Journal of Bodywork and Movement Therapies (2006) **10**, 134–146

Journal of
Bodywork and Movement Therapies

www.intl.elsevierhealth.com/journals/jbmt

REVIEW

Is chronic hyperventilation syndrome a risk factor for sleep apnea? Part 1

Jodianne C. Coffee, LMT,BA,BS

NREMT for Pinellas County EMS, All Children's Pediatric & Neonatal Intensive Care Transport Team, Florida, USA

Received 20 October 2004; received in revised form 4 May 2005; accepted 14 July 2005

KEYWORDS
Hyperventilation;
Hypocapnia;
REM;
Atonia;
Sleep;
Apnea

Summary There are many pathologies that are proven risk factors for disordered breathing during sleep, including obstructive pulmonary diseases, neuromuscular diseases, poliomyelitis, obesity, heart failure, and cranio-facial anomalies. When these risk factors are coupled with the normal physiology of sleep, REM sleep in particular, sleep apnea-hypopnea occurs, thereby resulting in nocturnal hypoxia, disrupted sleep, and sleep deprivation. This author proposes that chronic hyperventilation syndrome (HVS) and other upper-chest breathing pattern disorders (BPD) are also risk factors for sleep apnea–hypopnea because of persistent hypocapnia (in chronic HVS) and poor respiratory muscle mechanics, leading to diaphragmatic weakness. Research in this area is seriously lacking considering the enormous impact that both chronic HVS/BPD and sleep apnea–hypopnea syndrome have on human health and public safety.

Introduction

Patients with poor respiratory muscle function are negatively affected by the normal physiology of REM sleep. These negative effects include hypopneic and apneic episodes during sleep, thereby causing nocturnal hypoxia, disrupted sleep patterns and sleep deprivation. It is proposed that upper chest breathing patterns, particularly chronic hyperventilation syndrome, may be risk factors for sleep apnea–hypopnea because of the negative effects these breathing patterns have on diaphragmatic muscle function. Although very little research was found to directly support this hypothesis, studies will be presented which show that any process that impairs respiratory muscle function, especially that of the diaphragm, has the potential to cause disordered breathing during REM sleep.

Mechanics of normal breathing (Fig. 1)

The primary respiratory muscle in humans is the diaphragm, producing 70–80 percent of the inhalation force necessary for sufficient ventilation

E-mail address: jcof33@yahoo.com.

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doi:10.1016/j.jbmt.2005.07.003

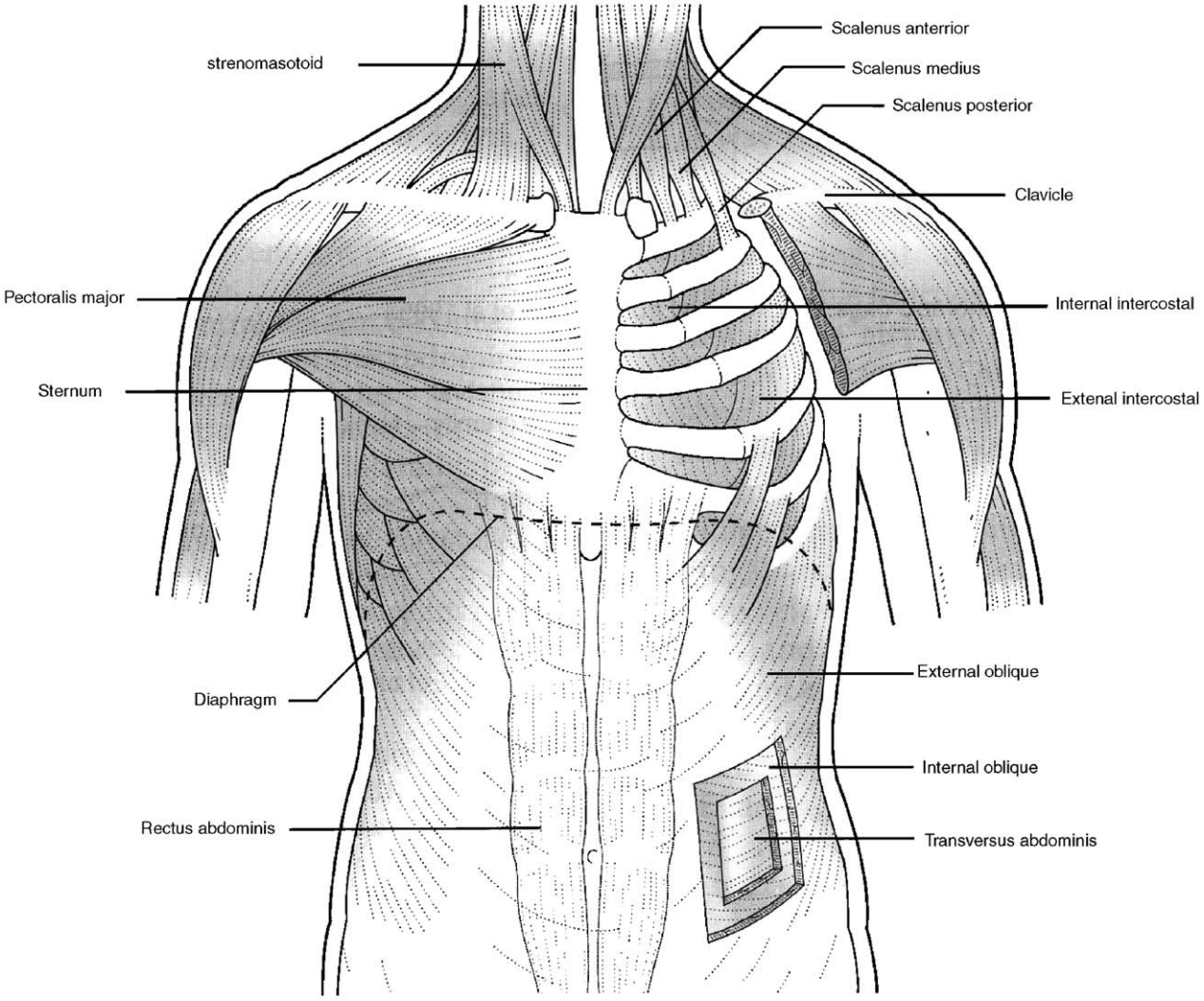

Figure 1 Muscles of ventilation, including accessory muscles. Reprinted with permission from Chaitow et al. (2002), Fig. 1.12.

