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A Hyperventilation Theory of Job Stress and Musculoskeletal Disorders

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Background *There is evidence of a link between job stress and upper extremity work-related musculoskeletal disorders. However, the biobehavioral mechanisms by which psychosocial stress factors contribute to the development of musculoskeletal disorders are uncertain.*

Methods *Based on established principles of breathing and job stress and the relevant empirical literatures, a hyperventilation theory of job stress and work-related musculoskeletal disorders was developed.*

Results *Hyperventilation (overbreathing) refers to a drop in arterial CO_2 caused by ventilation that exceeds metabolic demands for O_2. Excessive loss of CO_2 (increase in rate of flow of CO_2 from cells to longs) that results from hyperventilation produces a rise in blood pH (i.e., respiratory alkalosis). This disruption in the acid–base equilibrium triggers a chain of systemic physiological reactions that have adverse implications for musculoskeletal health, including increased muscle tension, muscle spasm, amplified response to catecholamines, and muscle ischemia and hypoxia. Hyperventilation is often characterized by a shift from a diaphragmatic to a thoracic breathing pattern, which imposes biomechanical stress on the neck/shoulder region due to the ancillary recruitment of sternocelidomastoid, scalene, and trapezius muscles in support of thoraci breathing.*

Conclusions *A hyperventilation theory provides an innovative framework for understanding how job stress contributes to pathophysiological processes that increase the risk of work-related musculoskeletal disorders. With respect to the control of these disorders, a hyperventilation theory has important implications for establishing effective work organization interventions and individual stress-management methods. In this regard, breathing is a biobehavioral metric for assessing whether psychosocial aspects of work organization are in balance with a worker's needs and resources. A hyperventilation theory also provides a unique rationale for coping with job stress and musculoskeletal discomfort through breathing training, light physical exercise, and rest breaks.* Am. J. Ind. Med. 41:420–432, 2002. © *2002 Wiley-Liss, Inc.*

KEY WORDS: biobehavioral; biopsychology; breathing; ergonomics; hyperventilation; musculoskeletal disorders; job stress; respiration

INTRODUCTION

Work-related musculoskeletal disorders of the neck/shoulders, arms, elbows, wrists, and hands are a leading occupational health problem. Terms used to describe these disorders include cumulative trauma disorders, overuse injury, repetitive motion injury, and repetitive strain injury [Putz-Anderson, 1988]. Hagberg et al. [1995] refer to work-related musculoskeletal disorders as a descriptive term for disorders and diseases of the neck and upper

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extremities. Causal mechanisms include exposure to physical and psychosocial factors at work, as well as individual factors.

With respect to physical factors, a broad convergence of research from biomechanical, epidemiological, and clinical studies supports the premise that excessive force, high repetition, and awkward postures have adverse implications for musculoskeletal health [Hagberg et al., 1995; Bernard, 1997; National Research Council, 1999]. The National Research Council [1999] concluded in a comprehensive review of the scientific literature on work-related musculoskeletal disorders that:

> There is a higher incidence of reported pain, injury, loss of work, and disability among individuals who are employed in occupations where there is a high level of exposure to physical loading than for those employed in occupations with lower levels of exposure (p 27).

Research on the causes of musculoskeletal disorders has focused mainly on biomechanical risk factors. However, there is increasing evidence that psychosocial aspects of work organization (e.g., quantitative workload, lack of job control, and job-future uncertainty) also contribute to the development of work-related musculoskeletal disorders. Epidemiological investigations [Theorell et al., 1991; Bernard et al., 1994; Hales et al., 1994; Lim and Carayon, 1995; Conway et al., 1996] and experimental studies [Westgaard and Bjorklund, 1987; Waersted et al., 1991, 1996; Lundberg et al., 1994; Schleifer et al., 1997] provide empirical support for a link between psychosocial stress factors and musculoskeletal symptoms/disorders of the neck and upper extremities.

Drawing on an extensive job stress literature, recent theoretical models have postulated a variety of biologically plausible mechanisms by which psychosocial factors at work might increase the risk of musculoskeletal disorders. Smith and Carayon's [1996] work organization model of musculoskeletal disorders includes a number of psychobiological mechanisms, including increased blood pressure, corticosteroids, peripheral neurotransmitters, muscle tension, and decreased immune system response. Sauter and Swanson's [1996] ecological model of musculoskeletal disorders includes increased muscle tension and other autonomic effects as an outcome of psychological strain. Melin and Lundberg's [1997] descriptive model of musculoskeletal disorders postulates that increased muscle tension due to work stress may be independent of or interact with increased catecholamine and cortisol secretions. Feuerstein et al. [1999] hypothesize that musculoskeletal disorders are associated with an individual work style, or a patterning of behavioral, cognitive, and physiological responses to a set of psychosocial demands. Pathophysiological processes include increased muscle tension, sympathetic arousal, peripheral vasoconstriction, and immunosuppression.

Another biophysiological mechanism by which job stress factors might contribute to musculoskeletal disorders is breathing [Schleifer and Ley, 1996]. The premise that there is a relationship between breathing and musculoskeletal disorders is not new. Among the known clinical symptoms of hyperventilation (i.e., overbreathing), Lewis [1959] included musculoskeletal symptoms of muscle pains, tremors, and tetany. Lum [1976] noted more than a decade prior to the recent epidemic of work-related musculoskeletal problems that the symptoms of hyperventilation resemble a variety of underlying disease processes, including carpal tunnel syndrome, thoracic inlet [outlet] syndrome, and disc lesions. Lum [1976] also observed that hyperventilation played an important role in Gottlieb's [1969] list of symptoms of nonorganic diseases diagnosed as anxiety states, including skeletal symptoms of weak limbs, painful limbs, and vague pains. More recently, Ley [1995], Schleifer and Ley [1996], and Nixon [1995] proposed that an association exists between stress-induced hyperventilation and upper extremity musculoskeletal disorders at work.

Until recently, breathing has received little attention in the job stress literature. However, the neglect of breathing is not surprising. Monitoring respiratory parameters such as CO_2 requires a gas analyzer, which is a relatively expensive and complex machine that necessitates daily calibration and is cumbersome to operate in the workplace. By comparison, heart rate and blood pressure, commonly used indicators of stress, can be readily monitored in work settings using a stethoscope, cuff, and sphygmomanometer.

The purpose of the present paper is: (a) to propose a hyperventilation theory of job stress that attempts to explain how psychosocial factors at work increase the risk of musculoskeletal disorders and (b) to discuss the implications of a hyperventilation theory of job stress for workplace interventions, and the control of musculoskeletal disorders.

