randomised controlled trial. *Disabil Rehabil* 2015; 37: 763–770.
61 Courtney R, van Dixhoorn J, Greenwood KM, *et al.* Medically unexplained dyspnea: partly moderated by dysfunctional (thoracic dominant) breathing pattern. *J Asthma* 2011; 48: 259–165.
62 Ritz T, Meuret AE, Trueba AF. Psychosocial factors and behavioral medicine interventions in asthma. *J Consult Clin Psychol* 2013; 81: 231–250.
63 Han JN, Zhu YJ, Li SW, *et al.* Medically unexplained dyspnea, psychological characteristics and role of breathing therapy. *Chin Med J* 2004; 117: 6–13.
64 Clark PS, Gibson JR. Asthma, hyperventilation and emotion. *Aust Fam Physician* 1980; 9: 715–719.
65 Chetta A, Foresi A, Marangio E, *et al.* Psychological implications of respiratory health and disease. *Respiration* 2005; 72: 210–2015.
66 Seeman M, Seeman TE. Health behavior and personal autonomy: a longitudinal study of the sense of control in illness. *J Health Soc Behav* 1983: 144–160.
67 Eriksson M, Lindstrom B. Antonovsky's sense of coherence scale and its relation with quality of life: a systematic review. *J Epidemiol Community Health* 2007; 61: 938–944.
68 Lougheed MD. Variability in asthma: symptom perception, care and outcomes. *Can J Physiol Pharmacol* 2007; 85: 149–154.
69 Wright RJ. Stress-related programming of autonomic imbalance: role in allergy and asthma. *Chem Immunol Allergy* 2012; 98: 32–47.
70 Lehrer PM. Emotionally triggered asthma: a review of research literature and some hypotheses for self-regulation therapies. *Appl Psychophysiol Biofeedback* 1998; 23: 13–41.
71 Lehrer PM, Vaschillo E, Vaschillo B, *et al.* Biofeedback treatment for asthma. *Chest* 2004; 126: 352–361.
72 Lahmann C, Nickel M, Schuster T, *et al.* Functional relaxation and guided imagery as complementary therapy in asthma: a randomized controlled clinical trial. *Psychother Psychosom* 2009; 78: 233–239.