

INDEX OF PAPERS and POSTERS

INTERNATIONAL SOCIETY FOR THE ADVANCEMENT OF RESPIRATORY PSYCHOPHYSIOLOGY FIRST ANNUAL MEETING 1994

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AN EXTENSIVE SUMMARY AND COMMENTARY OF THE 1994 CONFERENCE

was written by RONALD LEY, Ph.D.

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The Capnometer and Oximeter in the Biofeedback Treatment of Asthma and Emphysema

Robert Fried

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Biofeedback Clinic, Institute for Rational Emotive Therapy, NY, NY, USA

I have previously reported encouraging results of deep-diaphragmatic breathing training with PETC02 (capnometry) biofeedback to correct respiratory alkalosis in persons with chronic hyperventilation (Fried, 1987,1993). These results included the reduction of numerous symptoms ranging from dyspnea to anxiety, panic attacks, and even epileptic seizures.

More recently, they also include the symptoms of asthma and emphysema. I now routinely add oximetry, to monitor arterial blood O₂ concentration (SaO₂), as a biofeedback modality to the training protocol. In most cases, oximetry added to capnometry and, in some cases, thoracic EMG seem to be a particularly effective incentive to the patient to do the required work of deep breathing during training, because it shows virtually breath-by-breath progress in raising SaO₂.

The following cases illustrate the procedure and outcome: CASE 1: A 34-year-old married, professional, woman was in treatment for asthma for about 7 months when she experienced an attack in the office. Slide 1 shows (a) PETC02 and SaO₂ during the attack, (b) the effect of Albuterol inhalation on the breathing pattern, and on PETC02, and SaO₂, (c) these parameters at the initiation of self-regulation of breathing, and (d) successful self-regulation of deep-diaphragmatic breathing at about 3 breaths/minute.

The patient also suffered from multiple psychological disorders including depression, anxiety, panic attacks, and obsessive-compulsive disorder, which predictably fluctuated in severity with the degree of her asthmatic condition. It is important to note that her emotional symptoms have, in numerous citations in the clinical and research literature, been attributed to chronic cerebral hypoxia and disturbances of regional cerebral blood flow, a common feature in breathing disorders featuring erratic fluctuation in blood C0₂ levels.

CASE 2: A 67-year-old homemaker with severe emphysema was undergoing her first diaphragmatic breathing training session: Note, that (a) fluctuations in SaO₂ follow PETC0₂, and that (b) as breathing deepens, it slow down with SaO₂ rising from 92.9% to 95%, and PETC0₂ rising from 2.1% (16 torr) to 4.6% (35 torr).

This case also illustrates that hypocapnia may, in rare cases, be a neglected aspect of emphysema. But in most cases that I have seen, normocapnia prevails even in the face of extremely low breathing rates where respiratory acidosis should be expected to prevail.

Communications should be addressed to Dr. Robert Fried, 1040 Park Ave., New York, NY 10028-32, USA.

Psychosocial and Environmental Influences on Upper Respiratory Symptomatology and the Incidence of the Common Cold in Children Suffering from Asthma

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Previous research has shown that episodes of asthma in children are frequently preceded by upper respiratory infection. This study investigates the relationship between the occurrence of upper respiratory symptoms and infection in children suffering from asthma and the incidence of positive and negative life events and long term experiences and other psychosocial variables, as measured by semi-structured interview.

As part of another longitudinal study investigating the role of acute life events and long term experiences on the course of childhood asthma, a sample of 94 children aged 6 to 12 years, suffering from moderate to severe asthma, were selected at random from the current attenders at a Specialist Asthma Clinic, the Royal Hospital for Sick Children, Glasgow. 78 of these children were included as subjects in the current study.

Life events and long term psychosocial experiences were assessed by a baseline and two follow-up interviews carried out separately with the child and his/her main caregiver at nine-month intervals. Questionnaires on perceived self-competence/self esteem and anxiety were also administered.

A record of daily upper respiratory symptoms and the incidence of infection were recorded by subject pairs in a daily asthma diary and when appropriate, throat swabs taken and submitted for microbiological examination.

The one year period under study was analyzed and segregated into two further periods according to those days considered to be either low or high on the level of influence exerted by physical environmental triggers such as allergens. The pattern of symptom presentation within and between those periods was examined. The relationship between positive and negative life experiences and psychosocial factors, and the level of upper respiratory problems experienced by groups of these children was also examined. The results indicate a clear relationship between these variables.

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Influence of Neuro-Psychological Factors on the Pathogenesis of Bronchial Asthma

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We studied 61 pre-asthmatic patient (31 with neuropsychic pathogenic mechanism) and 27 asthmatic patients with the same mechanism. The methods used were: R. Cattell questionnaire and FES scale of R. X. Moos.

We consider the development of pre-asthma and bronchial asthma as a result of an interaction of the personal (the structural peculiarity of personality serving human adaptation) and micro social factors (the patient's family). The last ones under certain conditions may have the

pathogenic influence on the organism. Most probably a disease develops when a complex influence of decompensating factors on all the links of the system are taking.

The first factor in the pathological process is- "The Weak Point." Observations indicate that the asthmatic attack developed as a result of very strong emotional stress in the background of somatic well-being. In this case the involvement of biological factors became possible according to the decrease of protecting-adaptive function of the personality and related adaptive functions of the organism.

Usually, the influence of psychological factors develops gradually, according to such destructive processes as personal peculiarity of the parents, educational approach of parents, frequent parent's conflicts.

These families commonly have a very stiff hierarchic structure of interrelations, the dominating role of one of the parents (more often is of mother) and the formation of symbiotic connections between hyperwardship parent and the sick child. Such children more often developed neurotic disturbances: in conflict situations they reacted by pathological, destructive stereotypes with involvement of hormonal (hypothalamus-hypophysis adrenal system) or immune reactions. In life they had more neurotic, allergic and respiratory disturbances. Actualization of the personally significant conflict with combination of respiratory sickness and long emotional stress preceded the beginning pre-asthma or bronchial asthma.

The investigation of these patients in this period revealed differences between them and patients without the influence of neuro-psychic factors on the disease. The former were inadequate in the perception of the environment, more tense, had lower integration of "Ego", had more dependence on significant others; they were more compliant and trustful. In families, they were hard, symbiotically dependent, moral problems meant little.