WHAT IS HYPERVENTILATION?

Under daily stressful conditions that generate emotional arousal, well-established changes in breathing patterns occur. Respiration rate and minute ventilation increase, the mode of respiration shifts from diaphragmatic or abdominal breathing to thoracic or chest breathing, and hyperventilation (overbreathing) occurs [Timmons and Ley, 1994; Ley, 1999].

Hyperventilation refers to breathing that exceeds the metabolic requirements for oxygen. Hyperventilation should not be confused with hyperpnea (voluminous breathing) or tachypnea (rapid breathing or panting), which occur in response to increased metabolic demands (e.g., exercise). Hyperventilation can occur regardless of whether the rate of respiration is fast or slow. It is determined by the rate of flow

for a given volume of air breathed per unit of time that exceeds the metabolic need for O_2, and in so doing causes a low concentration of CO_2 in expired air [see Gravenstein et al., 1995].

End-tidal CO_2 refers to the peak concentration of CO_2 in a single breath of expired air at the end of the ventilation cycle. A graphic representation of the ventilation cycle and end-tidal CO_2 is shown in Figure 1. The peak concentration of CO_2 is attained at the point where the expiratory phase ends and the inspiratory phase begins. A gas analyzer (capnograph) and nasal cannulae are used to measure end-tidal CO_2 in samples of expired air. End-tidal CO_2 is a very close estimate of the partial pressure of CO_2 in arterial blood (P_aCO_2). Typically, end-tidal CO_2 concentrations of approximately 5% (i.e., 38–40 mm Hg) correspond with normal breathing patterns under relaxed conditions. Hyperventilation is defined by a reduction of any amount in P_aCO_2, even when the CO_2 concentration remains in a normal range [Lum, 1976], but clinically relevant hyperventilation refers to a drop in P_aCO_2 that produces a rise in arterial pH (i.e., respiratory alkalosis) that precipitates symptoms.

THE RELATIONSHIP BETWEEN STRESS AND HYPERVENTILATION

With few exceptions studies have routinely found a relationship between stress and respiratory function [Duffy, 1962; Sternbach, 1966; Cohen et al., 1975]. For example, Suess et al. [1980] reported that end-tidal CO_2 decreased from baseline among 27 of 29 subjects who performed a perceptual-judgment task under threat of electric shock for making too many errors. Reductions in end-tidal CO_2 were accompanied by increases in heart rate and self-reported anxiety scores. Ley and Yellich [1998] found that reductions in end-tidal CO_2 were greater among high test-anxious students than low test-anxious students during a computation and word-recall test.

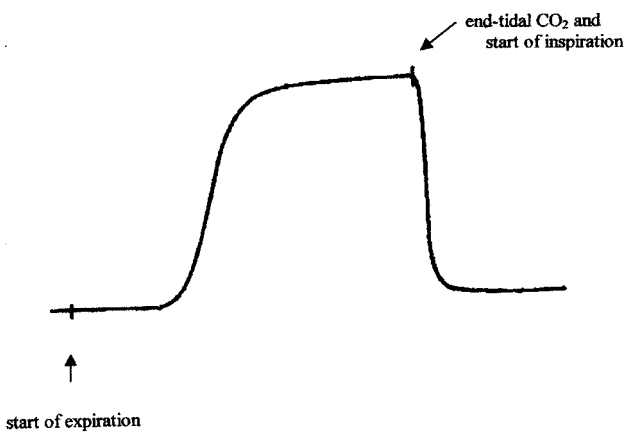


FIGURE 1. CO_2 wave form during the ventilation cycle.

Several other studies have reported the typical inverse relationship between the magnitude of stress and P_aCO_2 or end-tidal CO_2 [Beckmann, 1915; Dudley et al., 1964a,b; Motta et al., 1971; Garssen, 1980]. Conversely, the expected rise in end-tidal CO_2 levels under conditions of quiet adaptation, suggesting a linear relationship between stress and end-tidal CO_2 and P_aCO_2, has been reported by Naifeh and Kamiya [1981] and Suess et al. [1980].

HYPERVENTILATION AND JOB STRESS

Based on the apparent relationship between stress and hyperventilation, Ley [1987; 1994] proposed using end-tidal CO_2 to index the physiological effects of job stress. Schleifer and Ley [1994a] subsequently compared the effectiveness of end-tidal CO_2, respiration rate, and cardiac inter-beat interval in discriminating among the following conditions: self-relaxation, progressive muscle relaxation, and sustained, repetitive computer work. Figure 2 shows that end-tidal CO_2 increased from baseline to relaxation, decreased from relaxation to work, and decreased from baseline to work. In comparison to respiration rate, cardiac inter-beat interval, and self-ratings of relaxation and tension, end-tidal CO_2 was the only indicator that differentiated all three conditions, indicating the utility of this breathing parameter as an index of job stress.

Schleifer and Ley [1994b] also found that end-tidal CO_2 discriminates between high- and low-mental-workload (i.e., electronic performance monitoring vs. no electronic performance monitoring) in a computer-based data-entry task. As shown in Figure 3, the decrease in end-tidal CO_2 from baseline to Day 1 in each of the three work periods was the same for both conditions prior to the introduction of electronic performance monitoring. However, following the introduction of electronic performance monitoring, the decrease in end-tidal CO_2 from Day 1 to 3 for the first and second work periods was significantly greater under the high- than the low-mental-workload condition, indicating a physiological stress effect. The findings of this study as well as of those cited earlier indicate that workers will hyperventilate under stressful job conditions. This apparent cause–effect relationship between job stress and overbreathing is an important requirement of a hyperventilation theory of job stress and musculoskeletal disorders.

