



Master class

The functions of breathing and its dysfunctions and their relationship to breathing therapy

Rosalba Courtney*

RMIT University, School Health Science, 11 Binburra Ave, Avalon, N.S.W 2107, Australia

ARTICLE INFO

Article history:

Received 8 April 2009

Accepted 29 April 2009

Keywords:

Dysfunctional breathing

Functions of breathing

Breathing therapy

Hyperventilation

ABSTRACT

Breathing is unquestionably a key function of the human body; it sustains life by providing oxygen needed for metabolism and removing the by-product of these reactions, carbon dioxide. Breathing, however, has other functions apart from the ventilation of air and the maintenance of oxygen and carbon dioxide. Breathing affects motor control and postural stability and plays several roles in physiological and psychological regulation. Breathing can influence homeostatic functions in other system including the autonomic nervous system, the circulatory system, chemical regulation and metabolism.

Breathing becomes dysfunctional when the person is unable to breathe efficiently or when breathing is inappropriate, unhelpful or inefficient in responding to environmental conditions and the changing needs of the individual.

Impairment of the functions of breathing affects people's lives, challenging homeostasis, creating symptoms and compromising health. The efficiency with which breathing fulfills its various functions can be diminished because of musculo-skeletal dysfunction, disease, chronic psychological stress or other factors that affect respiratory drive and respiratory control. The neurological control of breathing shows high levels of neuroplasticity as shown by its ability to adapt to a wide range of internal and external conditions.

Breathing therapy generally aims to either correct dysfunctions of breathing or enhance its functions. Breathing, unlike most physiological functions, can be controlled voluntarily and it can serve as an entry point for physiological and psychological regulation.

© 2009 Elsevier Ltd. All rights reserved.

1. The functions and dysfunctions of breathing

There is a developing interest in impact of dysfunctional breathing in common conditions such as asthma, chronic back and neck pain, postural stability, cardiovascular disease, anxiety and depression. Also breathing therapies are being increasingly used as components of treatment strategies for these conditions. Osteopaths have long recognized that breathing is a commonly disturbed function in the body, which if not addressed has far reaching effects on structure and function.¹ Others have also argued that while dysfunctional breathing (DB) is common, it is often overlooked and when untreated results in unnecessary suffering.^{2–7} The prevalence rate of DB in the general population has been suggested to be as high as 5–11% in the general population^{2,8,9} around 30% in asthmatics¹⁰ and up to 83% in anxiety sufferers.¹¹

Understanding the true prevalence of dysfunctional breathing (DB) and its impact on health is difficult because the parameters

of DB are not clearly defined. Clinicians and breathing therapists argue about the perimeters of DB and its definition and the correct approaches to use in its clinical assessment. A practical approach to DB that has heuristic value is to define it as breathing which is unable to perform its various functions efficiently and is inappropriate for the needs of the individual at that time.

To evaluate the significance of breathing dysfunctions in health and to develop and refine the use of breathing therapy require further understanding of the functions and dysfunctions of breathing. This article explores some of the key functions of breathing and discusses the multi faceted nature of breathing dysfunctions and some of their consequences on mental and physical processes.

1.1. The respiratory pump and the movement of air

The 21,000 breaths per day taken by the average person come about as the breathing muscles attached to the chest wall act to change its shape. As dimensions and form of the chest wall are

* Tel.: +61 2 99183460; fax: +61 2 99187489.

E-mail address: courtney2107@optusnet.com.au

altered, resulting changes in pressure within the intrapleural and alveolar spaces drive the movement of air.¹²

In its normal relaxed state the 2 compartments of the chest wall, the rib cage or thorax and the abdomen, create an effective respiratory pump which moves in response to co-ordinated actions created by the diaphragm, scalenes, intercostals, abdominals and accessory muscles of respiration.^{13,14} However under particular circumstances this co-ordinated action is lost.

This pump becomes impaired and or distorted in various diseases such as kyphoscoliosis, neuromuscular diseases, obesity, emphysema and asthma.¹⁴ Its function can also change in response to psychological stress.^{15–17} The changes in breathing pattern that occur in response to psychological states or various disease states may be appropriate responses to increased ventilatory or metabolic needs or helpful compensations for pathology. Inappropriate habits of muscle use may however be retained after the psychological, physiological or environmental conditions that initiated their development have passed. In this case they are dysfunctional and can complicate disease conditions and increase symptoms.

2. Dysfunctions of the biomechanics of breathing

2.1. The diaphragm

The diaphragm is the key to the function of other respiratory muscles and the primary driver of respiration. If the diaphragm is dysfunctional then the other respiratory muscles will change their function, often becoming overloaded. The normal phasic respiratory action of this large domed shaped muscle is to descent and flatten during inhalation, lifting and widening the lower 6 ribs. In most cases this action is accompanied by slight anterior motion of the abdomen. When the diaphragm is functioning normally, forward displacement of the abdomen is accompanied by lateral expansion and elevation of the lower 6 ribs. The extent of contribution from abdominal or lower rib cage motion can vary and still remain functional. The abdomen can displace anteriorly during inhalation or not move at all.¹⁸ Decreased anterior motion of the abdomen can be compensated by more expansion in the lateral rib cage and vice versa enabling the decreases in intra thoracic pressure necessary for inspiratory airflow through a combination of abdominal and lower rib action.¹⁴

2.2. Asynchronous and paradoxical motion between rib cage and abdomen

Typically in the case of a dysfunctional diaphragm the abdominal muscles will alter their pattern of respiratory activity.^{14,18,19} Paradoxical or asynchronous motion of abdomen, where the dimensions of the abdomen decrease during inspiration can be a sign of diaphragm dysfunction, weakness or paralysis.^{18,20,21} However paradoxical inward motion of the abdomen during inspiration is not always dysfunctional. In fact inward abdominal motion during inspiration can be a normal and functional response to increased lung volume, physical activity, rapid respiratory maneuvers or standing posture that maintains abdominal pressure and helps the diaphragm to maintain a more ideal length and curvature.^{21,22} During inhalation, paradoxical breathing is clearly dysfunctional when it is not adequately compensated by lateral motion of the rib cage and it is observed that the lower rib cage narrows instead of widening during inspiration.²²

2.3. Upper body muscle dysfunctions

In the case of increased ventilatory demand or when breathing is inefficient the respiratory muscles of the upper rib cage, such as the

scalenes, sternomastoid, upper trapezius and other anterior neck muscles such as hyoid and long colli increase their activity.^{19,23,24} This results in increased vertical motion of the rib cage and elevation of the shoulders during the inspiratory phase of breathing.

A tendency to carry the head forward of the body with anterior rotation of the cranial base is a postural change commonly associated with breathing difficulty. Forward head posture is a well-known response to obstructed breathing and is common in children with chronic nasal allergy and mouth breathing because this head position opens the upper airways. Forward head posture can also indicate the presence of short flat diaphragm and weak abdominals due to the fact that positioning the head in front of the body increases the resting length of diaphragm.^{19,25} This head posture while facilitating breathing has several adverse effects on the biomechanics of the head, neck and jaw and is associated with temporal mandibular joint syndrome, neck pain and headache.¹⁹

Shoulder problems may also result because hypertonic trapezius muscles contract during the initial stages of shoulder movement rather than towards the end.²⁵ This altered pattern of scapulo humeral motion is associated with shoulder pain and rotator cuff dysfunction.