Personal peculiarities of the patients with pre-asthma are closely connected to micro-social climate.

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Voluntary Control of Breathing in Exercising Asthmatic Subjects

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Reducing breathing frequency is a rehabilitation technique that has been used in this treatment of obstructive diseases for many years, although its effects are highly controversial.

In this experiment, we tested the hypothesis that asthmatic subjects may improve the efficacy of breathing, assessed by VE/V_{O2} and VE/C_{O2} , without detrimental effects on performance.

Sixteen young asthmatic subjects were randomly assigned to either Low Frequency Breathing (LFB) or Control groups. Each subject underwent two incremental exercise tests on a cycle ergometer with a 3 -min plateau corresponding to Heart rate of 150 bpm.

Between these two tests, the subjects underwent 9 training sessions. During these sessions, the LFB subjects learned to decrease breathing frequency by synchronising ventilation with pedalling with $TI/TE = 1/2$. Control subjects did not receive any instruction concerning breathing.

The results of the two tests showed that LFB had significantly decreased VE/V_{O2} , VE/VC_2 , and VD/VT ratio, without inducing significant changes in HR, HR/VP2 and dyspnea scores.

However, PCO_2 tended to increase in LFB subjects, and P_{O_2} to decrease, compared with Controls.

We conclude that voluntary decrease in breathing frequency may improve the efficacy of breathing, but it may cause a risk of hypoxia and hypercapnia. Further experiments should be done before recommending this technique in routine clinical practice.

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Lines, Numbers and Words in the Scaling of Dyspnea

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The use of psychometric scaling of the intensity of the sensation of dyspnea has become widespread in recent years and has helped in our understanding of both the neurophysiological basis of dyspnea and in the search for more effective therapeutic strategies in its clinical management.

Currently, the two main techniques used for scaling dyspnea are the Visual Analogue Scale (VAS - a straight line the ends of which represent the extremes of dyspnea) and the Borg scale (a scale comprising verbal descriptors and numbers).

The aim of the present study was to compare the utility of the essential components of these two scales in scaling dyspnea.

Nine healthy subjects performed stair climbs at three different levels (1,3, and 5 flights). At the end of each climb, they were asked to scale the intensity of their breathlessness (dyspnea) using either a VAS a simple number (between 0 and 10) or a verbal descriptor (based on those in the Borg scale). Each type of scaling was performed twice for each level of stair climb.

The study was performed over 6 experimental sessions within 2 weeks. All scales were reproducible on a test-retest basis. Intensity levels were generally comparable across the different scales although at light intensity work (1 flight of stairs) lower scores were obtained with the VAS compared to numbers and verbal descriptors. All scales were sensitive at detecting differences in the intensity of dyspnea reported at the different levels of exercise although the VAS performed better than the other scales in this respect. Finally, this study clearly showed that the relative "intensity value" of some of the verbal descriptors used was quite different from their position on the Borg scale.

This study shows that the intensity of dyspnea can be reliably scaled with the various types of scaling modalities and suggests that the use of simple numbers could prove valuable in a clinical setting.

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Automaticity and Specificity of the Ventilatory Changes Induced by Mental Images

Sonja Denot-Ledunois and Jorge Gallego (1)
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The first aim of this study was to determine whether ventilatory changes caused by mental images of exercise are automatic or whether they are part of the voluntary strategies used by the subject to facilitate mental imagery. The second aim was to study whether the ventilatory changes observed during imaging of exercise are specifically related to the content of mental images.

Thirteen swimmers and 16 judokas, all young well-trained athletes, participated in one session during which they performed mental images of an actual past competition. The subjects were first trained to perform mental images and their attention was deliberately oriented toward non-respiratory aspects of the imagined situation.

The three successive images concerned (1) the Observation of the environment, (2) the Preparatory phase, and (3) the Exercise phase. TTOT consistently decreased over these phases, whereas VT displayed a transient increase during Preparation.

Breathing parameters were not significantly different between swimmers and judokas, but heart rate was significantly lower during exercise in judokas, a difference which remember the actual situation.

Post-experimental questionnaire showed that most subjects had felt that their breathing had changed during the experiment, but all but four subjects reported that they had not produced these changes voluntarily. In addition, the exact nature of these changes was imprecise in all the subjects.

We concluded that mental images of exercise may cause genuine automatic changes in breathing, but it is unclear whether these changes are specifically linked to the content of mental images, rather than to non-specific factors such as arousal, mental effort, or emotions.

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End-tidal PCO₂ as an Index of Psychophysiological Activity Under High and Low Data-Entry Workload Demands

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The utility of end-tidal PCO₂ (peak concentration of carbon dioxide in a single breath of exhaled air) as an index of psychophysiological activity under high and low workload demands was assessed among forty-seven data-entry workers.

Workers assigned to the high workload condition were expected to meet data-entry performance standards of > 200 keystrokes per minute and < 6 errors per minute. Computer monitoring and feedback of keystroke and error rates were used to induce compliance with these standards.

Workers assigned to the low workload condition were not expected to meet any performance standards and there was no computer monitoring or performance feedback.

End-tidal PCO₂ and heart rate were monitored on a continuous basis over three consecutive workdays. Planned comparisons across the workdays indicated that the decrease in end-tidal PCO₂ was greater under the high workload condition than under the low workload condition, indicating an increase in psychophysiological stress. There were no differences in heart rate or heart rate variability between the two workload conditions.

These findings indicate that end-tidal PCO₂ can discriminate psychophysiological stress effects of different data-entry workload demands, and that end-tidal PCO₂ is a more valid index of these effects than are conventional indicators of cardiac activity.

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Pavlovian Conditioning of Dyspneic-Fear in the Rat

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The purpose of the present study was to attempt to establish experimental evidence for the Pavlovian conditioning of dyspneic-fear, i.e., fear, that accompanies uncontrollable suffocation/asphyxia.

Eight rats were subjected individually to a five-minute period in an atmospherically controlled chamber permeated by the odor of vanilla (conditioned stimulus-CS). During the last 30 seconds of the five-minute period, the chamber was infused with 100% CO₂ (unconditioned stimulus-UCS).