during periods of normal oxygen demand (Simons et al., 1999). During inhalation the diaphragm contracts and the dome flattens, pulling its central tendon down and increasing the vertical dimension of the thoracic cavity. As the dome of the diaphragm descends, it is resisted by the abdominal viscera. At this point, the costal attachments of the diaphragm, with their fibers running cranially along the inner aspect of the lower ribs, contract and elevate the lower ribs while simultaneously moving them laterally. This movement creates a pivoting action of the rib, which puts torque on the costal cartilage, elastically loading it. The result is elastic recoil of the costal cartilage during the exhalation phase (Kapandji, 1974). Vertical expansion of the upper thoracic cage is accomplished by the parasternal (intercartilaginous) internal intercostals, scalenes, upper and more lateral external intercostals, and the levator costae muscles. The role of the scalenes during normal respiration is to counter the suction created by the action of the diaphragm and prevent the sternum from being pulled down and in (Simons et al., 1999; Kapandji, 1974).

Exhalation during quiet respiration should be effortless and is largely dependent on the elastic recoil of the lungs, pleural membrane and costal cartilage. The abdominal wall and lower intercostals relax downward and the diaphragm ascends back to its original domed position (Simons et al., 1999; Kapandji, 1974).

During periods of increased oxygen demand, an increased load is placed on the accessory muscles of inspiration (sternocleidomastoid, scalenes, upper trapezius, serratus anterior, serratus posterior superior, pectorals, latissimus dorsi, thoracic erector spinae, subclavius, and omohyoid), which

Box 1 Glossary of terms (Stedman's Concise Medical Dictionary, 2001)

Apnea	Cessation of breath.
Asthma	An inflammatory disease of the lungs characterized by reversible (in most cases) airway obstruction.
Caudad	In a direction toward the tail.
Cephalad	In a direction toward the head.
Cheyne–Stokes breathing	Cheyne–Stokes respiration is an abnormal breathing pattern which commonly occurs in patients with decompensated congestive heart failure and neurologic diseases, in whom periods of tachypnea and hyperpnea alternate with periods of apnea. In the majority of these patients, the ventilatory patterns may not be recognized, and the clinical features are generally dominated by the underlying disease process. Cheyne–Stokes respiration may, however, have profound effects on the cardiopulmonary system, causing oxygen desaturation, cardiac arrhythmias, and changes in mental status (Lieber and Mohsenin, 1992). When the duration of an apneic episode exceeds 10 s and if there are more than 5/h, the person is diagnosed with CSA. Impaired cardiac function causes blood to circulate more slowly and CO_2 receptors are not sending current information to the brain about CO_2 levels but, rather, levels of CO_2 in circulating blood from several seconds earlier (Apneos Corporation, 2003).
COPD	(Emphysema) a condition of the lung characterized by increase beyond the normal in the size of air spaces distal to the terminal bronchiole (those parts containing alveoli) with destructive changes in their walls and reduction in their number. Clinical manifestation is breathlessness on exertion due to the combined effect of reduction of alveolar surface for gas exchange and collapse of smaller airways with trapping of alveolar gas in expiration; this causes the chest to be held in the position of inspiration (barrel chest) with prolonged expiration and increased residual volume.
Cranially	See cephalad.
Electroencephalography (EEG)	Registration of the electrical potentials recorded by an electroencephalograph (a system for recording electric potentials of the brain derived from electrodes attached to the scalp).
Electrocardiograph (EKG)	Method of recording electrical activity of the heart: impulse formation and conduction, depolarization and repolarization of atria and ventricles.
Electrooculograph (EOG)	Electrodes placed on the skin adjacent to the eyes measure changes in standing potential between the front and back of the eyeball as the eye moves.
Electromyography (EMG)	The recording of electrical activity generated in muscle for diagnostic purposes; both surface and needle recording electrodes can be used.
Engram	A physical habit or memory trace made on the protoplasm of an organism by the repetition of stimuli.
Fibromyalgia syndrome (FMS)	A condition of chronic, diffuse, widespread aching and stiffness affecting muscles and soft tissues.
Functional residual capacity (FRC)	The volume of gas remaining in the lungs at the end of normal expiration.
Hemoglobin	Red respiratory protein of erythrocytes that transports oxygen from the lungs to the tissues.
Hypercapnia	Abnormally increased arterial carbon dioxide tension.

Hyperventilation	Increased alveolar ventilation relative to metabolic CO_2 production so that alveolar carbon dioxide pressure decreases below normal.
Hypocapnia	Abnormally decreased arterial carbon dioxide tension.
Hypophosphatemia	Abnormally low concentrations of phosphates in the circulating blood.
Hypopnea	Breathing that is shallow and/or slower than normal.
Hypoventilation	Reduced alveolar ventilation relative to metabolic CO_2 production so that alveolar CO_2 pressure increases above normal.
Hypoxemia	Subnormal oxygenation of arterial blood.
Hypoxia	Decrease below normal levels of oxygen in inspired gases, arterial blood, or tissues.
Ischemia	Local anemia due to mechanical obstruction of blood supply.
Kussmaul respirations	Kussmaul breathing is the rapid, deep, and labored breathing of people who have acidosis. The cause of Kussmaul breathing is respiratory compensation for a metabolic acidosis as seen in renal metabolic acidosis or diabetic ketoacidosis (DKA).
Muscular dystrophy	A general term for a number of hereditary, progressive degenerative disorders affecting skeletal muscles and often other organ systems.
NREM	Non-rapid eye movement.
$PaCO_2$	Partial pressure of arterial carbon dioxide.
Polysomnography	Simultaneous and continuous monitoring of relevant, normal, and abnormal physiological activity during sleep.
PREM	Phasic REM.
REM	Rapid eye movement.
SaO_2	Oxygen saturation of hemoglobin.
Sinoatrial Node	The mass of specialized cardiac muscle fibers that normally acts as the 'pacemaker' of the cardiac conduction system; it lies under the epicardium at the upper end of the sulcus terminalis.
SpO_2	Arterial oxyhemoglobin noninvasively measured by a spectrophotometric device using selected wavelengths of light.
Tachycardia	Rapid beating of the heart, conventionally applied to rates over 100 beats/min.
Tidal volume	The volume of air that is inspired or expired in a single breath during regular breathing.
TREM	Tonic REM.