3. Factors affecting efficiency of biomechanics of breathing

3.1. Hyperinflation and lung volumes

End expiratory lung volume is an important influence on the power of the diaphragm affecting its ability to act efficiently on the rib cage. Conditions like COPD, asthma and other conditions associated with increased inspiratory drive and inefficient expiration can lead to trapping of air in the lungs or hyperinflation. When this occurs the diaphragm becomes shorter and loses its curvature, as it is forced to take a lower resting position in the thorax. This shortening of the diaphragm fibers decreases the power and efficiency of the diaphragm due to the laws of length, tension relationships which apply to all contracting muscles.²⁶ It is also associated with loss of the curvature or doming of the diaphragm and a reduction of the zone of apposition. In this case the diaphragm fibers, which are attached to the lower 6 ribs, become orientated transversely rather than vertically. When the diaphragm contracts, it is ineffective in lifting and widen the lower rib cage. Instead there is a tendency for the diaphragm to pull the rib the lower lateral rib cage inward, decreasing the transverse diameter of the lower rib cage during inhalation.^{14,27,28}

The more lung volume increases, the more the zone of apposition is reduced. When the lungs are at 30% of inspiratory capacity the costal diaphragm no longer expands the rib cage, and above 30% inspiratory capacity the costal diaphragm has a rib cage deflating action. At enlarged volumes there is also a tendency for the motion of the abdomen to reverse its timing so that inspiration is accompanied by inward motion of the abdomen and expiration is accompanied by outward motion of the abdomen.²¹

Reducing lung volumes can improve the function of the diaphragm. This is clearly demonstrated in COPD patients who experience reductions in lung volume after undertaking surgery to remove sections of their lungs. Lung reduction surgery has been shown in several studies to improve neuromechanical coupling of the diaphragm and individuals who receive this surgery consistently report increased exercise tolerance and reduced symptoms of dyspnea.^{29–31}

3.2. Abdominal weakness

Abdominal muscle weakness aggravates diaphragm dysfunction.^{1,19} Both tonic and phasic contraction of abdominal muscles

assist the function of the diaphragm during inspiration and expiration and can to some extent compensate for diaphragm dysfunction.^{22,26,32} Contraction of the abdomen during inspiration prevents the diaphragm from shortening excessively during standing posture and during rapid and large volume respiratory maneuvers.^{22,33} During expiration contraction of the abdomen assists diaphragm doming, increasing its length and curvature in preparation for effective contraction during inspiration. People with dysfunctional diaphragms, such as many individuals with COPD, can become more breathless and develop more abnormal breathing if tonic contraction of the abdomen is reduced.³⁴ On the other hand, increasing abdominal tone artificially using abdominal binding can assist these individuals.

3.3. Rib cage stiffness

During hyperinflation the ribs shift from their normal oblique position to a more horizontal position, impairing inspiratory action of rib cage muscles and making the rib cage stiff and difficult to expand.³⁵ Restrictions of the rib cage then further inhibit diaphragm function.³⁶

3.4. Respiratory drive

When respiratory control centres in the brain receive messages from the cortex, limbic system, chemoreceptors or mechanoreceptors that ventilation is inadequate, the respiratory muscles adjust their functions to increase ventilation. If respiration is stimulated for prolonged periods, the diaphragm and accessory muscles of breathing may become chronically hypertonic.^{13,21,37} Typical changes in breathing pattern that reflect increased respiratory drive include, upper chest breathing with decreased lateral expansion of the lower rib cage and tendency to asynchronous and paradoxical breathing.

Disease processes can increase ventilatory needs, stimulate respiratory drive and alter respiratory control often creating characteristic changes in breathing pattern. This is seen in respiratory conditions such as asthma and COPD and in heart disease.^{38–40} Psychological and emotional states also alter respiratory control and respiratory rhythm generation.^{41–44} Subsequently changes in breathing pattern which can be dysfunctional are very common in people with respiratory and cardiovascular disease and under psychological stress.

4. Breathing in postural and motor control

The use of breathing muscles during respiration affects how these muscles are used for non-breathing movement and for postural support. Muscles such as the diaphragm, transverse abdominus and pelvic floor muscles are important for motor control and postural support as well as for breathing. If their function is compromised there is an increased susceptibility to back pain and injury.^{45–48} The respiratory functions of these muscles need to be integrated with their many other functions, such as swallowing, speech, valsalva maneuvers, spinal stabilisation and movement of the trunk and limbs. This need for integration of often unrelated functions places considerable demands on mechanism of motor control.^{49,50}

In situations where respiratory drive is increased such as stress, disease or physical exercise, the ability of the respiratory muscles to perform their postural tasks is reduced. Interestingly the presence of respiratory disease is a stronger predictor for lower back pain than other established risk factors.⁵¹ A study by McGill showed that artificially stimulated respiration led to decreased support of the spine during a load challenge.⁵² Hodges in a subsequent study

showed that the postural functions of the diaphragm were significantly reduced and in some cases abolished when respiration was stimulated.⁵³

Respiratory muscles that are responding to increased respiratory drive due to stress or disease produce different breathing patterns to those seen in normal subjects.^{33,54} The respiratory muscles in these situations can become shortened or hypertonic and subsequently less powerful and less efficient.²⁶ They also lose their normal co-ordinated function and one would expect this to result in increased demands on motor control mechanisms. These factors compromise the ability of respiratory muscles to create the fine tuning adjustment required for postural support. The relationship between dysfunctional breathing and postural stability, motor control and back pain has not been studied extensively however clinical observation supports the notion that patients with poor breathing muscle co-ordination are more prone to chronic back pain and neck pain.^{56,57}

5. Biomechanical influences on hemodynamics and the lymphatic system

A functional respiratory pump creates rhythmic pressure fluctuations between the thorax and the abdomen that are important for the movement of body fluids such as blood and lymph. Normal pressure development during the respiratory cycle is characterized by a decrease in intra thoracic pressure during inhalation and an increase in intra abdominal pressure during expiration.^{58,59} Paradoxical motion of the abdomen and dysfunction of the diaphragm alters normal pressure relationships between the thoracic and abdominal compartment during inspiration and expiration. In paradoxical motion of the abdomen during inspiration, abdominal pressures can decrease rather increase during inspiration.²¹

Osteopaths have long considered that restoration of lymphatic function and treatment of oedema and infection was related to proper function of the diaphragm, rib cage and abdomen.^{1,60,61} Miller, one of the early Osteopathic developers of manual pump techniques for lymphatic drainage, noted that thoracic and abdominal pump techniques were exaggerations of respiratory movements.⁶²

Respiration is one of the important extrinsic influences on lymphatic flow, interacting with intrinsic motility of lymph vessels and organs to either enhance or dampen their activity.⁶³ The pressure differentials created by respiration create fluctuations in central venous pressure which directly affect lymph drainage from the lungs and abdomen^{64,65} and affect transmural pressure in lymphatic vessels which modulate the function of intrinsic pacemakers in the lymphatic system. Recent research also shows that the lymphatics in the diaphragm itself form a specialised system for draining fluid from the peritoneal cavity and returning it to the vascular system.⁶⁶ This suggests that breathing patterns which alter normal respiratory pressure dynamics have detrimental effect on the function of the cardiovascular and lymphatic systems.