Twenty-four hours following this single conditioning trial, four of the eight subjects were placed in the conditioning chamber for eight minutes with the CS (conditioning context) but without the UCS; the remaining four were placed in the chamber without the CS (neutral context).

For the purpose of determining the effects of the CS (odor of vanilla in the conditioning context) independent from the UCS, a third group of four rats was placed in the test chamber in the presence of the CS but absence of UCS.

The subjects that received the paired presentation of the CS and UCS on the previous day (100% CO₂ preceded by the odor of vanilla) displayed a significantly lower rate of ambulatory behavior (more eight- second periods of "freezing-no observable movement) compared with the group tested in the neutral context (no UCS) and the group tested in the conditioning context (no prior exposure to the CS or UCS).

Since the inhibition of behavior ("freezing") is a manifestation of fear, these results support the hypothesis that dyspneic-fear can be acquired through Pavlovian conditioning procedures.

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Breathing and Evaluation of Somatic Pain: Significance of Instant Measurement of Inspiratory Flow

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P. Cros

Faced with the polymorphism of painful states (somatic, neurogenic, psychogenic), an easily measured physiological parameter, which would give both a quantitative and proportional indication of the intensity of pain, would be of significant interest.

The widely spread out nature of cortical projections are probably the reason for the absence of proportionality in the amplitude of components P40 and P100 of somesthetic potentials. This absence has shown the need to avoid repetitive and identical test stimulations in the search for a pain threshold. However, electrodermal reaction is both reliable and stable.

Used in a protocol including a perception-motor task, this has allowed us to determine the pain threshold by the stimulation of the median nerve at the wrist (threshold: around 10mA; stimulation duration: 1 ms; n = 5 subjects), and to differentiate moderate pain (at 10 to 15 mA) from acute pain (at 15 to 27 mA) in a linear relation of the electrodermal reaction amplitudes as a function of the strength of stimulation ($r = 0.995$; slope 0.480).

With these stimulation conditions, the variations of the respiratory frequency have not correlated with the strength of the pain stimulation.

However, the use of a pneumograph (instant respiratory flow), not using perception/motor cuing illustrates that the amplitude of inspiration increase significantly when the stimulation at the median nerve is felt as clearly painful (15 mA) as compared to non-painful infralimnar stimulations.

This constant increase ($m = + 48\% \pm 5\%$) of the instant inspiratory flow in a clearly painful situation, demonstrates the interest of instant and differential methods of evaluation of pain as compared to methods using data such as respiratory frequency per minute where variations can have a multifactorial origin.

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Occupational Exposure to Air Pollution and Reduced Lung Function

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Cigarette smoking is an important determinant of respiratory disease. Occupational exposure to air pollutants (particularly from motor vehicles) and their association to impaired lung function is still controversial.

Garage mechanics are exposed to a wide range of noxious substances, such as sulphur dioxide, nitrogen dioxide, hydrocarbons and asbestos.

21 male garage mechanics (7 non smokers and 14 smokers, mean age 29.5 (\pm) SD 8.3 years) and 22 male controls (10 non smokers and 12 smokers, mean age 28.8 (\pm) SD 7.3 years) from an inner city area were investigated to see if there was any relationship between working in a garage and reduced lung function.

The mechanics have been working in a garage environment for 122.4 (\pm) SD 7.8 years. The mean cigarettes smoked/day by mechanics was 15.9 (\pm) SD 7.6 and by controls was 14.8 (\pm) SD 7.5.

The lung function tests included the Forced vital capacity (FVC), Forced expiratory volume in 1 second (FEV) and Peak Expiratory Flow (PEF), measured on site using a Vitalograph and Wright peak flow meter.

Garage mechanics showed lower mean values of lung function compared to controls. Smoking mechanics also showed lower mean values compared to smoking controls, but non-smoking mechanics showed no significant difference in lung function compared to non smoking controls.

We conclude that cigarette smoking and occupational exposure to air pollution together result in a significant reduction of lung function.

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Anxiety mimicking or complicating asthma in children: a cognitive behavioural approach
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Abstract Pending

The Relationship Between FEV1 and End-Tidal PCO2 Following Inhaled Antigen Challenge

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The association between the severity of asthma and hyperventilation (HV) has been well described (McFadden ER, Lyons HA. NEJM 1968;278:100027:1027-1032). Less is known about the relationship between the degree of bronchoconstriction and end-tidal PCO₂ (PETCO₂) in both the early and delayed bronchoconstrictor phases following challenge with inhaled antigen.

We studied this in 11 mild atopic asthmatics (FEV₁ 80% pred., age range 21-43 yrs) who had previously demonstrated a delayed bronchoconstrictor response with a fall in FEV₁ of more than 20% below baseline following the inhalation of house dust mite antigen extract (DerPI). FEV₁ was recorded every 5 minutes for 20 minutes following antigen challenge, then at 10-minute intervals for a further 40 minutes and thereafter half hourly for up to 12 hours post inhalation.

A rapid response infrared CO₂ analyser was used in association with a computerized data acquisition system to measure and record PETCO₂ continuously, breath by breath and in real time, for the duration of the study.

Mean pre challenge baseline PETCO₂ was 35.2 (SD = 4.4) mmHg. During the early response, FEV₁ decreased by an average of 24% below baseline with a significant correlation between FEV₁ and PETCO₂ in 5 (45%) subjects (range: $r = 0.44$, $p < 0.05$, to $r = 0.82$, $p 0.001$) with a PETCO₂ nadir below baseline of 5-10mmHg in 4(36%) subjects and > 10 mmHg in another 3(27%) subjects. During the Delayed response, FEV₁ decreased by an average of 55% below baseline. This was accompanied by a fall in PETCO₂ by 5-10mmHg in 3(27%) subjects and > 10 mmHg in 1 (9%) subject.

There was a significant correlation (range: $r = 0.44$, $p < 0.05$, to $r = 0.89$, $p 0.001$) between PETCO₂ and FEV₁ in 6(55%) subjects and no correlation in the remaining 5. Four subjects in whom a significant fall in PETCO₂ occurred during the early response did not hyperventilate during the delayed response and conversely one subject with hyperventilation during the delayed response had no fall in PETCO₂ during the early response. Three (27%) subjects had no evidence of hyperventilation in either phase.