lift and expand the thoracic cage quickly. Exhalation now becomes an active rather than passive process. The muscles of forced exhalation are the abdominal muscles, interosseous internal intercostals, transversus thoracis, the subcostal muscles and some of the lower back muscles, including the quadratus lumborum and serratus posterior inferior. The function of the abdominal and lower back muscles during forced exhalation is to accelerate expiratory airflow by squeezing the abdominal viscera upward while pulling the thoracic cage downward (Simons et al., 1999).

At rest, the adult respiratory rate ranges from 10 to 14 breaths per minute moving a volume of 3–5 l of air per breath. During periods of increased oxygen demand, such as physical or emotional stress, respiratory rates and volumes will fluctuate. These should, however, return to normal resting patterns once stimuli cease (Chaitow et al., 2002) (Box 1).

Hyperventilation syndrome

Hyperventilation is a very functional autonomic nervous system response programmed during our evolution to prepare us for quick action in the face of danger. It boosts sympathetic nervous system activity, sharpens sensory activity, increases blood sugar levels, increases cardiac output, increases blood pressure (shunting blood away from digestive organs to the muscles, heart, and brain), increases oxygen intake, and lowers carbon dioxide levels in the blood, all with the anticipation that physical

exertion will follow. The response is quick and outside of our conscious control and, while it can be lifesaving when faced with real physical danger, it leads to a barrage of health problems when the only existing threat is our psyche (Gilbert, 1999).

Physiology of HVS

Hyperventilation syndrome (HVS) is a breathing pattern disorder characterized by breathing in excess of the body's metabolic needs at the moment, resulting in hypocapnia. Lum (1975) points out that although the majority of the literature defines HVS by the classic triad of massive over-breathing, paraesthesiae, and tetany, these symptoms are only observed in about 1 percent of cases, with tetany being the rarest of manifestations. Ninety-nine percent of patients suffering from HVS present with a collection of bizarre, apparently unrelated multi-system complaints and a 'fat folder' of various diagnoses depending on which specialists have been consulted, often without any appreciable relief of their symptoms (see Fig. 2). Magarian et al. (1983) reasons that since the complaints often are not particularly suggestive of hyperventilation, and because there is minimal discussion of HVS in medical school and textbooks, HVS remains an under-diagnosed disorder.

The necessary diagnostic criteria for hyperventilation that differentiates it from other breathing pattern disorders is hypocapnia. Over-breathing can rapidly eliminate enormous amounts of carbon dioxide (CO_2) resulting in decreased blood levels of CO_2 leading to respiratory alkalosis. Hypocapnia and the rise in pH associated with it are almost immediate. Carbon dioxide is the most important regulator of vascular tone, especially in the brain, and the decrease in blood levels of CO_2 (PCO_2) quickly leads to vasoconstriction and cerebral hypoxia (Magarian et al., 1983). This is made worse by the physiological effects of alkalosis. When pH rises, oxygen (O_2) has an increased affinity for hemoglobin, resulting in decreased tissue oxygenation (Bohr effect) (Magarian et al., 1983). Alkalosis also affects intracellular shifts of phosphorus

Nijmegen questionnaire					
	Never 0	Rare 1	Sometimes 2	Often 3	Very often 4
Chest pain					
Feeling tense					
Blurred vision					
Dizzy spells					
Feeling confused					
Faster or deeper breathing					
Short of breath					
Tight feelings in chest					
Bloated feeling in stomach					
Tingling fingers					
Unable to breathe deeply					
Stiff fingers or arms					
Tight feelings round mouth					
Cold hands or feet					
Palpitations					
Feelings of anxiety					
		Total:	/64*		

*Nijmegen. Patients mark with a tick how often they suffer from the symptoms listed. A score above 23/64 is diagnostic of hyperventilation syndrome.

Figure 2 The Nijmegen questionnaire: Never = never; rare = less than monthly; sometimes = more than monthly but less than weekly; often = at least weekly but not daily; very often = at least every day. Reprinted with permission from Chaitow and Delany (2005), Fig. 16.1, p. 120.

causing hypophosphatemia, which further reduces O_2 availability to the tissues (Magarian et al., 1983).

Hypocapnia and respiratory alkalosis develop rapidly after the onset of hyperventilation but in an acute attack, symptoms usually resolve after the stimulus that provoked the attack is gone, and the body returns to homeostasis (Gilbert, 1998a; Lum, 1975). Some of the physiological changes that occur during attacks of *acute* hyperventilation are easily recognized by most people. These physiological changes include (Magarian et al., 1983; Gilbert, 1999; Chaitow et al., 2002):

- obvious over-breathing (lowers blood CO_2 levels and raises pH);
- vasoconstriction (2 percent reduction in cerebral blood flow for every 1 mm decline in $PaCO_2$);
- cerebral hypoxia (may cause fainting);
- poor tissue perfusion (Bohr effect and hypophosphatemia);
- changes in serum calcium and magnesium levels;
- hyperadrenergic state (increased excitability of corticospinal system speeding up spinal reflexes and hyperirritability of motor and sensory axons);
- smooth muscle constriction;
- increased skeletal muscle tone;
- carpal-pedal spasm;
- visual disturbances;
- sinus tachycardia;
- coronary vessel constriction (ischemia makes cardiac muscle tissue very irritable, especially when it affects the Sinoatrial node, leading to cardiac arrhythmias);
- increased cardiac output (coupled with ischemia will increase the rate of ischemia).