6. The role of breathing in physiological regulation

Breathing also affects physiological regulation because of its ability to entrain respiratory oscillations to oscillations in other systems and its role in maintaining homeostasis of oxygen, carbon dioxide and pH.

7. Oxygen, carbon dioxide and pH

Breathing, by exchanging carbon dioxide (CO₂) for oxygen (O₂), controls the fundamental gaseous fuels of life's energy and assists in maintaining optimal conditions for the biochemical aspect of the

internal milieu. Oxygen concentrations are generally well maintained by anyone who does not have severe pathology of the lungs, heart or central nervous system. Functional breathing disorders do not generally affect O₂ however even relatively healthy people can over breathe sufficiently to become depleted in CO₂.

Low CO₂ levels can develop quickly because of the very high solubility of CO₂ (twenty times more soluble than oxygen). Its ease of excretion means that increased ventilation resulting from non-metabolic stimuli, for example stress, anxiety or increase sensations of dyspnea, can result in depletion of CO₂. Breathing in excess of metabolic demands is called hyperventilation. The effects of hyperventilation and carbon dioxide depletion are far reaching and include inhibiting the dissociation of oxygen from hemoglobin in the blood, resulting in low oxygen concentration in tissues. The effects on the brain and the nervous system of hypocapnia are particularly pronounced and include reduced cerebral blood flow and increased neuronal excitability. Regulation of cerebro spinal fluid pressure, intracellular pH and cellular metabolism are impaired. Hypocapnia produces bronchoconstriction in the lungs and vasoconstriction in the blood vessels. Blood pressure, myocardial contractility and cardiac blood flow can also be adversely affected as can pH regulation and electrolyte balance.^{67,68}

8. Hyperventilation and hypocapnia

Given the large number of physiological effects of hypocapnia it is not surprising that until the 1990s scientific literature primarily linked breathing dysfunction with hyperventilation syndrome (HVS). The diagnosis of HVS was generally made on finding an assortment of symptoms believed to be caused by either acute or chronic hypocapnia or the consequences of respiratory alkalosis.^{2,5,70}

8.1. History of hyperventilation syndrome

Since the beginning of the nineteenth century it has been known that symptoms such as numbness, dizziness, muscle hypertonicity and tingling sensations could be brought on by overbreathing⁷¹ and that these symptoms could be attributed to hypocapnia and respiratory alkalosis.⁷² The idea of a hyperventilation syndrome characterized by a larger number of psychological and somatic symptoms that could be related to either acute or chronic hyperventilation began to develop after Kerr reported his findings on the effects of hyperventilation challenge on 35 patients with unexplained symptom. His patients were able to reproduce their symptoms by voluntary and prolonged hyperventilation.³ The name Hyperventilation Syndrome began to be used at around this time and was considered to exist mostly in neurotic patients and to be a relatively rare condition.⁴ The range of symptoms attributed to Hyperventilation Syndrome gradually increased until a large number of symptoms of central and peripheral neurovascular, muscular, respiratory, cardiac, gastrointestinal origin were attributed to this syndrome.⁶ Dr. Claude Lum diagnosed 700 patients with medically unexplained symptoms such as palpitations including chest pain, dizziness, parasthesia, breathlessness, epigastric pain, muscle pain, tremor, tetany, dysphagia, tension and anxiety as suffering from hyperventilation syndrome. The diagnosis seemed to be confirmed when the majority of these patients recovered from most of their symptoms after 7 weeks of intensive breathing training.² Later studies such as those by Han, however, were to show that individuals with the symptoms generally attributed to HVS who received benefit from breathing retraining did not necessarily improve because of changes in their carbon dioxide levels.⁷³

8.2. Doubts about the role of chronic hypocapnia in hyperventilation syndrome

From the late 1980s scientists began to question the role of chronic hypocapnia in HVS. Symptoms which were produced by voluntary hyperventilation were assumed to be elicited by acute hypocapnia, but on closer investigation could not always be consistently linked with chronic carbon dioxide deficit. Howell measured PCO₂ levels in 31 patients with disproportionate breathlessness and other symptoms of hyperventilation and found that they had mostly normal levels of carbon dioxide.⁷⁴ Han in comparing 399 symptomatic hyperventilators with 347 normals found no difference in ETCO₂.⁷⁵ In a major review of hyperventilation Hardonk and Beumer found in their own and other studies that ETCO₂ levels were not significantly different in symptomatic or normal controls when measured in the laboratory. By the mid 1990s Gardener in a review of HVS described the “uncertainty and lack of consensus about the boundaries and even existence of this syndrome”. He reported that patients could have low carbon dioxide but no symptoms, while other people could have relatively normal carbon dioxide levels but still exhibit the symptoms of HVS.⁷⁶

Acute hypocapnia can be induced by the hyperventilation provocation test (HVPT) and the onset of symptoms in HVS patients after performing the HVPT was believed to be due to the acute hypocapnia produced by this voluntary overbreathing.⁷⁷ A debilitating and almost fatal blow to the hyperventilation syndrome came from Hornsveld and Garsen whose research appeared to indicate that acute hypocapnia was not the mechanism of the gold standard of HVS diagnosis, the HVPT. In their study 115 patients believed to have HVS were given the Hyperventilation Provocation Test (HVPT). 74% of their subjects were positive on the test and reported the onset of their symptoms after hyperventilation. However 65% of these responders were also positive on a placebo test, during which CO₂ levels were kept stable through manual titration. A second stage of this study, involving transcutaneous monitoring of CO₂ levels of patients in their daily lives, showed that patients suffering from attacks of the HVS symptoms suffered only a very slight drop in CO₂ levels at the onset of their symptoms and this usually followed rather than preceded the onset of symptoms.⁷⁸ As their study, and others, found that neither chronic or acute deficiency of CO₂ could be experimentally linked to HVS these authors recommended that the term hyperventilation syndrome be discontinued.⁸ In recognition of the fact that causes of breathing related symptoms were unclear and often associated with psychological disturbance, researchers proposed that the term hyperventilation syndrome be replaced with behavioral breathlessness⁷⁴ or unexplained breathing disorder (UBD) or chronic symptomatic hyperventilation.⁷⁹ Subsequently the term hyperventilation syndrome is used infrequently and the terms dysfunctional breathing¹⁰ breathing pattern disorder⁸⁰ have become more common.