These results show that both acute and delayed response to antigen challenge is associated with ventilatory stimulation and hypocapnia in some but not all subjects. The lack of association between acute and delayed responses suggests the possibility of different mechanisms for hyperventilation in the two phases.

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Controlled Outcome Studies of Breathing Retraining-based Treatments for Hyperventilators

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Breathing retraining is often a major component of the treatments for hyperventilators and asthmatics currently offered by physiotherapists, psychologists, breathing therapists, physicians and other health care professionals. The cumulative clinical experience of these therapists has indicated to them the effectiveness of such training.

While it is difficult to estimate the contribution of breathing retraining relative to other components of treatment, such as counselling, there are now several controlled outcome studies which support the clinical observations.

These studies are reviewed in tabular form with identification of the following factors: clinical populations; number of subjects; age, sex, medications of subjects; method of breathing retraining; duration of breathing retraining; qualifications of trainers; other components of treatment; type of controls; measurements; results; and follow up studies.

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A New Device for Pulmonary Rehabilitation Based on Visual Feedback

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This device is aimed to allow patients to practice ventilatory movements with the help of an intermitten visual feedback.

The device includes a jacket, a signal processing system and a common PC. Ventilatory movements are recorded using the principle of respiratory inductance plethysmography. A special jacket without sleeves made of a material with a texture allowing only horizontal wiredrawing includes the two coils of the inductance plethysmograph. Four sizes have been made to get the most close fitted jackets. Tests have been performed to assess the signal variability, it was less than 10% from one day to another. "The jacket is plugged to the signal processing system and the signal digitisation is performed with an ADC card inserted in a common PC. A computer program usable in physiotherapist office has been developed.

The main phases of this program are (i) to process on line the respiratory inductance plethysmography signals for recording and working sessions, (ii) to build a target the patient has to follow during the working sessions, (iii) to provide a visual feedback by simultaneously displaying the target and the actual breath and (iv) to calculate breath-by-breath the performance.

The target is a selected breath with an envelope which is a tolerance zone surrounding this breath. The latter has been selected among the patient's spontaneous breaths to take into account the individuality of breathing pattern.

The performances of a working session are given in terms of scores on the whole breath and on the inspiratory and expiratory phases.

In the course of a session, the patient wearing the jacket sits on a chair, his arm on his leg; he remains at rest for few minutes in order to reach a ventilatory steady state.

The therapist observes the signal on the monitor and records 30 seconds of ventilatory motions when a steady-state is reached. From this recording he selects a breath either automatically or manually with the PC's mouse. The target is then built and the patient may now start the exercise: he is asked to perform ventilatory movement which should be inside the envelope as he can see simultaneously on the monitor the envelope and his actual breath. In addition, this visual feedback is intermittent; i.e. ten cycles during which the patients ventilatory movements are displayed followed by 5 breaths during which only the target is displayed and the patient is asked to imagine his breath inside the envelope. The working session is operated over 105 breaths.

This new device is non-invasive and the computer program is user friendly. Further development will consist in designing a device for practicing pulmonary rehabilitation at the patient's home under the control of a regularly visited therapist.

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The Role of Respiration during VDT Work: Implications for Repetitive Strain Injury

Erik Peper, San Francisco State University

Repetitive strain injury (RSI) among video display terminal (VDT) operators threatens to inflict individuals with illness and overwhelm corporations with increasing medical costs and lost productivity. The Center for Disease Control estimates that 15-20% of computer operators are at risk

This panel explores within a systems perspective what respiration changes occur during VDT work, what possible mechanisms (such as attention and vigilance) cause respiratory changes, what components related to respiratory changes may increase the risk of repetitive strain injury, and what respiratory interventions may prevent, reduce, or ameliorate the risk of the occurrence of RSI.

Participants:

Overview: A systems perspective of RSI associated with VDT work.

Erik Peper, Ph.D. Chairperson, San Francisco State University

The role of body position and breathing patterns.

Erik Peper, Ph.D.

Respiratory rate and upper thoracic electromyographic changes during keyboard data entry: Implications of RSI.

Erik Peper, Ph.D. San Francisco State University

Relationship between end-tidal CO₂ and heart rate during VDT work.

Ronald Ley, Ph.D. University at Albany, State University of New York

Stress effect of electronic data monitoring during VDT work.

Lawrence Schleifer, Ph.D. National Institute for Occupational Safety and Health

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The Effect of Volitional Hyperventilation on Postural Sway

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One of the possible consequences of hypocapnic hyperventilation could be a reduction in cerebral blood flow leading to tissue hypoxia and CNS depression.

To test this hypothesis we investigated the effect of hyperventilation on postural sway.

Nine subjects, performed two tests, "+C02" and "-C02," 20 min apart. Subjects stood on a Mills-McIntyre postural swaymeter breathing through a mouthpiece while wearing a noseclip. The "-C02", test consisted of 4 min normal breathing (Rest), followed by hyperventilation for 3 min or until their Pet, C02 fell below 25 mmHg. Subjects then breathed normally for a further 4 min (Post-Hyper).

Subject's postural sway was measured at 1,2 and 3 min of rest, 1 and 2 min of hyperventilation, and 20 sec, 1 min and 3 min post-hyper. The "+C02 test was the same except that during hyperventilation Pet, C02 was maintained near its normal level.

VE was significantly lower 1 min and 3 min post-hyper than rest in "-C02" test. Due to the relative hypoventilation, all subjects demonstrated a marked drop in Pet O2 following hyperventilation in arterial gas tensions and the variations in movement associated with breathing at different tidal volumes, postural sway did not vary significantly throughout the test.

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Are Respiratory Diseases a Predisposing Factor in Panic Disorder?

Kees Verburg (1), Eric Griez, Henk Pols, and Jaap Meijer
The Netherlands

Previous research has shown that panic disorder is more prevalent in pulmonary patients than in the general population. In a file study we showed that panic patients had a higher childhood and life-time prevalence of respiratory diseases than psychiatric controls. The present study is a follow-up on this file study.

150 consecutive anxiety patients filled in a specially designed questionnaire, which asked for the occurrence of respiratory and other somatic disorders before the onset of their anxiety disorder. The sample was divided into 82 panic disorder patients and 68 other anxiety patients serving as controls.