A significant advantage that end-tidal CO_2 has over other physiological stress indicators, such as heart rate or blood pressure, is its unique ability to differentiate between the psychosocial and metabolic demands of a task. Psychosocial stress effects are indicated by reductions in end-tidal CO_2 (i.e., breathing that exceeds metabolic demand for oxygen), while metabolic effects are indicated by increases in end-tidal CO_2. By comparison, heart rate, respiration rate, or measures of ventilation such as minute volume,

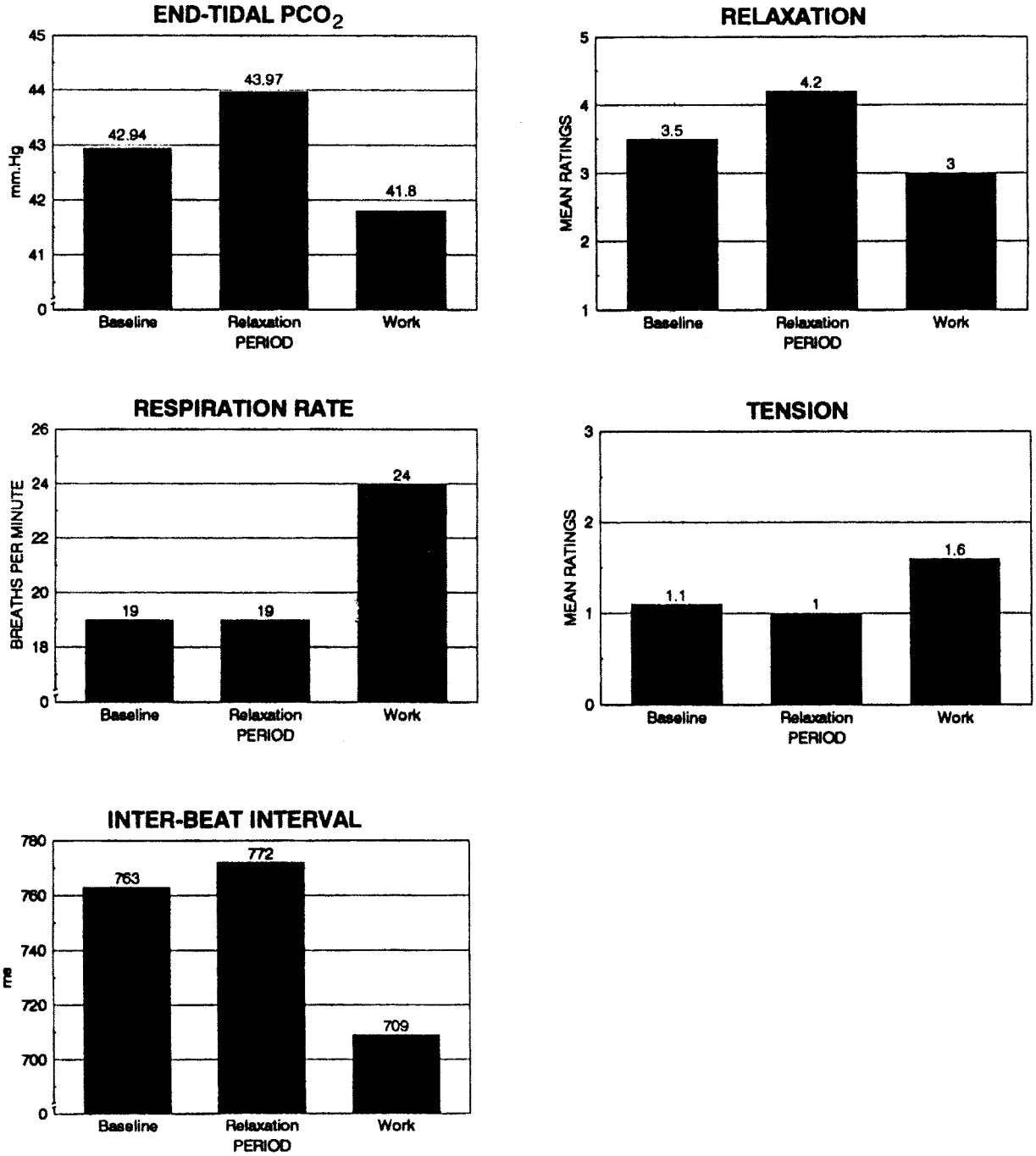


FIGURE 2. From "End-Tidal CO_2 as an Index of Psychophysiological Activity During VDT Data-Entry Work and Relaxation" by Schleifer and Ley [1994a]. Ergonomics 37:245–254. Prepared under US Government sponsorship. This article is in the public domain.

can *increase* as a result of either psychosocial stress or the metabolic demands of physical work (e.g., repetitive keying), thus inextricably confounding one effect with the other. While the metabolic demands of computer keyboard tasks are relatively light, several studies [Ruyssenaars, 1970; Mulder and Mulder-Hajonides Van Der Meulen, 1973a,b; Luczak, 1979; Lee and Park, 1990] indicate that even the metabolic load of finger tapping can significantly alter heart rate and heart rate variability.

End-tidal CO_2 holds promise of being a reliable and valid indicator of job stress. In this regard, it provides for an important methodological breakthrough in terms of evaluating the influence of psychosocial stress factors on muscle activity separate from that due to biomechanical stress