However the door on hyperventilation and hypocapnia may have been shut prematurely. Hypocapnia may not be the prime suspect in HVS symptoms but an accomplice whose contribution varies according to individual susceptibility and exact symptoms. Recent studies indicate that carbon dioxide functions as one contributing factor to symptoms and its influence is probably moderated by neurological and other factors that influence symptom perception.⁸¹ Not all symptoms of HVS appear to be equally related to carbon dioxide levels, a greater relationship exists for neurovascular symptoms than for uncomfortable respiratory sensations.⁸²

The physiological effects of hypocapnia have been too well documented to be irrelevant.^{67,68} Modern day researchers need to

interpret the finding of Hornsved et al. while still keeping in mind Haldanes experiments in 1908 which clearly shown that hyperventilation caused central and peripheral neurovascular symptoms of dizziness, sensations of numbness and tingling and tetany in healthy people and the long history of studies on symptomatic individuals which repeatedly showed that voluntary acute hyperventilation caused the recurrence of patients primary and secondary complaints which could not be explained in other ways.⁷⁰ Also worth remembering are the studies which demonstrated that administration of carbon dioxide either through re-breathing or by administration of CO₂ enriched gas mixtures was able to eliminate the symptoms of hyperventilation that either came on spontaneously or were brought on by hyperventilation provocation tests.^{4,5}

Chronic hypocapnia when found can and should be normalised, particularly in symptomatic individuals and individual responses to acute hypocapnia. Clinical experience also suggests that the hyperventilation provocation test may indeed be relevant to diagnosis and treatment if used in conjunction with other investigations.

9. Breathing, homeostasis and oscillations

Breathing, by creating fluctuations in the chemical composition of the blood, circulation and vascular pressure and by its effects on autonomic reflexes acts as an extrinsic influence on other oscillating physiological systems. Oscillations, which are defined as systematic rhythms in physiological variables, are found in most living system including those of the human body. Oscillations in single systems and synchronisation between oscillating systems help physiological control systems to maintain homeostasis and appropriate and rapid responsiveness to the continual changing needs of the body. When oscillations of two or more systems are synchronised it increases physiological efficiency by enabling the functions of these systems to be co-ordinated. This prevents energy being wasted on non productive functions.^{85,86} Cyclic activity also allows systems to rest and renew themselves during cycles of decreased activity.

One aspect of breathing functionality is the ability of breathing oscillations to interact with oscillations in other physiological systems in ways that optimise their functions. Breathing oscillations interact with oscillations of heart rate and blood pressure,^{87,88} the lymphatic system⁶³ the digestive system,^{89,90} brain waves⁹¹ and probably the rhythmic fluctuations occurring in cellular metabolism.⁹² When respiratory oscillations entrain other oscillating systems it can enhance the physiological function of both systems. A well-known example is the phenomenon of respiratory sinus arrhythmia (RSA), where heart rate variability is entrained to respiratory frequencies. RSA improves the efficiency of gas exchange by coupling increased heart rate to the inspiratory phase of respiration.⁹³

9.1. Breathing pattern dysfunctions and resonant frequencies of breathing

Breathing can be consciously manipulated to increase its ability to entrain other oscillations and increase physiological regulation. When breathing frequency is slowed to between 4 and 6 breaths per minute (0.06–0.1 Hz) oscillations in blood pressure, heart rate and autonomic nervous system tend to synchronise at this frequency and be amplified due to resonance effects between these systems.^{94–96} These resonance effects between cardiorespiratory oscillations and autonomic function are important for homeostasis and maintenance of health as evidence by the fact that training the bodies ability to increase them assists people with a range of

conditions including asthma, COPD, depression, hypertension and irritable bowel syndrome.^{97–100} Individuals with paradoxical breathing and thoracic dominant breathing have a decreased ability to achieve resonance effects between oscillators (unpublished data), indicating that dysfunctions of breathing pattern impair the body's ability to regulate itself through co-ordinating homeostatic cardiorespiratory oscillations.

10. Breathing, stress, emotion and the autonomic nervous system

Chronic emotional stress and increased mental load can alter respiratory regulation in several ways: 1) with regard to drive; 2) breathing pattern and timing; and 3) metabolic appropriateness of the respiratory response.⁴²

Breathing is particularly sensitive to states of hyperarousal, during which signs of increased respiratory drive are evidence of the body's readiness for action. Hyperarousal brought on by mental and emotional processes contributes to allostatic load and affects the capacity of the body to maintain its stability and response to change.¹⁰¹ Anticipation of coming physical and emotional events has the distinct effect of increasing respiratory rate, reducing time of exhalation and changing respiratory pattern.^{44,102} Breathing irregularity is a common feature of patients with anxiety and panic disorder, indicating dysregulation of normal breathing control mechanisms in these individuals.^{75,103} Specific effects on the diaphragm are also seen. Fluoroscopic studies show that in situations of emotional stress, the diaphragm shows signs of hypertonicity becoming flattened and immobile.^{15,16}

Breathing which is responding to feelings and thoughts, rather than metabolic cues from chemoreceptors, may not be aligned to the actual physical needs of the body or its metabolic requirements.^{44,88} When mental and emotional factors such as fear, grief, anxiety or depression drive breathing regulation, homeostatic and biomechanical functions of breathing can be disturbed.^{42,43} Emotions are drives to particular types of action or expression. Repeatedly frustrated ability to act on strong emotional drives can lead to physiologically and biomechanically inappropriate breathing, tuned to the anticipation of action that does not occur.

A fundamental survival function of increased respiratory drive is to prepare the body for fight and flight. However homeostasis is best served through the functions of the parasympathetic nervous system, whose activity is associated with a relaxed, slow and abdominal breathing pattern.¹⁰⁴ Rapid, shallow and thoracic dominant breathing with high levels of tonic contraction of respiratory muscles as found in situations requiring high levels of ventilation probably indicates a system that is having difficulty returning to a state of rest. In this situation energy is wasted and homeostatic functions needed for repair and renewal are impaired.

Controlled respiration can help the system to return to a physiological rest state. It appears to act on the brain and the autonomic nervous system, synchronising neural elements in the brain and autonomic nervous system and creating a state of psycho-physiological coherence.^{105,106} The changes induced by controlled breathing appear to order and regulate neurological function, improving psychological and emotional states.^{107,108} The regulatory effects of breathing on the autonomic nervous system have been investigated from several perspectives. Several studies have shown that in the short term controlled breathing can decrease sympathetic nervous system activity and increase parasympathetic nervous system and with continued, regular practice can create long term improvements in autonomic balance and increased vagal tone.^{109–113}

11. Breathing as a therapeutic tool

There are a large number of breathing therapies utilizing a wide range of techniques and several of these appear beneficial. The main rationales for breathing therapies are that they: 1) correct some aspect of dysfunctional breathing; 2) support one or several of the functions of breathing and thus stimulate healing; or 3) provide a means for regulation of mental and emotional states. The territory covered by breathing therapies is large covering a broad area ranging over psychology, physiology, spirituality and biomechanics. The evidence for the ability of breathing therapies to correct breathing dysfunctions and support the functions of breathing is relatively sparse as research has tended to focus on disease or psychological outcomes rather than investigating effects on breathing parameters.