During an acute attack of hyperventilation, a person may subjectively describe feelings of chest pain, anxiety, fear, dyspnea (air hunger), dizziness, weakness, and fatigue.

The physiological changes of *chronic* HVS are milder, more difficult to detect, but more persistent than acute HVS. Two major differences between chronic and acute HVS are the lack of obvious over-breathing in chronic HVS and the long-term compensatory mechanisms of chronic HVS that are not found in acute attacks. These compensatory mechanisms are explained below.

Once a pattern of over-breathing has been established, it can be maintained indefinitely by frequent sighing, yawning, or breath holding alongside an otherwise normal rate of breathing. Because the anticipated respiratory pattern of obvious over-breathing is not present, the diagnosis of hyperventilation is often rejected or unrecognized (Magarian et al., 1983; Lum, 1975; Gilbert, 1998a).

Unlike the alkalosis present in acute hyperventilators, the pH of patients with chronic HVS is relatively normal even in the presence of hypocapnia (see Fig. 3). This finding is also deceptive. The human body is very intent on maintaining its ideal pH of 7.4 (Gilbert, 1998a). Lum (1975) describes three systems for maintaining the body's pH. The buffer system, bicarbonate (HCO_3^-) and carbonic acid, he argues is quantitatively the most important system in the extra-cellular fluid. The other two systems are renal regulation of hydrogen (H^+) excretion and respiratory regulation of CO_2 excretion. Over-breathing results in decreased blood levels of CO_2, an excess of bicarbonate ions, and a deficiency of hydrogen ions. The body compensates for the decrease in CO_2 levels by renal excretion and tissue storage of bicarbonate. Although hypocapnia and the shift in pH towards alkalosis are almost immediate, the compensatory excretion of bicarbonate can take hours to days.

This compensatory mechanism maintains the proper pH in the body but it reduces the acid-base reserve. A new condition is created in the body, which is somewhat stable, but is dependent on persistent hyperventilation. The body will have adjusted to tolerate hypocapnia as the norm. This means that the threshold for symptoms of acute hyperventilation is decreased and physical or emotional disturbances will trigger a chain reaction of increased ventilation, decreasing levels of CO_2, increasing anxiety, followed by more ventilation (Newton, 2004; Gilbert, 1998a; Lum, 1975). The physiological changes associated with chronic HVS are (Magarian et al., 1983; Gilbert, 1998a, 1999):

- hypocapnia;
- relatively normal pH;
- low plasma levels of bicarbonate;
- normal or slightly increased respiratory rate, irregular (sighing, breath-holding);
- suppression of hydrochloric acid in the stomach (indigestion) (Bradley, 2001; Gilbert, 1998a; Lum, 1975);
- retention of hydrogen by the kidneys;
- lactic acid production;
- effort syndrome (easily exhausted by exercise-related to low plasma bicarbonate levels);
- hypophosphatemia (general muscle weakness and impaired nerve conduction);
- tendency to breath primarily with thoracic musculature;
- sinus tachycardia and supraventricular arrhythmias on ECG *even during sleep*.