Panic disorder patients also filled out a panic symptom list (PSL III-R, consisting of the 13 DSMm III-R panic symptoms, scores for each symptom ranging from 0 to 4, the total symptom score ranging from 0 to 52), with the instruction to score the symptoms of an average panic attack.

Panic patients had a significantly higher prevalence of respiratory diseases before the onset of their anxiety disorder than the controls (42.7% vs. 16.2%). This higher prevalence was mainly due to a higher prevalence of bronchitis (26.8% vs. 8.8%).

Prevalence of other respiratory disorders was also higher in panic disorder patients, but these differences did not reach statistical significance.

These differences in prevalence appeared not to result from a tendency to hypochondria, as there were no differences in reporting other diseases, neither between panic disorder patients and controls, nor between panic patients with and without a history of respiratory disease.

Panic disorder patients with a history of respiratory disease did not have more severe respiratory symptoms during panic attacks than panic patients without a history of respiratory disease.

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Somatic Changes in the Hyperventilating Subject: An Osteopathic Perspective

William Garland, London, UK

In transient or persistent hyperventilation, alteration of the breathing pattern brings about changes in the musculoskeletal mechanics of the breathing apparatus.

This tends towards a pattern of considerably reduced diaphragmatic ability with a commensurate restriction of the lower rib cage and an increasing dependence on the upper rib cage and accessory muscles of respiration:

- (a) Prolonged contraction of the diaphragm,
- (b) Loss of lower rib excursion,
- (c) Visceral stasis pelvic floor weakness,
- (d) Abdominal/erector spinae muscular imbalance,
- (e) Fascial restriction from the central tendon via the pericardial fascia to the basi-occiput,
- (f) Upper rib elevation with increased costal cartilage tension,
- (g) Thoracic spine segmental dysfunction and subsequent sympathetic disturbance,
- (h) Accessory muscle hypertonia and fibrosis,
- (I) Promotion of a rigidity in the cervical spine and the formation of a fixed lordosis in the low cervical spine, and
- (j) Reduction in mobility of the 2nd cervical segment causing disturbance in the vagal outflow.

Physically and physiologically this configuration runs against a biologically sustainable pattern, and, in a vicious cycle, abnormal function (use) alters normal structure, which disallows return to normal function.

Assistance can be given to individuals who hyperventilate by minimizing the effect of the somatic changes that occur and by trying to maintain within the structures an ability to change. In this way therapeutic intervention by breath retraining and counselling will be able to be effective.

It is the role of the osteopath to examine the relationship between the structures and their function, both demanded and idealized, and in hyperventilation, where psychology overwhelms physiology, their role can be very beneficial.

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Panic Disorder and Hyperventilation: What are the Signs for Hyperventilation at Rest?

Nola Rushford, Royal Melbourne Hospital

Hyperventilation (HV) could contribute to panic disorder (PD)

(a) as a normal component of the anxiety response, by contributing to the severity of symptoms during an attack;
(b) through acute hyperventilation, by causing symptoms leading to a panic attack or
(c) as the major aetiological disorder, the hyperventilation syndrome (HVS), with

(i) possible chronic hyperventilation precipitating attacks easily or
(ii) by causing a shift in sensitivity of the central CO₂ chemoreceptors against which arterial PCO₂ is set.

Previous research by the present investigator had shown that (c)(ii) was not applicable, as CO₂ sensitivity was normal in PD; (c)(i) was partially eliminated because HV provocation, under controlled conditions, did not cause a greater than normal frequency of panic attacks.

The present study continued investigating (c)(i), by observing respiration at rest in PD.

Presumptive signs of resting hyperventilation (respiratory patterns) and confirmatory signs (partial pressure of end-tidal carbon dioxide [PETCO₂]) were compared in 34 patients with PD and 42 normal control (NC) subjects.

Mean(sd) resting respiratory cycle time and its variability, amplitude variability, and PETCO₂ [NC = 39.0(5.7)mm Hg, PD = 38.3(9.0)mm Hg; $p = .66$] were similar.

Numbers of subjects with at least 1 value beyond the 5th or 95th NC percentiles were similar [NC = 16%, PD = 26%; NS].

The results suggested that hyperventilation at rest in the study group of PD patients was not significant.

What needs to be tested now is ambulatory monitoring of PCO₂, to determine the extent of involvement of HV in conditions (a) and (b), above.

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Is Resting Respiration Different from Normal in Panic Disorder?

Nola Rushford, Royal Melbourne Hospital

Patients with panic disorder (PD) complain of respiratory symptoms and are often seen to be breathing with excessive energy. The present study compared resting respiratory traces of the upper and lower thorax in 34 PD subjects and 42 normal control (NC) subjects.

The only significant difference was in the "stress response", the ratio of inspiratory time/expiratory time (ti/te) at the diaphragm (NC == 0.57(0.14), PD == 0.69(0.21), $p = .01$). The ti and te patterning had differential respiratory effects in each group, however: (a) the strongest determinant of PetCO₂ in the NC group was diaphragmatic ti [rNC = .327, $p = .017$, rPD = .015, $p = .356$; SD, $p = .050$] and in the PD group was upper thoracic ti/te [rNC = .030, $p = .43$; rPD = .596, $p = .001$, SD, $p = .000$], and (b) increases in ti/te were marked by relative increase in ti in NC and relative decrease in te in PD.

They were differentially related to anxiety: (a) state anxiety, controlled for age and vital capacity, was inversely related to regional ti and te in PD but not in NC [for example, rNC = .006, $p = .49$; rPD = .405, $p = .043$, SD, $p = .035$, for upper thoracic ti and (b) trait anxiety was related to ti when controlled for state anxiety, age and vital capacity [rNC = .004, $p = .471$; rPD = .719, $p = .001$, SD, $p = .000$ for the diaphragm].

PetCO₂ decreased with increasing trait anxiety in the PD group only [rNC = .039, $p = .13$; rPD = .388, $p = .051$, SD, $p = .033$].

The differential cross group relationships between anxiety, PetCO₂ and the respiratory trace were marked and, although there was little evidence for hyperventilation at rest in PD, it is apparent that the respiratory adjustments to maintain PetCO₂ that normal individuals use in the face of anxiety were not available to the PD subjects.