There are several breathing therapies that aim to correct hyperventilation and restore normal carbon dioxide tension. Breathing biofeedback using a capnometer to monitor end-tidal carbon dioxide levels during breathing training is one approach to treating hypocapnia. Biofeedback training that employs the use of a capnometer aims at normalizing end-tidal CO₂ at approximately 5%. There are various systems for capnometry biofeedback available to the practitioner and in recent years these have become increasingly available.^{114,115} Other breathing therapists use no instrumentation and rely on a combination of slow controlled breathing, breathing pattern correction and relaxation strategies.^{116,117} The Buteyko Breathing Technique (BBT) is yet another technique whose primary aim is the correction of acute and chronic hypocapnia. It uses a unique set of breathing techniques in which breath holding is combined with reduced volume breathing. BBT exercises aim to increase carbon dioxide and reset chemoreceptor thresholds however they may also be useful in reducing hyperinflation.¹¹⁸

The practice of controlled breathing as a means of self-regulation and restoration of mental and emotional balance is a part of Indian and Taoist Yoga and is also in the domain of modern respiratory psychophysiology. Modulation of the breath and mindful attention to the breath are important parts of many meditation techniques.¹¹⁹ Ancient systems such as Indian Yoga pranayama and the breathing techniques of Qi Gong teach that specific types of breathing exercises can direct vital force or energy and through this benefit the health of the mind and body.^{120–123}

Respiratory psychophysiology uses a scientific approach to understanding the physiological processes that link mental and emotional states to breathing. Practitioners of respiratory psychophysiology utilize evidence based breathing techniques including resonant frequency breathing to promote emotional self regulation and to achieve efficient physiological states that promote healing.⁹⁵

Another interesting breathing therapy used mostly to enhance athletic performance in athletes is Intermittent Hypoxic Training (IHT).¹²⁴ This therapy exposes individuals to carefully controlled levels of hypoxia interspersed with rest periods during which they breathe normal atmospheric air or oxygen enriched air. IHT has the effect of enhancing adaptation to the specific stress of hypoxia but also results in a general increase in stress tolerance. Other benefits of IHT include enhanced antioxidant capacity, improved metabolism and increased aerobic capacity. IHT is believed to enhance mitochondrial function as it has been shown to increase the efficiency with which the body uses oxygen. In Russia IHT has been studied extensively for over 30 years and is used to treat a large number of diseases.¹²⁵

There is a growing body of scientific evidence for the effectiveness of breathing therapies in a range of diseases including asthma, heart disease, anxiety and depression, and they are being increasingly included in therapeutic protocols. Research into Buteyko Breathing Techniques has focused on asthma and there have been at

least five published clinical trials on BBT for this condition.^{126–132} These clinical trials indicate that people learning the Buteyko Method are able to substantially reduce medication with no deterioration in their lung function or asthma control, although no studies have demonstrated objective changes in lung function. The quality of evidence of the Buteyko Method according to an Australian Department of Health report is stronger than any other complementary medicine treatment of asthma.¹³³ Recent studies indicate that several other types of breathing therapies also help asthma including resonant frequency breathing biofeedback,⁹⁷ capnometry biofeedback¹³⁴ and breathing rehabilitation.^{116,135}

Breathing therapy has also been found to be very helpful for cardiovascular disease. In a study comparing patients who received standard cardiac rehabilitation with those receiving additional training in breathing therapy after myocardial infarction (MI), it was found that the breathing therapy group had about a 30% decrease in cardiac events at 5 year follow-up.¹³⁶ Another study showed that exercise training in patients with MI was not always successful in preventing future cardiac events, however the risk of treatment failure was reduced by half when relaxation and breathing training was added to exercise training.¹³⁷ Other breathing therapy based on yoga breathing was also found to improve hemodynamics and various cardiorespiratory risk factors in cardiac patients.^{138,139}

The effectiveness of breathing therapies in psychological conditions and chronic stress has also been shown in several studies. In major depression, both resonant frequency biofeedback and yoga based breathing techniques appear to be effective.^{99,139,140} People with anxiety and panic disorder also show beneficial response to capnometry and other breathing therapy protocols.^{73,141,142} The effects of breathing on the autonomic nervous system have been demonstrated in several studies on yoga pranayama. Regular and prolonged practice of several brief breathing protocols combining slow breathing, long breath retentions and nostril breathing have the effect of increasing resetting autonomic balance and amplifying parasympathetic nervous system function.¹⁰⁹

This sampling of the literature on breathing dysfunction and breathing therapies, while not exhaustive, strongly suggests that breathing therapies have the potential to be of benefit to patients with many types of conditions. It appears that use of breathing therapies is not proportional to the level of evidence supporting their efficacy. One reason for this may be the lack of coherent models for explaining the mechanisms of breathing therapies. Understanding of these mechanisms needs to be developed to refine and direct the use of breathing interventions. I propose that there are likely to be a range of mechanisms for breathing therapies and these should be sought within the various functions of breathing and its dysfunctions.

Part two of this paper will elaborate on the role of breathing dysfunction in various diseases and present some diverse approaches to evaluation of various aspects of breathing dysfunction.

Acknowledgements

The Australian Osteopathic Association for financial and administrative support for research associated with this review article.

References

1. McConnell C. The diaphragm. *J Am Osteopath Assoc* 1928;**31**:87–91.
2. Lum LC. Hyperventilation: the tip and the iceberg. *J Psychom Res* 1975;**19**:375–83.
3. Kerr WJ, Dalton JA, Glibe PA. Some physical phenomena associated with the anxiety states and their relation to hyperventilation. *Ann Int Med* 1937;**11**:961.