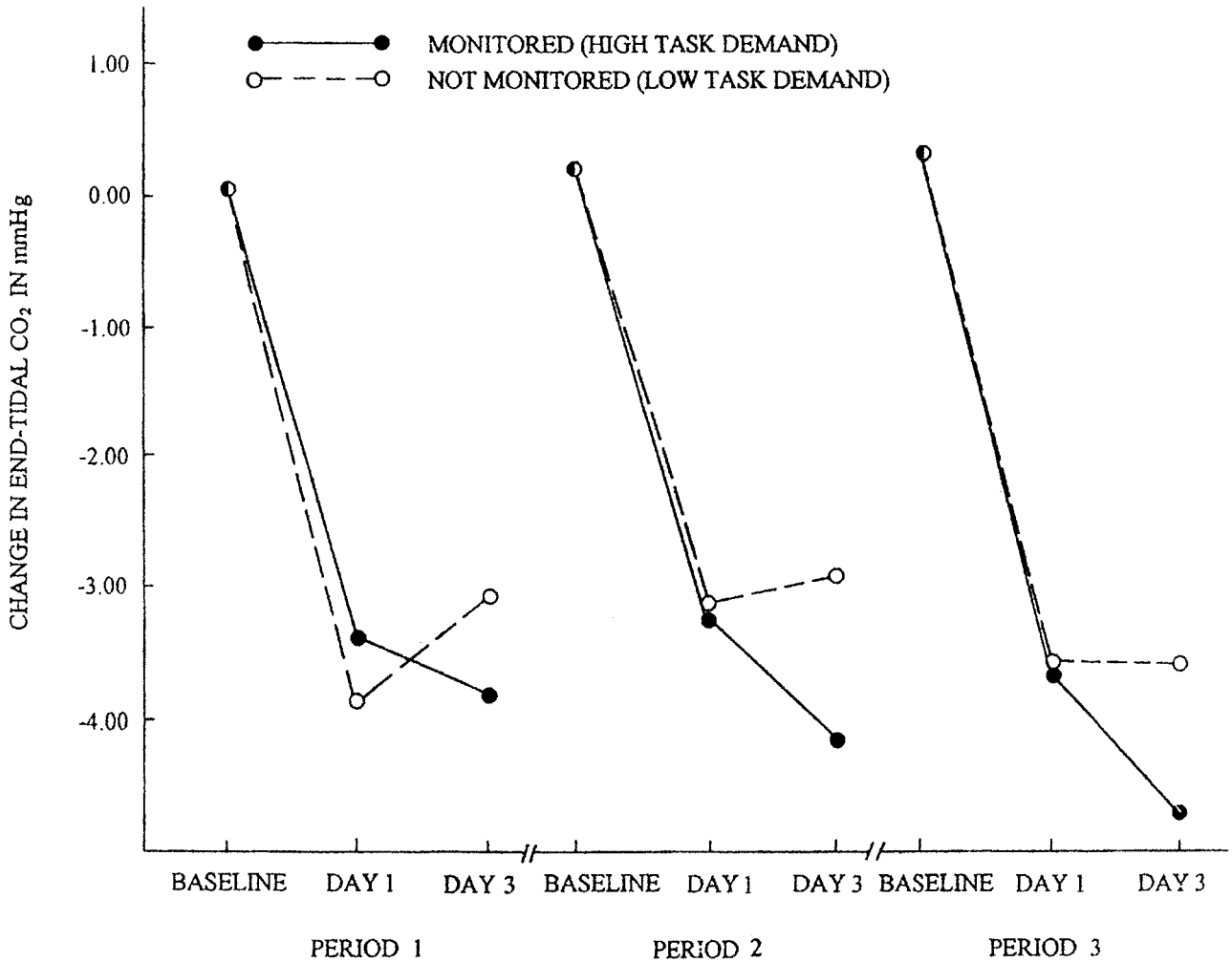


FIGURE 3. From "End-Tidal CO_2 as an Index of Psychophysiological Activity Under High and Low Data-Entry Workload Demands" by Schleifer and Ley [1994b]. Presented at the inaugural meeting of the International Society for the Advancement of Respiratory Psychophysiology, Saint Flour, France.

factors. This measurement capability allows for empirical testing and validation of a hyperventilation theory of job stress that postulates biobehavioral mechanisms in musculoskeletal disorders.

Another important advantage of end-tidal CO_2 as a physiological indicator of job stress is its high signal-to-noise ratio, or sensitivity, in comparison to other physiological indicators. For example, Schleifer and Ley [1994a] observed that under sustained, repetitive computer work, the mean/standard deviation ratio for end-tidal CO_2 was 19.62, while the mean/standard deviation ratio for the cardiac inter-beat interval was 7.94, indicating that end-tidal CO_2 is a more sensitive indicator of physiological stress loads than is the cardiac inter-beat interval. Recognizing the inherent variability of physiological measures, the high sensitivity of end-tidal CO_2 offers a clear advantage in the detection and quantification of psychosocial stress effects that have implications for musculoskeletal disorders.

A HYPERVENTILATION THEORY

The premise that hyperventilation induced by job stress contributes to the development of musculoskeletal disorders is based on the well-established effects of overbreathing [see Lum, 1976; Timmons and Ley, 1994; Guyton and Hall, 1996]. If breathing exceeds the metabolic requirements for oxygen due to psychosocial stress factors at work, hyperventilation (i.e., rapid rate of flow of CO_2) will occur. The resulting drop of carbonic acid in the blood produces respiratory alkalosis, i.e., a rise in plasma pH above 7.45 [Lum, 1976; Guyton and Hall, 1996]. As illustrated in Figure 4, this disruption in acid–base equilibrium triggers a chain of systemic physiological reactions that have adverse implications for muscle-tissue health.

At the cellular level, an immediate homeostatic reaction to relatively mild elevations in plasma pH (e.g., +.1) is the migration of CO_2 from intracellular to extracellular fluids.

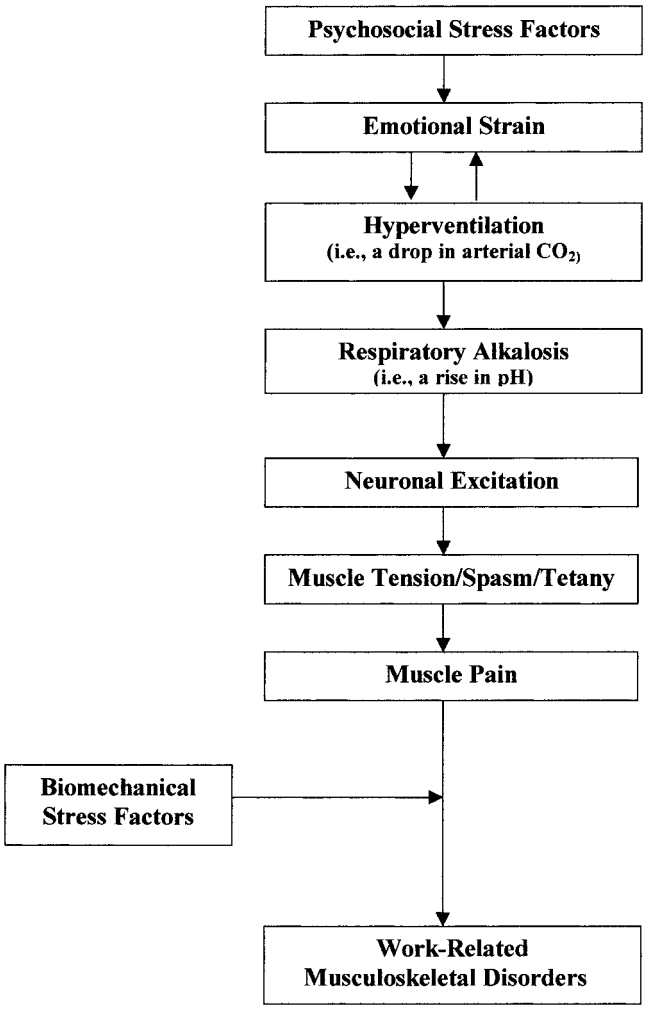


FIGURE 4. An illustration of salient variables in the pathway from stress-induced hyperventilation to work-related musculoskeletal disorders.

This causes a loss of CO_2 in peripheral motor unit neurons as well as in brain stem neurons (e.g., sympathetic neurons). Since neurons are highly sensitive to increases in pH, there is an increased likelihood of motor unit neuron depolarization or excitation [Guyton and Hall, 1996], thus increased muscle tension, muscle spasm, and amplified responses to catecholamines [Lum, 1976].

Perhaps the most dramatic example of the effects of overbreathing on the nervous system (i.e., loss of CO_2 in neurons) can be observed among individuals predisposed to epileptic seizures. A rise in pH of just a few seconds can cause an attack [Guyton and Hall, 1996].