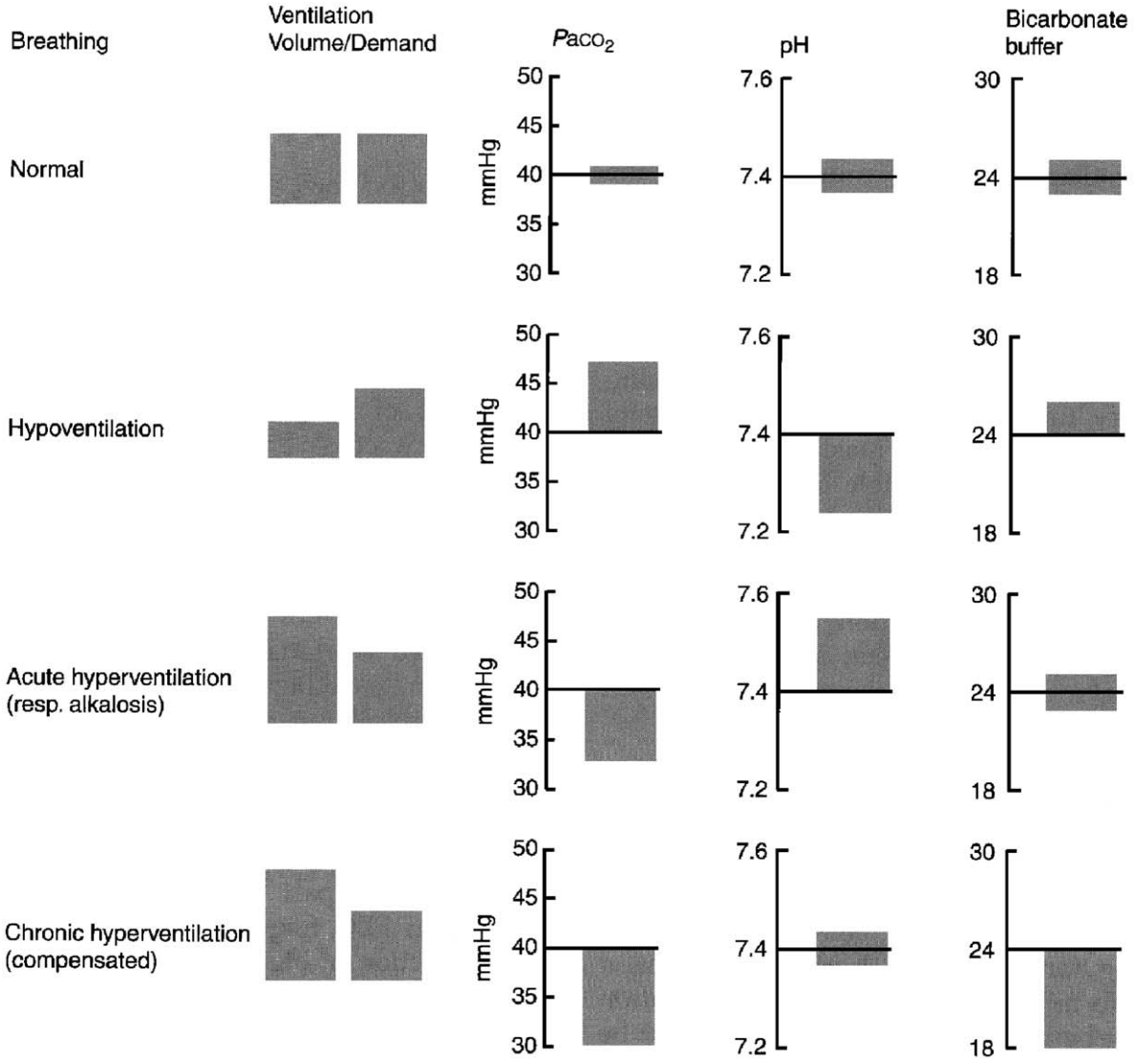

Figure 3 When breathing volume does not match what the body needs (demand) there are changes in $PaCO_2$ which affect pH. The bars are meant to show directional shifts rather than exact values. Reprinted with permission from Chaitow et al. (2002), Fig. 3.3, p. 67.

Symptoms that patients with chronic HVS might describe are cold hands, chest pain/tightness (possible coronary vasoconstriction with cardiac ischemia), dyspnea, muscle pain/sensitivity, anxiety, *poor sleep with nocturnal panic attacks*, and 'foggy' thinking.

Nixon and Andrews (1996) emphasize the importance of aerobic conditioning to increase the threshold for hyperventilation. In de-conditioned individuals the threshold for hyperventilation is lower because of an increased reliance on anaerobic metabolism. This results in dyspnea and early fatigue during aerobic exercise.

Mechanics of HVS

The mechanical effects of chronic HVS on the breathing apparatus are due to misuse and over-use of accessory respiratory and ventilatory musculature. Lum (1975) believed that the exaggerated tendency to breathe with the thoracic musculature is an important factor in the development and persistence of chronic HVS (see Figs. 4a and b). Newton (2004) points out the tendency to breathe using the upper thorax rather than the diaphragm results in chronically over-inflated lungs (similar to that found in obstructive pulmonary disease patients) putting the diaphragm at a mechanical disadvantage.

The metabolic effects of hypocapnia on muscle tissue, including altered levels of calcium, magnesium, and phosphorus, can impair the ability of the muscle tissue to contract and relax. However, impaired respiratory muscle function can exist in BPD patients without hypocapnia. The excessive

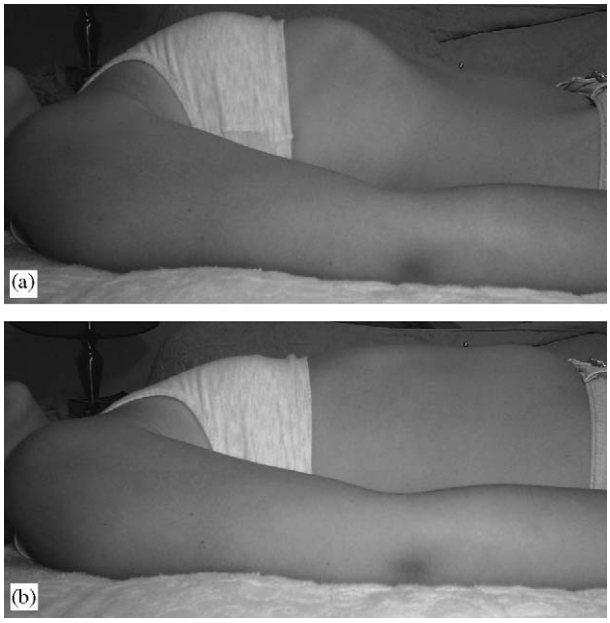

Figure 4 (a) Upper chest breathing pattern. Note the excessive upper thoracic efforts on inhalation with inward movement of abdomen. (b) Diaphragmatic breathing pattern. Note abdominal protrusion with only minimal anterior movement of the sternum towards the end of inhalation.

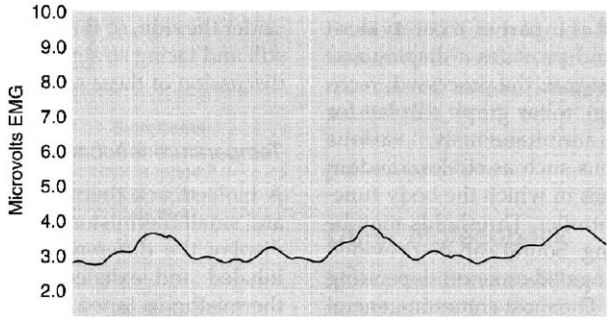

Figure 5 EMG tracing of scalene muscle (side of neck) during normal breathing (three breaths) with abdominal expansion and shoulders relaxed. Reprinted with permission from Chaitow et al. (2002), Figs. 8.4, p. 214.