4. Soley MH, Shock NW. The aetiology of effort syndrome. *Am J Med Sci* 1938;**196**:840.
5. Magarian G. Hyperventilation syndromes: infrequently recognized common expressions of anxiety and stress. *Medicine* 1982;**61**:219–36.
6. Lewis BL. The hyperventilation syndrome. *Ann Int Med* 1953;**38**:918–27.
7. Nixon PGF. The grey area of effort syndrome and hyperventilation. *J R Coll Physicians of Lond* 1993;**27**.
8. Hornsveld HK, Garsson B. Hyperventilation syndrome: an elegant but scientifically untenable concept. *Nether J Med* 1997;**50**:13–20.
9. Fried R. *The hyperventilation syndrome*. Baltimore/London: John Hopkins University Press; 1987.
10. Thomas M, McKinley RK, Freeman E, Foy C. Prevalence of dysfunctional breathing in patients treated for asthma in primary care: cross sectional survey. *BMJ* 2001;**322**:1098–100.
11. Cowley DS. Hyperventilation and panic disorder. *Am J Med* 1987;**83**:923–9.
12. West J. *Respiratory physiology*. 3rd ed. London: Williams and Wilkins; 1985.
13. Konno K, Mead J. Measurement of separate volume changes of rib cage and abdomen during breathing. *J Appl Physiol* 1966;**22**:407–22.
14. De Troyer A, Estenne M. Functional anatomy of the respiratory muscles. *Clin Chest Med* 1988;**9**:175–93.
15. Faulkner WB. The effect of the emotions upon diaphragm function: observations in 5 patients. *Psychosom Med* 1941;**3**:187–9.
16. Wolf S. Diaphragmatic spasm: a neglected cause of dyspnea and chest pain. *Integr Physiol Behav Sci* 1994;**29**:74–6.
17. Masaoka Y, Homma I. Anxiety and respiratory patterns: their relationship during mental stress and physical load. *Int J Psychophysiol* 1997;**27**:153–9.
18. Fitting JW, Grassino AE. Diagnosis of diaphragm dysfunction. *Clin Chest Med* 1987;**8**:91–103.
19. Hruska R. Influences of dysfunctional respiratory mechanics on orofacial pain. *Dent Clin North Am* 1997;**41**:211–27.
20. Jubran A, Tobin M. The effect of hyperinflation on rib cage-abdominal motion. *Am Rev Respiratory Diseases* 1992;**146**:1378–82.
21. Wolfson D, Strohl K, Dimarco A, Altose M. Effects of an increase in end-expiratory volume on the pattern of thoracoabdominal movement. *Respir Physiol* 1983;**53**:273–83.
22. De Troyer A. Mechanical role of the abdominal muscles in relation to posture. *Respir Physiol* 1983;**53**:341–53.
23. Verschakelen JA, Demedts MG. Normal thoracoabdominal motions: influence of sex, age, posture, and breath size. *Am J Respir Crit Care Med* 1995;**151**:399–405.
24. De Troyer A, Estenne M. Coordination between rib cage muscles and diaphragm during quiet breathing in humans. *J Appl Physiol* 1984;**57**:899–906.
25. Pryor JA, Prasad SA. *Physiotherapy for respiratory and cardiac problems*. 3rd ed. Edinburgh: Churchill Livingstone; 2002.
26. Finucane K, Panizza J, Singh B. Efficiency of the normal human diaphragm with hyperinflation. *J Appl Physiol* 2005;**99**:1402–11.
27. Zocchi L, et al. Effect of hyperinflation and equalization of abdominal pressure on diaphragmatic action. *J Appl Physiol* 1987;**62**:1655–64.
28. Celli B. Clinical and physiological evaluation of respiratory muscle function. *Clin Chest Med* 1989;**10**.
29. Laghi F, et al. Effect of lung volume reduction surgery on neuromechanical coupling of the diaphragm. *Am J Respir Crit Care Med* 1998;**157**:475–83.
30. Lahrmann H, et al. Neural drive to the diaphragm after lung volume reduction surgery*10.1378/chest.116.6.1593. *Chest* 1999;**116**:1593–600.
31. Cassart M, et al. Effects of lung volume reduction surgery for emphysema on diaphragm dimensions and configuration. *Am J Respir Crit Care Med* 2001;**163**:1171–5.
32. Cassart M, et al. Effect of chronic hyperinflation on diaphragm length and surface area. *Am J Respir Care Med* 1997;**156**:504–8.
33. Sharp JT, et al. Relative contributions of rib cage and abdomen to breathing in normal subjects. *J Appl Physiol* 1975;**39**:608–18.
34. Cahalin L, 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.
35. Tobin M. Respiratory muscles in disease. *Clin Chest Med* 1988;**9**:263–86.
36. Hussain S, et al. Effects of separate rib cage and abdominal restriction on exercise performance in normal humans. *J Appl Physiol* 1985;**58**:2020–6.
37. Muller J, Bryan A, Zamel N. Tonic inspiratory muscle activity as a cause of hyperinflation in asthma. *J Appl Physiol* 1981;**50**:279–82.
38. Loveridge B, et al. Breathing pattern in patients with chronic obstructive pulmonary disease. *Am Rev Respir Dis* 1984;**130**:730–3.
39. Allen J, et al. Thoracoabdominal asynchrony in infants with airflow obstruction. *Am Rev Respir Dis* 1990;**141**:337–42.
40. Bernardi L, Gabutti A. Slow breathing reduces chemoreflex response to hypoxia and hypercapnia and increases baroreflex sensitivity. *J Hypertens* 2001;**19**:2221–8.
41. Manning H, et al. Reduced tidal volume increases “air hunger” at fixed PCO₂ in ventilated quadriplegics. *Respir Physiol*; 1992::9019–30.
42. Wientjes C. Respiration in psychophysiology: methods and applications. *Biol Psychol* 1992;**34**:179–203.
43. Boiten FA. The effects of emotional behavior on components of the respiratory cycle. *Biol Psychol* 1998;**49**:29–51.
44. Homma I, Masaoka Y. Breathing rhythms and emotions. *Exp Physiol* 2008;**93**:1011–21.
45. Lewit K. Relationship of faulty respiration to posture with clinical implications. *J Am Osteopath Assoc* 1980;**79**: 525/75–529/79.