Another immediate effect of ventilatory losses in CO_2 is vasoconstriction of the extremities. Peripheral vasoconstriction is either a direct response to elevations in pH (i.e., alkalosis) or sympathetic activation [Lum, 1976]. Vasconstriction reduces blood flow to the upper extremity muscles and increases the possibility of tissue damage due to a decrease in muscle tissue oxygenation and a buildup in metabolites as a result of sustained, repetitive work. The decrease in muscle tissue oxygenation (i.e., hypoxia) is exacerbated by the Bohr effect (i.e., decreased ability of hemoglobin to release oxygen due to a rise in pH). Paradoxically, the constriction-induced reduction in blood flow and muscle tissue oxygenation occurs when they are needed most, during sustained, repetitive work, and might, therefore, contribute to the development of work-related musculoskeletal disorders [Carayon et al., 1999].

If stress-induced hyperventilation persists, the resulting loss of CO_2 is compensated by kidney excretion of bicarbonate [Lum, 1976]. Prolonged, chronic renal excretion of bicarbonate (i.e., basic urine) to maintain normal pH re-establishes the acid–base equilibrium (pH 7.45), but at a new lower set point of CO_2 levels in the blood. However, if the loss in CO_2 increases due to a greater severity of chronic hyperventilation, loss of bicarbonate will result in a progressive lowering of the CO_2 set point at which acid–base balance is re-established. Over time, the loss of bicarbonate in the extracellular fluids critically reduces the alkaline buffering system below levels required to maintain everyday physical activities [Nixon, 1994]. Under these conditions, physical effort related to work, even that of light duty office tasks, compromises the body's ability to buffer the buildup of metabolic byproducts in muscle tissue. This results in an increase in fatigue, muscular aches, pains, and neuronal activity that might contribute to the development of musculoskeletal disorders [Nixon, 1995].

The systemic stress effects of hyperventilation are remarkably similar to those described in established theories of stress and disease causation. Notably, the effects of hyperventilation correspond with Selye's [1956] general adaptation syndrome (GAS) and the stages of alarm reaction, adaptation or resistance, and exhaustion. During the alarm reaction stage, there is excessive ventilation and a reduction in arterial CO_2 that produces respiratory alkalosis. The adaptation stage is characterized by a migration of CO_2 from the intercellular fluid to the extracellular fluids and the excretion of bicarbonate via the kidneys, which re-establishes the acid–base equilibrium at a lower CO_2 set point. During the third stage, the alkaline buffering system is severely diminished due to the renal excretion of bicarbonate. It is during this final stage of exhaustion that the worker is most vulnerable to both psychosocial and biomechanical loads that increase the risk of musculoskeletal disorders.

A study by Schleifer et al. [1997] of the cognitive and biomechanical stress effects of repetitive computer work has favorable implications for a hyperventilation theory of job stress and musculoskeletal disorders. Twenty-three workers performed a numeric data-entry task for three consecutive days, with each day consisting of six 40-min work periods. End-tidal CO_2, key force, and keystrokes per minute were

recorded on a continuous basis during each workday. Self-ratings of right-hand complaints were taken at periodic intervals.

Consistent with a hyperventilation theory, Figure 5 shows that there are regular increases in right-hand musculoskeletal complaints and corresponding reductions in end-tidal CO_2 concentrations across the work periods within each of the three test days. These results suggest that the cognitive stress of repetitive data-entry work is associated with right-hand musculoskeletal complaints.

With respect to biomechanical factors, Figure 5 shows that the consistent increases in right-hand complaints across the work periods within each of the test days were not associated with any pattern of increased key force or keystroke