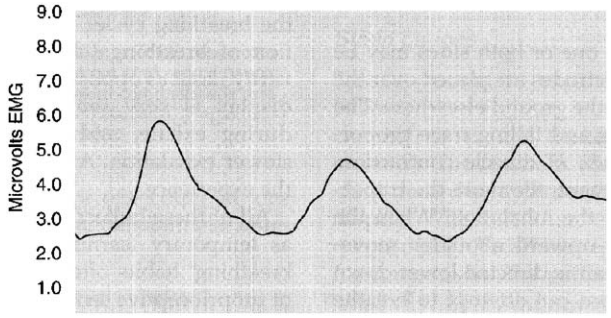

Figure 6 EMG tracing of scalene muscle (three breaths) with abdominal movement restricted; accessory muscles recruited. Reprinted with permission from Chaitow et al. (2002), Fig. 8.5, p. 214.

efforts of the thoracic musculature and the inhibition of diaphragmatic activity in patients with any breathing pattern disorder, can result in muscle tissue hypoxia, pain, fatigue, trigger points, shortening (in postural muscles), and weakness of the respiratory muscles (Gilbert, 1998b; Magarian et al., 1983; Chaitow, 2004). Although patients with chronic HVS or other upper chest breathing patterns may not present with obvious overbreathing, a keen observer may notice the patient's thorax rising and falling in the vertical plane rather than the anterior chest and abdomen moving in and out (see Figs. 5 and 6). Also, the lower ribs will be drawn in during inhalation instead of expanding laterally (Liebenson, 1999). This paradoxical respiratory pattern interferes with the normal function of the diaphragm, which will lose its ability to forcefully contract (Gilbert, 1998b; Chaitow, 2004). In addition, patients who activate the abdominal muscles during inhalation (abdominals should relax during inhalation while the diaphragm contracts) are reciprocally inhibiting the activity of the diaphragm (Simons et al., 1999).

The presence of chronic HVS or other breathing pattern disorders does not exclude the existence of more serious pathologies. In fact, organic disease could quite possibly be the perpetuating factor of hyperventilation, such as Kussmaul breathing, seen in renal metabolic acidosis or diabetic ketoacidosis (DKA).

Sleep physiology and its effects on respiration

Sleep is defined by Russo (2004) as a state of consciousness in which the brain is relatively more responsive to internal than to external stimuli. Non-REM (NREM) sleep is divided into four stages (I–IV) and is followed by rapid eye movement (REM) sleep. This sequence makes up one sleep cycle and lasts about ninety minutes, though the ratio of NREM to REM varies from cycle to cycle. REM sleep occurs about 4–5 times per 8–9 h of sleep with the majority of REM sleep occurring in the early morning hours (Benbadis, 2004; Russo, 2004).

During NREM sleep the EEG (electroencephalogram) reveals large amplitude slow waves (oscillations), body functioning slows, there are slow

rolling eye movements, the pupils constrict, the respiratory and heart rates decline, blood pressure decreases, and there is a reduction of total body oxygen consumption (USResolve.org, 2004). The functions of NREM sleep remain speculative. One theory suggests that the decreased metabolic demand facilitates replenishment of glycogen stores. Another theory suggests that the oscillations during NREM sleep consolidate memory and remove excess synapses (Russo, 2004).

REM sleep is characterized by cortical activation, low-voltage desynchronized EEG, rapid eye movements, and a strong suppression of postural muscle tone (atonia) (Russo, 2004; Leszek et al., 1998). REM has both tonic and phasic characteristics. Tonic REM (TREM) muscle atonia is present throughout REM sleep as a background during which intermittent bursts of phasic activity occur and is generated by mesenchephalic and pontine cholinergic neurons (see Box 2).

The intermittent bursts of phasic REM (PREM) activity are marked by rapid eye movements, PGO (pontine-geniculate-occipital) spikes on EEG, periodic skeletal muscle twitches (interruptions of tonic atonia) with increases and decreases of activity in muscles not rendered completely atonic, increased heart rate variability, pupil dilation, and increased respiratory rate (Russo, 2004; Leszek et al., 1998).

While the neural mechanisms of REM-sleep muscle atonia are still not completely understood, extensive research in this field has provided a basic understanding. The nervous system reduces neuronal activity by employing one or both of the following: state specific activation of inhibitory inputs, involving the amino acids glycine and GABA, or withdrawal of exitory inputs (defacilitation), involving the neurotransmitters serotonin and noradrenaline (Leszek et al., 1998).

During wakefulness, the release of acetylcholine (Ach) at the neuromuscular junction causes an influx of positively charged sodium ions through the sodium ion channels of the muscle cell membrane into the cell. This results in a depolarization across the cell membrane and an exitatory post-synaptic potential, stimulating the entry of calcium ions and triggering a muscle contraction (Simons et al., 1999).

During REM muscle atonia, Ach is present in the same levels as wakefulness but because of the activation of inhibitory input and/ or defacilitation, there is an increase in the permeability of the cell membrane to negatively charged chloride ions. The influx of chloride ions results in hyperpolarization (inside the cell membrane is more negatively charged than outside the cell membrane) and an inhibitory post-synaptic potential, decreasing the likelihood of a post-synaptic action potential. This post-synaptic inhibition renders postural muscles atonic and suppresses upper airway muscle tone, yet has very little effect on the diaphragm and the muscles of the eyes (Xi et al., 2001; Leszek et al., 1998).

Sleep has well-recognized effects on respiration, including changes in central respiratory control, airway resistance, and muscular contractility, which has no significant adverse impact in normal individuals. These effects cause mild hypoventilation with consequent hypercapnia and a diminished response to respiratory stimuli (chemical, mechanical, and cortical inputs), particularly during REM sleep (McNicholas, 2000, 2002).

During REM sleep, breathing appears to be relatively free of chemical feedback control depending, rather, on a higher cortical drive, resulting in breathing that is quite irregular. Orem (1980) states the arousal response to hypoxia alone is poor during REM sleep. Hypercapnia, however, is a strong arousal stimulus and results in waking of most individuals before a 15 mmHg rise in $PaCO_2$ occurs. It is worth speculating that *if baseline CO_2 levels are lower (hypocapnia) in patients with*

Box 2 REM muscle atonia vs. sleep paralysis

REM muscle atonia should not be confused with isolated or recurrent sleep paralysis (SP). REM muscle atonia is a normal function of REM sleep that is believed to prevent sleepers from acting out dreams and injuring themselves or others (as seen in people with REM behavior disorder). Sleep paralysis is when REM muscle atonia occurs immediately prior to falling asleep or upon awakening. In either case the person is alert but is unable to move (Orem 1980; Bonham and Terrillon, 1998).