46. Hodges P, Gandevia SC, Richardson CA. Contractions of specific abdominal muscles in postural tasks are affected by respiratory maneuvers. *J Appl Physiol* 1997;**83**:753–60.
47. Hodges P, Cresswell A, Thorstensson A. Preparatory trunk motion accompanies rapid upper limb movement. *Exp Brain Res* 1999;**124**:69–79.
48. Hungerford B, Gilleard W, Hodges P. Evidence of altered lumbopelvic muscle recruitment in the presence of sacroiliac joint pain. *Spine* 2003;**28**:1593–600.
49. Gandevia SC, et al. Balancing acts: respiratory sensations, motor control and human posture. *Clin Exp Pharmacol Physiol* 2002;**29**:118–21.
50. Rassel B, Kohl J. Coordination-related changes in the rhythms of breathing and walking in humans. *Eur J Appl Physiol* 2000;**82**:280–8.
51. Smith MD, Russell A, Hodges PW. Disorders of breathing and continence have a stronger association with back pain than obesity and physical activity. *Aust J Physiother* 2006;**52**:11–6.
52. McGill S, Sharratt M, Sequin J. Loads on spinal tissues during simultaneous lifting and ventilatory challenge. *Ergonomics* 1995;**38**:1772–92.
53. Hodges P, Heijnen I, Gandevia SC. Postural activity of the diaphragm is reduced in humans when respiratory demand increases. *J Physiol* 2001;**537**:999–1008.
54. Tobin M, et al. Breathing patterns 2. Diseased subjects. *Chest* 1983;**84**:286–94.
56. Perri MA, Halford E. Pain and faulty breathing: a pilot study. *J Bodyw Mov Ther* 2004;**8**:297–306.
57. Chaitow L. Breathing pattern disorders, motor control and low back pain. *J Osteopath Med* 2004;**7**:33–40.
58. Dornhorst AC, Howard P, Leathart GL. Respiratory variations in blood pressure. *Circulation* 1952;**6**:553–8.
59. DeBoer RW, Karemaker JM, Strakee J. Hemodynamic fluctuations and baroreflex sensitivity in humans: a beat to beat model. *Am J Physiol Heart Circ Physiol* 1987;**253**:680–9.
60. Degenhardt B, Kuchera M. Update on osteopathic medical concepts and the lymphatic system. *J Am Osteopath Assoc* 1996;**96**:97–100.
61. Chikly B. Manual techniques addressing the lymphatic system: origins and development. *J Am Osteopath Assoc* 2005;**105**:457–64.
62. Miller C. The lymphatic pump, its applications to acute infections. *J Am Osteopath Assoc* 1926;**25**:443–5.
63. Gashev A. Physiological aspects of lymphatic contractile functions: current perspectives. *Ann N Y Acad Sci* 2002;**979**:178–87.
64. Hedenstierna G, Lattuada M. Lymphatics and lymph in acute lung injury. *Curr Opin Crit Care* 2008;**14**:31–6.
65. Lattuada M, Hedenstierna G. Abdominal lymph flow in an endotoxin sepsis model: influence of spontaneous breathing and mechanical ventilation. *Crit Care Med* 2006;**34**:2788–92.
66. Abu-Hijleh M, Habbal A, Mogattash S. The role of the diaphragm in lymphatic absorption from the peritoneal cavity. *J Anat* 1995;**186**:453–67.
67. Lumb A. Changes in carbon dioxide tension, *Nunns applied respiratory physiology*. Oxford/Auckland/Boston/Johannesburg/Melbourne/New Delhi: Butterworth Heinemann; 2000 [chapter 22].
68. Laffey JG, Kavanagh BP. Hypocapnia. *N Engl J Med* 2002;**347**:43–54.
70. Hardonk HJ, Beumer HM. Hyperventilation syndrome. In: Vinken PJ, Bruyn GW, editors. *Handbook of clinical neurology: neurological manifestations of systemic disease*. Amsterdam: North Holland Biomedical Press; 1979.
71. Haldane JS, Poulton EP. The effects of want of oxygen on respiration. *J Physiol* 1908;**37**:390–407.
72. Vernon HM. The production of prolonged apnea in man. *J Physiol* 1909;**38**:18.
73. Han JN, Stegen K, De Valack C, Clement J, Van de Woestjine KP. Influence of breathing therapy on complaints, anxiety and breathing pattern in patients with hyperventilation syndrome and anxiety disorders. *J Psychom Res* 1995;**41**:481–93.
74. Howell J. Behavioural breathlessness. *Thorax* 1990;**45**:287–92.
75. Han J, et al. Unsteadiness of breathing in patients with hyperventilation syndrome & anxiety disorders. *Euro Respir J* 1996;**10**:167–76.
76. Gardner W. The pathophysiology of hyperventilation disorders. *Chest* 1995;**109**:516–33.
77. Vansteenkiste J, Rochette F, Demedts M. Diagnostic tests of hyperventilation syndrome. *Euro Respir J* 1991;**4**:393–9.
78. Hornsveld HK, Garsson B, Fiedeldij Dop MJC, Van Spiegel PI, De Haes J. Double-blind placebo-controlled study of the hyperventilation provocation test and the validity of the hyperventilation syndrome. *Lancet* 1996;**348**:154–8.
79. Bass C. The hyperventilation syndrome. *Respir Dis Pract* 1989;**41**:13–6.
80. Chaitow L, Bradley D, Gilbert C. *Multidisciplinary approaches to breathing pattern disorders*. Edinburgh/London/New York/Philadelphia/St. Louis/Sydney/Toronto: Churchill Livingstone; 2002.
81. Meuret A, et al. Changes in respiration mediate changes in fear of bodily sensation in panic disorder. *J Psychiatr Res* 2009;**43**:634–41.
82. Hornsveld H, Garsson B. The low specificity of the hyperventilation test. *J Psychosom Res* 1996;**41**:435–49.
85. Giardino ND, Lehrer P, Feldman J. The role of oscillations in self-regulation. In: McGuigan KA, editor. *Stress and health*. Harwood; 2000.
86. Moser M, et al. Why life oscillates-biological rhythms and health. *Conf Proc IEEE Eng Med Biol Soc* 2006;**1**:424–8.
87. Daly MdB. Interactions between respiration and circulation. In: Cherniack NS, Widdcombe JG, editors. *Handbook of physiology: the respiratory system*. Bethesda, MD.: American Physiological Society; 1986. p. 529–94.