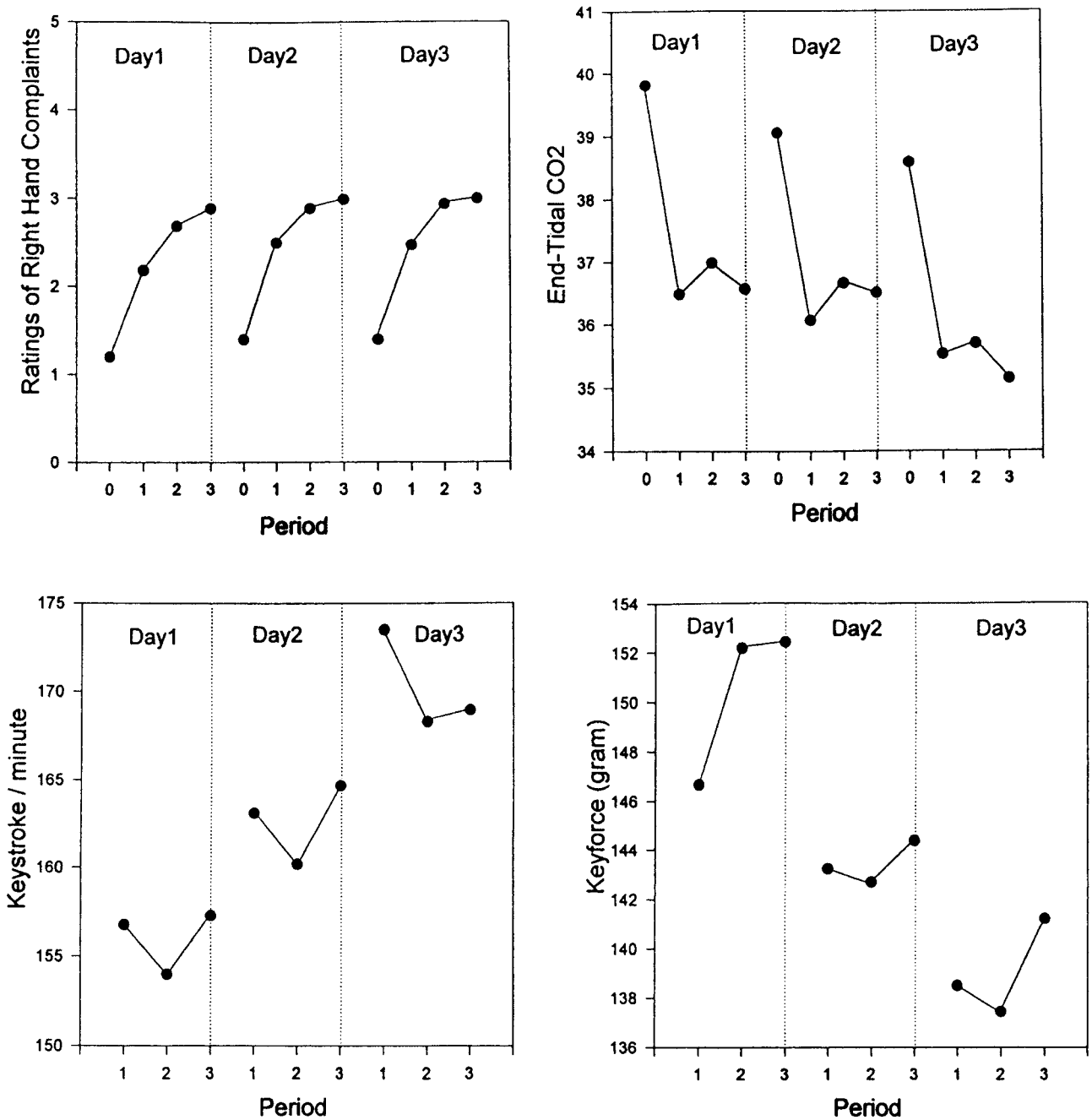


FIGURE 5. From "Breathing, Psychological Stress, and Musculoskeletal Complaints in VDT Data-Entry Work" by L.M. Schleifer, R. Ley, C. Pan, 1997. In: G. Salvendy, M.J. Smith, Richard J. Koubek, editors. Design of Computing Sytems: Cognitive Considerations, p. 545–550, Amsterdam: Elsevier. Reprinted with permission from Elsevier Science.

rates. In addition, the ratings of right-hand complaints were constant across the test days despite the fact that there were steady increases in keystroke rates and decreases in key forces. These results are contrary to conventional biomechanical models that postulate a positive association of key force and keystroke repetition with musculoskeletal problems of the hand and wrist.

JOBS STRESS, HYPERVENTILATION, AND NECK/SHOULDER DISORDERS

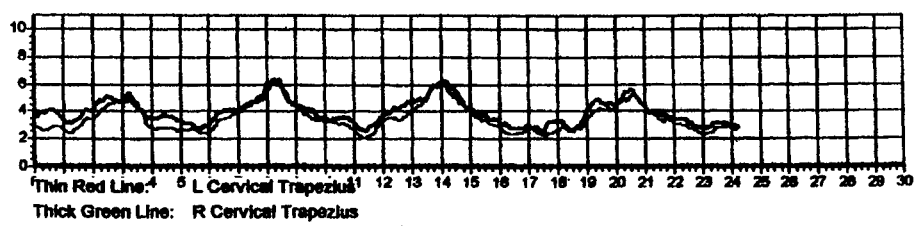
The neck/shoulder is a common body site for work-related musculoskeletal disorders. In fact, the evidence of a relationship between psychosocial stress factors and neck/disorders is more apparent than for other types of musculoskeletal disorders, including those of the hand/wrist [Bernard, 1997]. For example, a NIOSH field investigation [Hales et al., 1994] of computer workers in a telecommunications company found that of seven psychosocial factors, all were associated with neck/shoulder disorders, whereas only three were associated with the elbow and one with the hand/wrist. In addition, the odds ratios used in this study to measure the association between psychosocial factors and musculoskeletal disorders were greater for the neck/shoulder than for any other region of the upper extremity.

EMG studies also suggest that, under mental task demands, trapezius muscle activity is more evident than it is for most other body sites. Waersted and Westgaard [1996], for example, found that of 20 different muscle sites recorded, the level of EMG activity in response to cognitive tasks tended to be most pronounced in the frontalis and upper trapezius muscles.

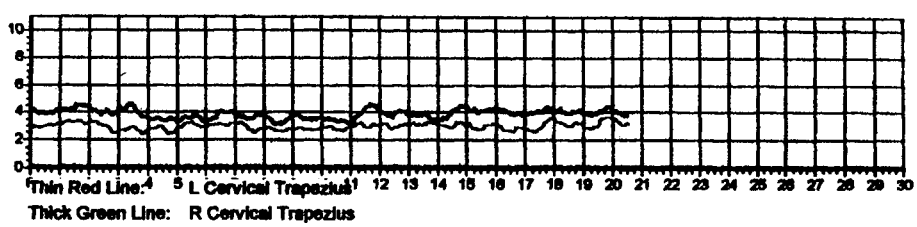
A plausible explanation for a differential influence of psychosocial stress factors on upper extremity disorders is that stress-induced hyperventilation imposes a biomechanical load that is specific to the neck/shoulder region. For example, the upper trapezius muscles are active during chest breathing, and they are quiescent during abdominal breathing [Cram, 2000]. As shown in the first panel of Figure 6, there was an undulating pattern of cervical trapezius EMG activity that corresponded with the respiration cycle in an individual who tended to be a chest breather. Cervical trapezius EMG activity increased during inspiration and decreased during expiration. The second panel in Figure 6 shows that the cervical trapezius EMG activity disappeared in the same individual following training in how to breathe abdominally.

Under normal conditions, inspiration and expiration are almost exclusively performed by the diaphragm [Guyton and Hall, 1996]. However, under stressful conditions, the pattern of ventilation shifts from diaphragmatic to thoracic or chest breathing [Naifeh, 1994]. The external intercostal muscles of the chest raise the rib cage during inspiration and the internal intercostal muscles pull the rib cage downward during expiration.

Ancillary neck/shoulder muscles are recruited in support of the intercostal muscles during the inspiration phase of chest breathing. These muscles include the sternocleidomastoid, which lifts the sternum upward; the scalene muscles, which lift the first two ribs; and the scapular elevators, which lift the collar bones and shoulder blades [Guyton and Hall, 1996]. Thus, breathing might be a biomechanical mechanism by which psychosocial stress factors differentially increase the risk of neck/shoulder disorders.



Cervical trapezius recording in a patient who tends to be a chest breather. The ancillary use of these muscles can be clearly seen in the four undulations.



Cervical trapezius recording in the same patient following training in how to breathe abdominally.

FIGURE 6. From "Electrode Atlas For sEMG" by Cram [2000]. Biofeedback 28:12–13. Reprinted with permission from J. Cram.

CONDITIONED HYPERVENTILATION

While occasional hyperventilation is a normal response to psychosocial work stressors, the harmful biobehavioral and biomechanical effects of overbreathing are most likely to occur under conditions of chronic hyperventilation, which result in diminution of the alkaline buffering system. Chronic hyperventilation refers to habitual overbreathing, even in the apparent absence of overt psychosocial work stressors. The question is: What is the process by which hyperventilation induced by job demands becomes a habitual breathing behavior?