Buzzi and Cirignotta (2000) state that isolated sleep paralysis (ISP) occurs at least once in a lifetime in 40–50 percent of normal subjects, while as a chronic complaint (recurrent sleep paralysis), it is an uncommon and scarcely known disorder. SP is one symptom of narcolepsy (the others being cataplexy and irresistible sleep) and many people report hypnogogic (while falling asleep) and hypnopompic (upon waking) hallucinations during episodes of SP, which suggest activation of the limbic system.

chronic HVS, will the sleeper be hypoxic for longer periods of time before CO_2 levels reach arousal levels? Or, will hypocapneic patients experience more frequent arousals from sleep due to a reduced bicarbonate reserve?

Breathing during NREM sleep is very regular, exclusively under chemical and mechanical feedback control, and appears to be so dependent on $PaCO_2$ levels that when they drop below a certain point, breathing efforts cease. This is called the apneic threshold. It is unclear if this apneic threshold is lowered in individuals who are chronically hypocapnic in order to maintain respiratory drive during sleep.

McNicholas (2000) states the reduction in accessory respiratory muscle contribution to breathing, particularly during REM sleep, results in a decreased functional residual capacity (FRC) and contributes to worsening ventilation/perfusion (V/Q) relationships during sleep. One study by Tusiewicz et al. (1977) revealed:

> In an awake subject in the supine position, the rib cage contributed forty four percent to the tidal volume and had essentially the same contribution in quiet sleep. However, in active, or REM sleep, the rib cage contribution fell to nineteen percent of the tidal volume. This was accompanied by marked reduction in the intercostal EMG.

Other research studied nine healthy adolescents to determine the effects of sleep state on ventilation and the mechanics of breathing:

> Minute ventilation was state dependent, decreasing by a mean of eight percent from wakefulness to NREM sleep. These changes were caused by changes in respiratory rate. Tidal volume (VT) was not affected by sleep state. The pattern of breathing during wakefulness was similar to that of REM sleep. During NREM sleep, intercostal and diaphragmatic muscle activity increased by a mean of thirty four percent and eleven percent respectively, as compared with wakefulness, indicating an increase in the respiratory workload. This was accompanied by a substantial increase in rib cage contribution to VT. REM sleep was associated with a marked decrease in intercostal muscle activity and a diminished rib cage contribution; VT was maintained due to a mean increase of thirty four percent in diaphragmatic muscle activity. (Tabachnik et al., 1981)

The reduction in rib cage contribution to breathing recorded during REM sleep, as compared with wakefulness and NREM sleep, was caused by the marked reduction in intercostal muscle activity while diaphragmatic contraction was minimally affected. McNicholas (2000, 2002) emphasizes the clinical significance of the fall in intercostal muscle activity for patients who are particularly dependent on accessory muscle activity to maintain ventilation. Therefore, any process affecting diaphragmatic function might pose significant challenges in maintaining ventilation and perfusion during sleep (Buzzi, 2000).

Sleep Apnea

Apnea comes from the Greek word 'apnoia', meaning 'want of breath' (Stedman's Concise Medical Dictionary, 2001). Sleep apnea refers to the temporary cessation of breath during sleep. The result is a disrupted sleep pattern and a myriad of negative physical, mental, and social effects on the patient. The majority of the current literature on sleep apnea defines two, sometimes three different pathophysiologies associated with sleep apnea–hypopnea syndrome, and, as Kingman (2003) points out, more than one form of apnea can occur in the same patient over a night.

Obstructive sleep apnea (OSA) is the most common presentation, associated with obesity, snoring, and upper airway obstruction by soft tissue, such as the tonsils (Chaitow et al., 2002).

Central sleep apnea (CSA) is less common and is attributed to a failure of the respiratory centers to initiate respiration. CSA is often diagnosed in patients with various neuromuscular diseases, such as muscular dystrophies and myasthenia gravis, as well as patients with brain injury. Heart failure is the most common cause of CSA. Forty to sixty percent of heart failure patients are diagnosed with CSA and a particular respiratory pattern called Cheyne-Stokes breathing (crescendo-decrescendo-apnea) (Baldwin and Quan, 2002; Apneos Corporation, 2003).

An abundance of research is available concerning the effects of sleep state on ventilation and perfusion in patients with neuromuscular diseases:

> Nocturnal sleep has some adverse influence on oxygen imbalance in these patients [Duchenne muscular dystrophy] as suggested by the occurrence of arterial oxyhaemoglobin desaturation occurring mainly during REM stages. (Manni et al., 1991)
>
> Apnea and hypopnea are mainly not obstructive and occur predominantly in REM sleep. They are associated with oxygen desaturation [myasthenia gravis]. (Gajdos and Quera Salva, 2001)

Regarding nine Duchenne muscular dystrophy, ten myotonic muscular dystrophy, eight mitochondrial myopathy, and six spinal muscular atrophy patients:

> Nocturnal hypoxemia occurs in relation to apnoeas or hypopnoeas, mainly of central type, especially when these breathing irregularities occur during REM sleep. (Cerveri et al., 1993)

The reduction in respiratory muscle function, especially the diaphragm, as the result of neuromuscular disease, caused significant oxygen desaturations in the above patients during REM muscle atonia, as opposed to wakefulness and NREM sleep, when accessory muscles of respiration were *almost* completely paralyzed.

The third classification of sleep apnea, not always mentioned in the literature, is restrictive sleep apnea. RSA is defined by a failure in the mechanics of the breathing apparatus. RSA largely occurs in patients who rely heavily on accessory respiratory muscles for ventilation because the ventilatory pump muscle, the diaphragm, is mechanically compromised. Some post-poliomyelitis patients suffer from RSA (Bieniek et al., 2001). Patients with COPD (emphysema) and acute asthma are also good examples. These diseases are characterized by airflow obstruction and a significant increase in accessory respiratory muscle workload, intrinsic weakness, and a mechanical disadvantage, especially in diaphragmatic length-force relationship, due to hyperinflation of the lungs (Barbarito et al., 2001).