88. Grossman P. Respiratory and cardiac rhythms as windows to central and autonomic biobehavioral regulation: selection of window frames, keeping the panes clean and viewing neural topography. *Biol Psychol* 1992;**34**:131–61.
89. Ford MJ, Camilleri MJ, Hanson RB. Hyperventilation, central autonomic control and colonic tonus in humans. *Gut* 1995;**37**:499–504.
90. Bharucha A, et al. Alpha 2 adrenergic modulation of colonic tone during hyperventilation. *Am J Physiol* 1997;**273**:G1135–40.
91. Busek P, Kemlink D. The influence of the respiratory cycle on the EEG. *Physiol Res* 2005;**54**:327–33.
92. Vern B, et al. Low-frequency oscillations of cortical oxidative metabolism in waking and sleep. *J Cereb Blood Flow Metab* 1988;**8**:215–26.
93. Yasuma F, Hyano J. Respiratory sinus arrhythmia. Why does heartbeat synchronize with respiratory rhythm. *Chest* 2004;**125**:683–90.
94. Song SH, Lehrer P. The effects of specific respiratory rates on heart rate and heart rate variability. *Appl Psychophysiol Biofeedback* 2003;**28**:13–23.
95. Lehrer P, Vaschillo E, Vaschillo B. Resonant frequency biofeedback training to increase cardiac variability: rational and manual for training. *Appl Psychophysiol Biofeedback* 2000;**25**:177–90.
96. Vaschillo E, Lehrer P. Heart rate variability biofeedback as a method for assessing baroreflex function: a preliminary study of resonance in the cardiovascular system. *Appl Psychophysiol Biofeedback* 2002;**27**:1–27.
97. Lehrer P, et al. Biofeedback treatment for asthma. *Chest* 2004;**126**:352–61.
98. Giardino ND, Chan L, Borson S. Combined heart rate variability and pulse oximetry biofeedback for chronic obstructive pulmonary disease: a feasibility study. *Appl Psychophysiol Biofeedback* 2004;**29**:121–33.
99. Karavidas MK, et al. Preliminary results of an open-label study of heart rate variability biofeedback for the treatment of major depression. *Appl Psychophysiol Biofeedback* 2007;**32**:19–30.
100. Gevirtz R. Resonance frequency training to restore autonomic homeostasis for treatment of psychophysiological disorders. *Biofeedback* 1999;**4**:7–9.
101. Sterling P, Eyer J. Allostasis: a new paradigm to explain arousal pathology. In: Fisher S, Reason J, editors. *Handbook of life stress, cognition and health*. New York: John Wiley and Sons; 1988. p. 629–49.
102. Masaoka Y, Homma I. The effects of anticipatory anxiety on breathing and metabolism in humans. *Respir Physiol* 2001;**128**:171–7.
103. Wilhelm FH, Gertvitz R, Roth W. Respiratory dysregulation in anxiety, functional, cardiac, and pain disorders: assessment, phenomenology, and treatment. *Behav Modif* 2001;**25**:513–45.
104. Recordati G, Bellini TG. A definition of internal constancy and homeostasis in the context of non-equilibrium thermodynamics. *Exp Physiol* 2004;**89**:27–38.
105. McCraty R. *The coherent heart: heart-brain interactions, psychophysiological coherence, and the emergence of system-wide order*. Boulder Creek, CA: Institute of Heart Math, www.heartmath.org; 2006.
106. Jerath R, et al. Physiology of long pranayamic breathing: neural respiratory elements may provide a mechanism that explains how slow deep breathing shifts the autonomic nervous system. *Med Hypotheses* 2006;**67**:566–71.
107. Brown R, Gerbarg P. Sudarshan Kriya yogic breathing in the treatment of stress, anxiety, and depression: part 1-neurophysiological model. *J Altern Complement Med* 2005;**11**:189–201.
108. Shannahoff-Khalsa D. Selective unilateral autonomic activation: implications for psychiatry. *CNS Spectr* 2007;**12**:625–34.
109. Pal G, Velkumary S, Madanmohan. Effect of short-term practice of breathing exercises on autonomic functions in normal human volunteers. *Indian J Med Res* 2004;**120**:115–22.
110. Bhargava R, Gogate MG. Autonomic responses to breath holding and its variations following pranayama. *Indian J Physiol Pharmacol* 1988;**32**:257–64.
111. Pramanik T, et al. Immediate effect of slow pace bhastrika pranayama on blood pressure and heart rate. *J Altern Complement Med* 2009;**15**:293–5.
112. Joseph C, et al. Slow breathing improves arterial baroreflex sensitivity and decreases blood pressure in essential hypertension. *Hypertension* 2005;**46**:714–8.
113. Raupach T, et al. Slow breathing reduces sympathoexcitation in COPD. *Eur Respir J* 2008;**32**:387–92.
114. Davis A, et al. A simple biofeedback digital data collection instrument to control ventilation during autonomic investigations. *J Auton Nerv Syst* 1999;**77**:55–9.
115. Nagler J, Krauss B. Capnography: a valuable tool for airway management. *Emerg Med Clin North Am* 2008;**26**:881–97.
116. 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**:1039–42.
117. Dixhoorn Jv. Whole-body breathing: a systems perspective on respiratory retraining. In: Lehrer PM, Woolfolk RL, Sime WE, editors. *Principles and practice of stress management*. New York: Guilford Press; 2007. p. 291–332.
118. Courtney R. Strengths, weaknesses and possibilities of the Buteyko method. *Biofeedback*; 2008.
119. Peng C, Henry Isaac, Mietus Joseph, Hausdorff J, Khalsa G, Benson H, Goldberger A. Heart rate dynamics during 3 forms of meditation. *Int J Cardiol* 2004;**95**:19–27.
120. Ramacharaka Y. *Science of breath: the oriental breathing philosophy*. Chicago: Yogi Publication Society; 1904.
121. Sovik R. The science of breathing—the yogic view. *Prog Brain Res* 2000;**122**:491–505.
122. Swami Rama BR, Hymes A. *Science of breath: a practical guide*. Honesdale, PA: The Himalayan Institute Press; 1976.
123. Janakiramaiah N, Gangadhar BN, Naga Venkatesha Murthy PJ, Harish MG, Subbakrishna DK, Vedamurthachar A. Antidepressant efficacy of Sudarshan Kriya Yoga (SKY) in melancholia: a randomized comparison with electroconvulsive therapy (ECT) and imipramine. *J Affect Disord* 1999;**57**:255–9.
124. Levine B. Intermittent hypoxic training: fact and fancy. *High Alt Med Biol* 2002;**3**:177–93.
125. Meerson F. *Essentials of adaptive medicine: protective effects of adaptation*. Geneva: Hypoxia Medical; 1993.
126. Bowler SD, Green A, Mitchell A. Buteyko breathing technique in asthma: a blinded randomised controlled trial. *Med J Aust* 1998;**169**:575–8.
127. Cooper S, et al. Effect of two breathing exercises (Buteyko and pranayama) in asthma: a randomised controlled trial. *Thorax* 2003;**58**:674–9.
128. McHugh P, et al. Buteyko breathing technique for asthma: an effective intervention. *N Z Med J* 2003;**116**:U710.
129. Slader C, et al. Double blind randomised controlled trial of two different breathing techniques in the management of asthma. *Thorax* 2006;**61**:651–6.
130. Opat A, Cohen M, Bailey M. A clinical trial of the Buteyko breathing technique in asthma as taught by a video. *J Asthma* 2000;**37**:557–64.
131. Cowie R, Underwood MF, Reader PG. A randomised controlled trial of the Buteyko technique as an adjunct to conventional management of asthma. *Respir Med* 2008;**102**:726–32.
132. Abramson M, et al. A randomised controlled trial of the Buteyko method for asthma. *Int J Immunorehabil* 2004;**6**:244.
133. Marks G, et al. *Asthma and complementary therapies: a guide for health professionals*. Canberra: National Asthma Council Australia & Australian Department of Health and Ageing; 2005.
134. Ritz T, et al. Changes in pCO₂ symptoms, and lung function of asthma patients during capnometry-assisted breathing training. 2009.
135. Thomas M, et al. Breathing exercises for asthma: a randomised controlled trial. *Thorax* 2009;**64**:55–61.
136. van Dixhoorn J, Duivenvoorden H. Effect of relaxation therapy on cardiac events after myocardial infarction: a 5-year follow up study. *J Cardiopulm Rehabil* 1999;**19**:178–85.
137. van Dixhoorn J, et al. Physical training and relaxation therapy in cardiac rehabilitation assessed through a composite criterion for training outcome. *Am Heart J* 1989;**118**:545–52.
138. Shannahoff-Khalsa D, et al. Hemodynamic observations on a yogic breathing technique claimed to help eliminate and prevent heart attacks: a pilot study. *J Altern Complement Med* 2004;**10**:757–66.
139. Murthy PJ, Janakiramaiah N, Gangadhar BN, Subbakrishna DK. P300 amplitude & antidepressant response to Sudarshan Kriya Yoga (SKY). *J Affect Disord* 1998;**50**:45–8.
140. Janakiramaiah N, Gangadhar BN, Naga Venkatesha Murthy PJ, Harish MG, Subbakrishna DK, Vedamurthachar A. Therapeutic efficacy of Sudarshan Kriya Yoga (SKY) in dysthmic disorder. *Nimhans J*; 1998;21–8.
141. Meuret A, et al. Feedback of end-tidal pCO₂ as a therapeutic approach for panic disorder. *J Psychiatr Res* 2008;**42**:560–8.
142. Tweedale PM, Rowbottom I, McHardy GI. Breathing training: effect on anxiety and depression scores in behavioural breathlessness. *J Psychom Res* 1994;**38**:11–21.