Based on a classical conditioning model of breathing by Ley [1999], hyperventilation can become chronic through the modification of breathing behavior by means of Pavlovian conditioning. Consistent with this premise, clinical observations by Lum [1976] suggest that "With frequent repetition, the response [hyperventilation] takes on the characteristics of a conditioned reflex [to neutral stimuli]" (p. 198). In other words, hyperventilatory responses that initially occur only in response to specific job stressors can generalize to the contextual work environment through conditioning.

A study by Ley et al. [1996] demonstrates the Pavlovian conditioning process and provides experimental evidence that hyperventilation can be acquired as a conditioned response (CR). In this study, the unconditioned stimulus (UCS) consisted of counting backwards by sevens very rapidly and the conditioned stimulus (CS) consisted of a tone. Under the experimental condition, the tone was presented while the subjects performed the backward sevens task during four conditioning trials (CS + UCS). Under the control condition, the tone was presented but backward sevens counting was limited to reading (not computing) the subtraction problems (CS − UCS). End-tidal CO_2 was monitored to measure changes in ventilation.

Figure 7 shows that end-tidal CO_2 was lower during the four conditioning trials (CS + UCS) than it was when the

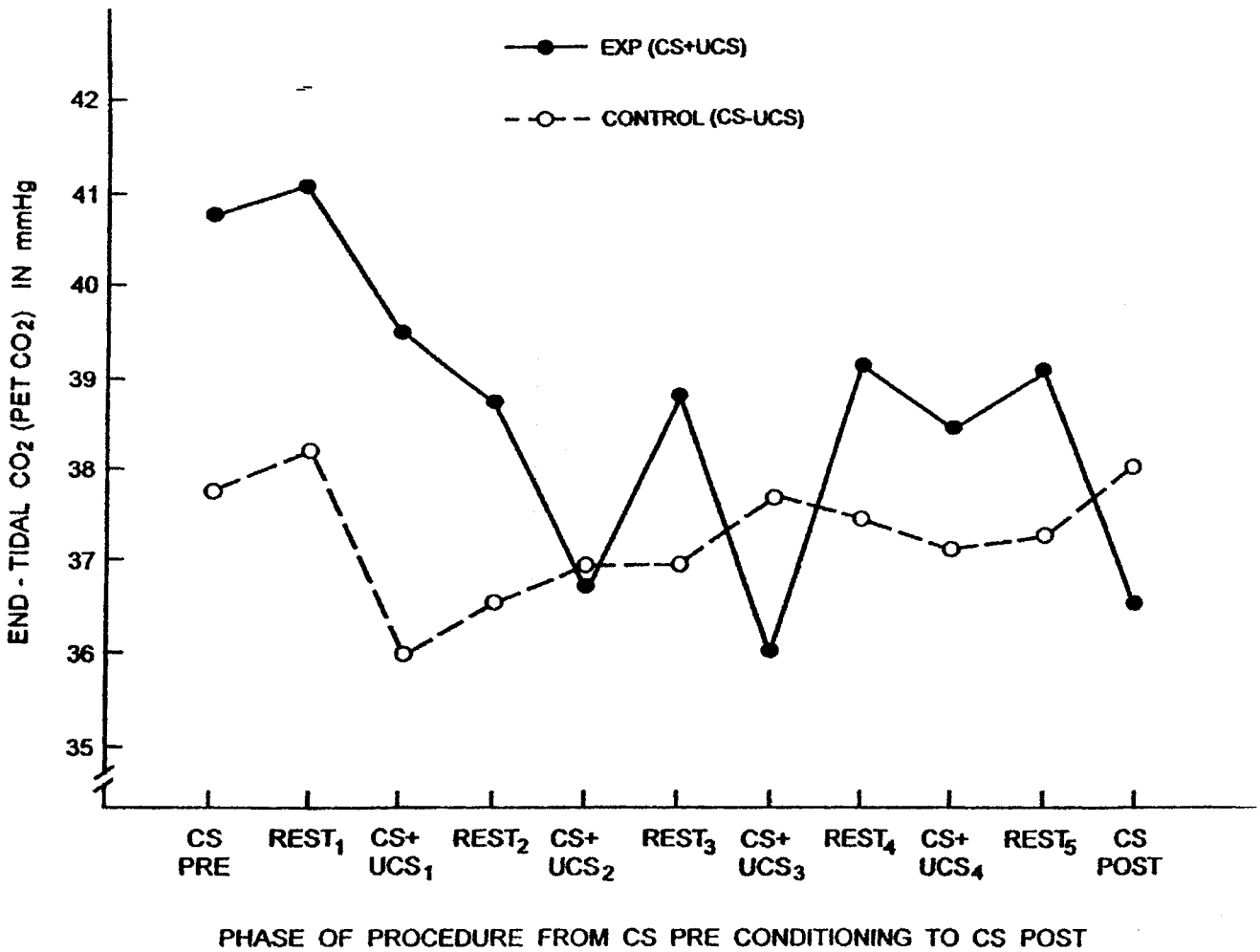


FIGURE 7. Based on data from "Pavlovian Conditioning of Hyperventilation" by Ley et al. [1996]. Psychophysiology 33 (Suppl):55.

tone was presented prior to conditioning (CS pre). End-tidal CO_2 also was lower during the CS + UCS pairings than during the rest periods that preceded each conditioning trial. Finally, end-tidal CO_2 was lower during the presentation of the tone following conditioning (CS post) than it was prior to conditioning (CS pre). This saw tooth pattern of increases and decreases in end-tidal CO_2 in the experimental group (CS + UCS), which was notably absent in the control group (CS − UCS), is evidence of conditioned hyperventilation.

In work settings, hyperventilation occurs, initially, as an unconditioned response (UCR) to intrinsically stressful stimuli (UCS), e.g., an abusive supervisor or high workload demands. If there is repeated exposure to these or other intrinsic work stressors, unconditioned hyperventilatory responses may become associated with otherwise neutral stimuli in the contextual work environment. In this case, a worker may hyperventilate as a result of merely sitting at the workstation, even when the abusive supervisor is not present. The neutral work environment becomes a CS that can elicit hyperventilation, a CR. This CR may then spread through the process of stimulus generalization to other cues in the work environment. In other words, hyperventilatory responses are not limited to the UCS that initiated the conditioning process.

In time, hyperventilation becomes pervasive; it occurs when a worker is sitting at the workstation, driving to work in the morning, or lying in bed at night thinking about the events of the workday. It is under these conditions that the deleterious effects of hyperventilation are most likely to occur.