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Recovery from Voluntary Hyperventilation in Panic Disorder

Nola Rushford, Royal Melbourne Hospital

Recovery over three minutes was monitored after up to three minutes of voluntary hyperventilation (VHV) with PetCO₂ at 20mmHg, in 34 PD patients and 42 normal control (NC) subjects.

Time spent hyperventilating; PetCO₂, hyperpnoea and apnoeic periods during recovery, and relationships to psychological measures were examined.

Subjects' traces were categorized according to PetCO₂ at baseline (a) PetCO₂>35mmHg [Category A; nNC = 33, nPD = 22]; (b) PetCO₂ within 30-35mmHg [Category B; nNc = 7, nPD = 9, and (c) PetCO₂<30mmHg [Category C - definite hyperventilation; nNc = 2, nPD = 3]. Time VHV (mean(sd)) was 174 (18)sec and 141(52)sec (p = .032), forNC and PD, respectively 10 PDA and 2 NCA, one NCB and one PDB and all 3 PDc failing to complete 3min [$\chi^2 = 10.6$, p = .000]. In PD, time VHV was inversely related to state anxiety [rNC = .061 p = .350; rPD = .465, p = .003, SD, p = .010].

Over recovery, two of the remaining 12 PDA and two of the 32 NCA subjects met Hardonk and Beumer's criterion for hyperventilation, failure to recover 65% baseline PetCO₂; all others had normal recovery.

In the first recovery minute, 12 of PAA were hyperpnoeic (f.20cpm), and in the third, 7 PDA but no NCA [p= .000]. Apnoea (breath-holding>5sec) occurred in 53% ofNC and 22% ofPD subjects [$\chi^2 = 6.33$, p = .011], 22/3 of apnoeic episodes being inspiratory. How unpleasant the PD subjects felt after recovery depended strongly on PetCO₂.

In summary, anxiety determined the performance of PD subjects during VHV, while PetCO₂ determined how they felt afterwards. The majority of NC subjects, but only a minority of PD, had inspiratory apnoea during recovery, an anomaly that may indicate a problem of respiratory control.

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Hyperventilation-Apnea Syndrome: A New Clinical Entity?

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We studied 13 patients with hyperventilation syndrome (HVS) (1st group), 7 patients with bronchial asthma (2nd group) and 10 healthy subjects of the same age and sex (3rd group). The criteria of Hardonc and Beumer (1979) for the diagnosis of HVS were used.

A polygraphic sleep recording was undertaken with special analysis of respiration. The number of pathological apneic episodes as well as the coefficient of variation of inspiration time at sleep stages I and II were significantly increased in the 1st group with respect to the 2nd and 3rd groups.

Special analysis of apnea after body movements revealed a predominance of apnea in HVS patients in all sleep sages. This predominance was most significative in the 1st group during deep sleep.

Our data presents HVS as a major problem linked to sleep apnea. HVS might be used as a marker of sleep apnea. It is possible that the combination of waking HVS and sleeping apnea can be clearly demonstrated and future research may show this to be a clinical entity.

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Changes in ventilation, arterial potassium and CO2 during moderate exercise following volitional hyperventilation

J.H. HOWELL, B.A. CROSS, S.G. SEMPLE

Abstract pending

Voluntary Control of the Diaphragm

L.C.Lum, Cambridge, U.K.

Some workers maintain that one cannot voluntarily control diaphragmatic contraction. Simultaneous records of intra-thoracic and intra-abdominal pressures demonstrate that intra-abdominal pressure can be varied at will by voluntary contraction and relaxation of the diaphragm.

PANIC and PHOBIA: Of 701 cases of chronic hyperventilation, 344 (49%) exhibited panic; 213 (930.4%) were goraphobic. School phobia and some cases of dyslexia are associated with a conditioned reflex of hyperventilation in these situations.

BLOOD SUGAR: A moderate fall in blood glucose grossly aggravates the effects of hyperventilation. This may be critical in the production of panic attacks and seizures.

FOOD ALLERGY: Many referrals have been initially misdiagnosed as food allergy. Pearson and Rix found that in a high proportion of such patients the diet was deficient in some essential nutrient. They also found that hyperventilation was the major cause of symptoms in 60% of patients referred as "food allergy." Hyperventilation occurs as a conditioned reflex to a suspect food.

MUSCLE SPASM: Rarely absent; it commonly affects the back, neck, and temporal muscles. It more rarely affects perineal muscles-levator ani spasm, proctalgia fagax.

MENSTRUAL CHANGES: Associated with premenstrual exacerbation; the therapeutic use of the contraceptive pill.

OCCUPATIONAL HYPERVF-NTTLATION: Singers, actors and wind instrumentalists are particularly prone as an occupational hazard. Many have to abandon the profession. (Rosa Ponselle). Fast breathless talking is a common feature, and must be corrected.

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Control of the Respiratory Cycle in Conscious Humans

G. F. Rafferty and W. N. Gardner'

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There is no simple model for control of breathing in man. There are a number of degrees of freedom in the respiratory cycle.

In inspiration, these include tidal volume (VT), inspiratory time (TI), and mean inspiratory flow (MIF or VT/IT), and in expiration, expiratory time (TE).

We have developed a new technique to clamp and vary these variables individually or in combination to determine the strength of the mechanisms limited change of each variable and the hierarchy of control between variables.

Subjects breathed into and from an open circuit via a mouthpiece and pneumotachograph and variables were measured breath-by-breath on-line by computer. Subjects coincided two beeps of different pitches activated by the computer, one triggered at a fixed VT, and one at a fixed time after the start of inspiration (i.e. TI), expiration being initiated at this point.

The threshold for each beep could be varied from the keyboard without the subjects' knowledge and were slowly applied to reduce subject awareness of the induced change.

End-tidal PCO₂ (PET.CO₂) was held constant by manipulation of the inspired gas.

We performed 4 sets of experiments in various combinations of 7 normal subjects in mild hyperopia at a constant PET.CO₂ slightly above resting. Each lasted 3/4-1 hour.