Barbarito et al. (2001) also explains that during the course of these diseases, respiratory muscles undergo structural and functional changes. For example, under some circumstances, the flattened diaphragm of COPD and acute asthma patients can reverse its effect by pulling the costal margin in rather than lifting it up and out (Simons et al., 1999). In acute asthma patients, Barbarito et al. (2001) noted persistent inspiratory muscle activity during expiration. This may explain why the following patients show EMG activity in sternocleidomastoid and scalene muscles during expiration, as noted here by Johnson and Remmers (1984).

> Hypoventilation contributes to oxyhemoglobin desaturation during rapid eye movement (REM) sleep in patients with severe chronic obstructive pulmonary disease (COPD). Due to hyperinflated lungs, these patients have mechanically impaired diaphragms and increased activity of other inspiratory muscles while awake …We, therefore, recorded scalene (SCA) and sternocleidomastoid (SCM) electromyograms in six subjects with severe COPD. SCA activity decreased seventy six percent from NREM to tonic REM [TREM] and decreased an additional seventeen percent during phasic REM [PREM]. SCM activity was much more variable during NREM, but when present, also decreased during REM. SCA activity correlated strongly with rib cage (RC) excursion. SCA and SCM activity, RC excursion, estimates of minute ventilation, and oxyhemoglobin saturation all decreased in parallel. Expiratory activity of the SCA and SCM, present during wakefulness and NREM, disappeared during REM. We conclude that loss of inspiratory activity of rib cage muscles during REM causes chest wall distortion and hypoventilation in patients with severe COPD.

The conclusion drawn from these sleep studies is that patients who rely heavily on respiratory accessory muscles for ventilation during wakefulness because of impaired diaphragmatic function, suffer from apneic or hypopneic episodes during REM sleep, thereby resulting in hypoxia (see Box 3).

Kingman (2003), however, argues that there is only one causative factor for sleep apnea. He

Box 3 Hypoxia, disturbed sleep, and fibromyalgia syndrome (FMS).
It is interesting that disrupted sleep patterns and low oxygen saturation levels (SaO_2%) during sleep are associated symptoms of fibromyalgia. Alvarez et al. (1997) evaluated sleep apnea and SaO_2% levels in a control group compared with patients with FMS concluded that patients with FMS showed small overnight falls in SaO_2% and spent more time during the night with SaO_2% below 92 percent than did the control group. Chaitow (2003) points out the potential for faulty breathing mechanics, present in upper chest breathing patterns, to persist throughout sleep and be directly responsible for the observed reduction in SaO2% recorded in FMS patients. This could potentially be the by-product of normal sleep physiology in a patient with abnormal breathing mechanics as seen in chronic HVS or other persistent upper-chest breathing pattern.

Nocturnal hypoxic events could potentially be responsible for the existence and/or persistence of myofascial pain and trigger points in chronic pain patients by increasing ischemia in soft tissues.

contends that it is the physiology of the sleep state itself that causes nocturnal apneic–hypopneic episodes and, while all of the above mentioned are risk factors, they are not the causes of sleep apnea–hypopnea syndrome:

> In sleep there is a reorganization of cortical (e.g. neural) control that includes changes in direct cortical drive to and peripheral reflex control of the muscles of the chest wall, upper airway, and ventilation. The disease-defining event is state related requiring sleep in the presentation of the disease [sleep apnea–hypopnea syndrome].

Obesity, tonsillar hypertrophy, obstructive pulmonary diseases, neuromuscular diseases, and cranio-pharyngeal anomalies are risk factors (among others) present during wakefulness but do not invariably produce apneic–hypopneic episodes (Kingman, 2003).

This is important because in patients with chronic HVS, the increased upper thoracic respiratory efforts cause hypertonicity, shortening, and weakness of respiratory accessory muscles. The overuse of muscles causes ischemia and a local energy crisis in the muscle cells. This reduces the muscle's ability to forcefully contract (see Box 5).

The diaphragm and lower thoracic cage muscles become inhibited, restricted, and also weak. For these reasons this author strongly believes that chronic HVS, and any chronic upper-chest breathing pattern disorder, is a risk factor for sleep apnea–hypopnea syndrome but, because of the subtle presentation and the bizarre symptoms reported by the patient, sleep apnea may be as under-diagnosed as chronic HVS itself.

The only study found linking diaphragmatic weakness and sleep apnea was a study done on rats by Gaspar Farkas, a University of Buffalo associate professor of physical therapy and exercise science (Baker, 1996). Since the original research written by Farkas was not found, the occurrence of apneic episodes is not documented.

> The researchers measured diaphragm function in lean and obese rats over a normal eighteen month lifetime, testing them when they were young (6–8 weeks), mature (10–12 months), and old (17–18 months). They found that the diaphragm muscles in the obese animals lost their ability to respond forcefully as the animals aged. Diaphragm response in the obese young animals was not compromised.

According to Baker (1996), Farkas points out that obesity was not the causative factor in sleep apnea in these rats. Obesity overloaded and weakened the diaphragm over time and during REM sleep, when the diaphragm is the only respiratory muscle that is active, this created a double deficit. Research in this area is seriously lacking and is worthy of consideration due to the enormous benefits to patients worldwide (see Box 4).

Box 4 The effects of sleep apnea on health

Cardiopulmonary (Baldwin and Quan, 2002; Chaitow et al., 2002)

- Nocturnal dysrhythmias
- Diurnal hypertension
- Pulmonary hypertension
- Right or left ventricle failure
- Myocardial infarction
- Stroke (CVA)

Neurobehavioral (Baldwin and Quan, 2002; Chaitow et al., 2002)

- Excessive daytime sleepiness
- Diminished quality of life
- Adverse personality changes
- Motor vehicle accidents

Cognitive (Baldwin and Quan, 2002)

- Attention deficits
- Impaired concentration
- Memory difficulties
- Decreased ability to initiate new mental processes and to inhibit automatic ones
- Tendency to repeat errors

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