RELATIONSHIP TO OTHER JOB STRESS MODELS OF MUSCULOSKELETAL DISORDERS

A hyperventilation model does not preclude the existence of other biobehavioral stress mechanisms associated with the development of work-related musculoskeletal disorders. Rather, the autonomic, biochemical, and neuroendrocrine mechanisms that are described in other models of musculoskeletal disorders [Sauter and Swanson, 1996; Smith and Carayon, 1996; Melin and Lundberg, 1997; Carayon et al., 1999; Feuerstein et al., 1999] appear to overlap and interact in a synergistic manner with those of a hyperventilation model. In this sense, hyperventilation is a fundamental component of fight-or-flight response [Cannon 1915], a primary causal mechanism in stress-related disorders.

A hyperventilation theory provides a unique biobehavioral explanation for how psychosocial aspects of work might contribute to the development of musculoskeletal disorders. It also provides an objective metric of job stress (viz., end-tidal CO_2) that lends support to the premise that psychosocial stress loads on the musculoskeletal system can be quantified. Thus, a hyperventilation theory enhances the biological plausibility of the ''job stress hypothesis'' in relation to musculoskeletal disorders.

It should be kept in mind that a hyperventilation theory is not an alternative to conventional biomechanical models of musculoskeletal disorders. Rather, the biobehavioral effects of overbreathing complement and potentiate the physical effects of force, repetition, and posture (see Fig. 4). This view is consistent with that of other models of musculoskeletal disorders that have integrated psychosocial and biomechanical factors [Sauter and Swanson, 1996; Smith and Carayon, 1996; Melin and Lundberg, 1997; Carayon et al., 1999; Feuerstein et al., 1999].

IMPLICATIONS FOR INTERVENTIONS

Recognizing that intrinsically stressful conditions cause hyperventilation, a primary intervention strategy in the prevention of hyperventilation and its potentially adverse effects on musculoskeletal health is to modify problematic job-design factors that give rise to psychosocial stress (e.g., lack of control, work overload). In this regard, Carayon et al. [1999] have asserted that work reorganization controls psychosocial stress by modifying how the work system is structured or organized, the manner in which workers are supervised, and the process or methods by which tasks are performed.

According to Smith and Sainfort's [1989] job design model, hyperventilation as well as other biobehavioral stress reactions are minimized when a balance exists among the interactive demands of the task, the organization structure, the technology, the physical and social environment, and the individual's resources and needs. A balanced work system removes or minimizes psychosocial stressors (UCS), which, in turn, extinguishes or weakens hyperventilatory responses (CR) to the contextual work environment (CS). In effect, if the sociotechnical work system is in balance, then breathing will be in homeostasis with the metabolic requirements for oxygen.

There are, of course, no ideal jobs, which are entirely stress-free. Imbalances between job demands and an individual's resources to cope will inevitably occur. Even job redesign efforts that succeed in the removal of psychosocial stressors will not always ensure extinction of conditioned hyperventilation responses to the contextual work environment.

As a result, it is useful to supplement work reorganization interventions with secondary stress-control interventions, such as relaxation training or breathing training. Relaxation training refers to any set of procedures such as progressive muscle relaxation or meditation that evoke inhibitory biobehavioral reactions [Schleifer, 1984]. Breathing training is another, perhaps more direct approach, to inhibiting hyperventilation and evoking relaxation by

shifting the pattern of respiration from thoracic to diaphragmatic breathing.

The benefits of relaxation training and breathing training are based on principle of reciprocal inhibition by Wolpe [1973]: ''If a response antagonistic to anxiety can be made to occur in the presence of anxiety-provoking stimuli so that it is accompanied by a complete or partial suppression of the anxiety responses [e.g., hyperventilation], the bond between these stimuli and the anxiety responses will be weakened'' (p. 17). Through a systematic desensitization process, hyperventilatory responses to the work environment can be counteracted or inhibited by the autonomic effects of relaxation training or breathing training.

Light physical exercise is another secondary stress-control intervention for stopping hyperventilatory responses to job stress. Walking or jogging controls hyperventilation by increasing CO_2 production and re-establishing the homeostatic balance between breathing and the metabolic requirements for oxygen. In this regard, rest breaks may be an especially effective intervention for reducing musculoskeletal discomfort in repetitive computer tasks when the worker gets up and takes a short walk away from the workstation [cf., Galinsky et al., 2000].

SUMMARY AND IMPLICATIONS FOR FURTHER RESEARCH

On the basis of established theories of stress and disease causation, various biobehavioral mechanisms have been postulated in an effort to explain how psychosocial factors of work organization contribute to the development of musculoskeletal disorders. A hyperventilation theory calls attention to the central role of breathing in mediating the biobehavioral effects of job stress. It is argued here that stress-induced hyperventilation amplifies the biophysiological load on the musculoskeletal system, and consequently, increases the risk of damage to tissue physiology. Based on the premise that the deleterious effects of stress can be controlled by preventing maladaptive breathing patterns, various interventions strategies, including work reorganization and individual stress management also were discussed.

The basis for a hyperventilation theory of job stress and musculoskeletal disorders is derived from an extensive literature on the biobehavioral effects of overbreathing. However, a definitive link to musculoskeletal disorders requires further research and evaluation. In this regard, an important attribute of the proposed hyperventilation theory is that it is subject to empirical evaluation.

For example, it is possible to test the hypothesis that self-ratings of psychosocial job stress and musculoskeletal symptom severity among office workers with neck/shoulder disorders will be higher when resting end-tidal CO_2 is low than when it is high. Based on the proposed hyperventilatory theory, the rationale for this hypothesis is that the lower the resting end-tidal CO_2 levels, the greater the propensity toward respiratory alkalosis, motor neuron depolarization, and muscle tension. A testable corollary hypothesis is that under stressful work conditions (e.g., high cognitive stress), trapezius EMG activity and musculoskeletal symptom severity will be higher among office workers with neck/shoulder disorders when resting end-tidal CO_2 is low than when it is high. Another testable hypothesis derived from a hyperventilation theory is that trapezius EMG activity and musculoskeletal symptom severity will be higher among officer workers with neck/shoulder symptoms when the mode of breathing is thoracic than when it is diaphragmatic.

The hypotheses described above involve unhealthy or symptomatic workers. However, a hyperventilatory theory is predicated on the assumption that breathing-related physiological changes precede the onset of musculoskeletal disorders among healthy workers. In this regard, it is possible to test the hypothesis that under high cognitive stress, the decrease in end-tidal CO_2 (i.e., increase in hyperventilation) and the increase in trapezius EMG activity will be greater than under low cognitive stress. These are a few examples of new research directions that would contribute to a better understanding of the relationship of job stress and breathing to musculoskeletal disorders.

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