The first experiment examined the range over which VT, and MIF could be increased and decreased by changing the threshold for the VT, beep in 50-100 ml steps every 3 minutes at constant TI. Subjects tracked the beeps without difficulty as VT, increased by 500 ml but were not able to reduce VT, by more than 20 ml below the resting value associated with a reduction in MIF of 12%.

In a second series of experiments, TI was changed by ± 800 msec at constant VT using a similar technique; no difficulty was experienced in either direction, TI, changing from, on average, 1.48 to 2.91 sec.

In a third series of experiments, VT, and TI were reduced together from starting values at inspired PCO₂ of 1,3 and 5% to keep MIF constant while reducing VT. At each level of chemical drive, VT overshoot the beeps and could not be reduced below the free breathing resting value.

These experiments show that timing can be changed over a wide range, that VT and MIF can increase without difficulty, but that it is impossible to reduce VT by more than a small amount below the resting values as dictated by the chemical drive.

In a fourth series of experiments, we studied rapid shallow breathing without feedback control at constant PET.CO₂ in 4 subjects. VT fell without difficulty for prolonged periods to about 1/2 resting but end-expiratory volume as measured by a respiratory inductive plethysmography increased to keep peak absolute tidal volume constant.

In summary, these results suggest that in conscious humans, the major role of the control mechanism is to prevent a fall of tidal volume below that dictated by the chemical drive, presumably to ensure the maintenance of metabolic requirements. Mechanisms controlling timing are of lesser importance, consistent with the needs for non-metabolic functions of breathing such as speech.

When VTI, is forced to decrease by a programmed manoeuvre such as panting, end-expiratory volume increases to keep absolute volume constant.

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Interaction Between Expiratory Time and Inspiration in Conscious Humans

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A breath can be divided into inspiratory and expiratory drive and timing variables. The regulation of expiratory time (TE) in conscious humans is important in speech and many non-metabolic respiratory functions. TE is linked to inspiratory variables via mechanical, vagal reflex, chemical, and central neuronal mechanisms. However, the mechanisms of these linkages are poorly understood in conscious humans and very little is known about the influence that TE has on the following breath, especially inspiratory time (TI).

To study these linkages, respiratory patterning variables were measured for each breath in real time by computer in various combinations of 17 normal awake humans breathing mildly hyperoxic (PI,O₂ 35%) and hypercapnic gas mixtures via a pneumotachograph into an open circuit.

Computer controlled auditory feedback allowed gradual and imperceptible 3-minute step alterations at a constant end-tidal PCO₂ (PET.CO₂) over 45-60 minutes of (1) TI, over a range of \pm 800 msec at constant inspired tidal volume (VTI), (2) VTI up and down in repeated steps of 200 ml at constant TI, and (3) TE over a range of \pm 2000 msec at constant VTI.

In each case, the timing of the uncontrolled half of the respiratory cycle was free to be determined by the subjects' automatic respiratory control mechanisms.

Variables were averaged over the final minute of each step and, in protocol 2, the average was obtained of 8 up and down steps respectively.

In protocol 1, TE changed significantly (P0.05) from 1.94 to 3.10 sec in parallel with a mean achieved change of TI, from 1.48 to 2.91 sec (N = 10) despite constant VTI. In protocol 2, TE and time for expiratory flow did not change significantly in response to a mean step change of VTI, of 150 ml at constant TI, averaged over 10 steps in 7 subjects. In protocol 3, change of TE from 1.63 to 4.87 sec (N = 8) had no significant influence on the subsequent TI, but VTI, increased slightly by a mean of 240 ml (P < 0.01) as TE lengthened despite attempts to clamp using the auditory feedback.

Thus, in conscious humans, inspiratory timing has a direct influence on expiratory timing independent of volume change and chemical drive, but expiratory timing has no influence on the inspiratory timing of the subsequent breath but has a small influence on tidal volume.

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Living Above the Anaerobic Threshold: Penalties and Remedies

P. G. F. Nixon, London, UK

Exertion above the anaerobic threshold is a common cause of hyperventilation. In chronic fatigue syndrome the anaerobic (AT) threshold may be below the requirement for many of the activities of daily living.

If the low AT is not diagnosed the subject is unlikely to be provided with the appropriate rehabilitation. Some have exercise prescribed without reference to AT, an intervention that can be self-defeating. Others may be given reassurance or psychotropic drugs without attention to the low AT.

Suggestions are offered for raising low AT. Attention to sleep, breathing behaviour and hypocapnic-adrenergic synergy is recommended as rational and prudent.

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Breath-Hold Duration and Respiratory Sensation during Muscular Exercise in Humans

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We have previously demonstrated that activation of the peripheral chemoreceptors (Pcs) by isocapnic hypoxia increases the perception of breathing during moderate exercise to a far greater extent than expected from the ventilatory (VE) effects.

The purpose of the present investigation was to examine how these naturally-occurring changes in PC ventilatory drive influence respiratory sensation during exercise.

Normal healthy subjects exercised on a cycle ergometer at a series of constant work rates (WR) below the lactate threshold. Respiratory airflow and respired gas tensions were monitored continuously for on-line derivation of VE and pulmonary gas exchange variables breath-by-breath.

In addition, the experiments were repeated with the PC "gain" increased and decreased using inspired O₂ fractions of 0.12 and 1.00, respectively.

At each work rate, the following procedures were conducted:

- (a) PC ventilatory drive was quantified by the transient "Dejours" O₂-inhalation technique;
- (b) subjects were asked to rate the difficulty of their breathing (D), using a standard visual analog scale; and
- (c) a volitional breath-hold (BH) manoeuvre was performed to the limit of tolerance, the duration of which (BHT) was taken as an independent and less subjective index of respiratory sensation.

BHT at a given work rate was longer with the hypoxic inspire and shorter with the hypoxic expire, relative to air-breathing.

For each O₂ fraction, BHT declined in a curvilinear fashion with increasing work rate, showing the largest fall in the low work rate range but a relatively small effect at the higher work rates. As a result, the end-BH alveolar PCO₂ was higher and the alveolar PO₂ lower, the higher the work rate. Despite this, a given reduction in BHT at high work rates was associated with a more marked increase in D than at low work rates.

Our results suggest that the ability to sustain a breath-hold is not uniquely determined by the intensity of respiratory sensation prevailing prior to the breath hold.

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ROUND TABLE

Breathing retraining for hyperventilators and asthmatics : recent advances and continuing controversies

Chair: J. GALLEGRO

*Panellists: D. BRADLEY, J. VAN DIXHOORN, R. FRIED, W.N. GARDNER, W. GARLAND,
P.G.F. NIXON, E. PEPER. K. WIENTGES, L.C. LUM.*

Discussants: G. BENCHETRIT, R. LEY, A. PITMAN, B. TIMMONS.

Convener: B. TIMMONS

Can Different Respiratory Sensations be Scaled Simultaneously?

H. Harty' and L. Adams

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Both normal subjects and patients can qualitatively distinguish between different respiratory sensations perceived during ventilatory stimulation or various states of disease and increasingly workers are attempting to measure such sensations in a quantitative manner.

The aim of this study was to investigate whether subjects could score breathlessness, respiratory effort and amount of breathing in response to volitionally targeted hypo- and hyperventilation during exercise.

Six subjects performed three 15. 5-min steady state bicycle ergometer exercise tests at 75% maximum predicted heart rate. Each run included a 7-min period of targeted breathing either at a normal ventilation, 15% below this normal level (hypoventilation) and 15% above this normal level (hyperventilation). The order of the runs was randomized.

Ventilation (Y) was recorded as 15-sec averages. Breathlessness (Breath), effort and breathing amount (randomized order) were consecutively scaled using a visual analogue scale (VAS) every 45 s. V, was significantly altered during periods of volitionally targeted hypo- and hyperventilation compared to that of normal.

Ratings of amount of breathing and respiratory effort tended to be higher during the hyperventilation run but large variations existed in all sensations scaled during either condition and non proved significantly different from control.

In a separate study when breathlessness was scaled alone, it was significantly increased during a similar period of volitionally decreased V1. These results suggest that either, sensations associated with under- and over-breathing during exercise are not different or, more likely, that subjects are unable to scale more than one sensation during a single exercise test.

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The Effect of Vocal Command on the Ventilatory Response to an Increase in Exercise Intensity Following Hypocapnic Hyperventilation

J. H. Howell' & B. A. Cross, University College London, Gower St., London, UK

Reducing VE by lowering arterial Pco₂ during mild exercise unmasks an abrupt increase in ventilation consequent to a step increase in exercise intensity.

This work has now been extended to determine whether the changes in ventilation were stimulated by the increase in exercise intensity per se or are associated with the signal identifying the onset of the higher workload to the subject (in this case, a verbal command).

Three fit male subjects performed four exercise tests while a further three performed at least two of the tests. Test 'N' was a two-stage maximal exercise test, three min per stage, at workloads of 50 to 150 W, preceded by three min rest.

In tests 'H', 'SHAM' and 'SURPRISE', the 50 W stage was extended to include a period of hyperventilation (3 min or until PET CO₂ fell below 25 mm Hg). Approx. 20 sec after the end of hyperventilation, in the 'H' test subjects were told "Workload going up now" and the workload was increased to 150 W; in the 'SHAM' test, the same command was given, but the workload was decreased to 48 W; in the 'SURPRISE' test, the workload was increased without any verbal command. These workloads were maintained for 4 min.

As previously reported, the increase in workload in the 'N' test was accompanied by an abrupt increase in VE, followed by a more gradual increase after the increase in workload. The increase in workload in the 'H' test was also accompanied by an abrupt increase in VE, but in this case VE fell back below its normal 50 W level before rising more gradually when PET CO₂ returned to its normal exercise level.

The majority of the initial rise in VE was due to a rise in f BR. In the 'SURPRISE' test, there was a small increase in VE, but this did not start to occur until the second or third breath of the higher workload, was blunted in onset, did not contain a significant f BR component and was much smaller in magnitude than the initial ventilatory response seen in the 'H' test. Conversely, the decrease in workload in the 'SHAM' test was accompanied by an abrupt increase in VE with a large frequency component. VE remained elevated for three to four breaths before falling significantly below the steady-state 48 W level.

It is clear from these results that the majority of the abrupt increases in VE seen in these subjects in tests 'N', 'H' and 'SHAM' is dependent on the presence of a signal identifying the onset of the higher workload, rather than the workload itself.

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Relationship Between End-Tidal CO₂ and Heart Rate During VDT Work

Ronald Ley and Lawrence Schleifer'
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The results of two independent studies designed to determine the effects of prolonged, stressful computer work (data-entry operations) on breathing (end-tidal CO₂) and heart rate are reported.

An examination of changes in breathing (decrease in PCO₂) and heart rate (decrease in inter-beat interval) make it clear that both measures are sensitive to the stressful performance demands of VDT data-entry work.

However, a comparative analysis shows that while heart rate increases indicate both physical and psychological workload demands, end-tidal CO₂ is the more sensitive discriminator of psychophysiological stress (mental workload demands).

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Pseudo-Allergy and Hyperventilation

L. C. Lum' and C. A. Lum, Cambridge, UK

The term allergy is generally applied to the reaction produced when an external antigen binds to an antibody on the surface of white blood cells, e.g., mast cells and basophils. This causes the release of histamine in excessive quantities, sufficient to cause inflammatory tissue damage. Hay fever and asthma are prototypes.

The first recorded pseudo-allergic reaction was produced by Sir James Mackenzie in 1886, when he caused a hypersensitive young woman to have an asthma attack by showing her an artificial rose.

Many industries employ chemicals known or thought to be toxic or to cause asthma, e.g., chemicals, glues, paints. My own industrial work has shown that any spillage which causes fumes is likely to affect some workmen with dizziness, faintness, vomiting, headache or collapse.

In studies of patients who believed themselves allergic to certain foods, Pearson (1985) found that, on blind trial, 20 of 24 were unable to distinguish mixtures containing supposed allergen from those which did not. Subsequently he found that 60% of such patients were hyperventilating.

Less of (1983) estimates that true allergy and specific intolerance account for less than 30%. The rest are psychological, hyperventilation ranking high. An intermediate group, with proven allergy (to house dust mite), is exemplified by 20 asthmatic children who were placed in hospital, away from their parents. On exposure to mites from their own home only one developed asthma.

These all suggest that a conditioned reflex of hyperventilation is a major factor in the above situations.

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