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Biological Psychology 59 (2002) 231–265

BIOLOGICAL PSYCHOLOGY

www.elsevier.com/locate/biopsycho

Proceedings of the Eighth Annual Meeting of the International Society for the Advancement of Respiratory Psychophysiology (ISARP)

Program Chair: William Gardner, MD

ISARP met in Oxford, UK on 14–16 September, 2001. The Society focuses on issues relating to respiration and psychological factors and meets annually, alternating between the US and Europe. This year, the conference was overshadowed by the September 11th terrorist attack on the World Trade Center in New York and the American delegates were unable to attend, but their abstracts will nevertheless be published.

The first day of the meeting consisted of a session devoted to therapists and the role of physical therapies in the management of hyperventilation and breathing disorders. The subsequent 2 days featured a return to basic-science issues related to asthma, hyperventilation and respiratory phenomena associated with panic and other anxiety disorders.

1. Physiotherapy for hyperventilation and breathing disorders

Hyperventilation syndrome and physiotherapy intervention: a retrospective study

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When this study started, there was no published evidence of predicted outcomes in the management of hyperventilation syndrome (HVS). This information looks at

0301-0511/02/$ - see front matter
PII: S0301-0511(02)00006-6

referral source, demographic data, treatment length, outcome data and costings. All patients had been assessed using the symptom-based Nijmegen questionnaire. Breath hold time and respiratory rate were recorded in many subjects. All patients had been offered a course of treatment using breathing pattern re-education, as described by Innocenti. Most patients were taught a relaxation technique. Panic attack control strategies were taught where these attacks were described by the patient as a significant part of the problem. Some 100 patient records were reviewed. A total of 70% completed the course of treatment. Their Nijmegen score (maximum score 64) fell from 32.1 to 14.7. Twenty-five had a recorded start and end breath hold, which increased from 19.6 to 28.1 s. The respiratory rate was recorded at start and end in 25 patients and decreased from 19.6 to 11.1 per minute. The mean attendance was 5.4 sessions equivalent to 2.5 h with an estimated cost of £35 per patient. This retrospective study showed a significant improvement in symptom score, respiratory rate and breath hold time. The intervention is not costly.

Research in physiotherapy interventions for hyperventilation syndrome (HVS)

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This presentation considers the complexities of researching physiotherapy interventions in HVS. It considers the complexity of the problem and the nature of breathing pattern disorders and it emphasises the need for more rigorous physiotherapy research. HVS has been variously defined as a physiological, psychological and more recently, a social phenomenon. There is a healthy debate amongst academics and clinicians about the relative importance of the neuro-chemical, cognitive/behavioral and social facets of this complex 'modern' phenomenon. Physiotherapy research into HVS is in its infancy and to date there is no definitive course of treatment that has stood up to rigorous cross-examination. This presentation will review some of the salient reasons for this situation using a case study drawn from the author's experience as a lecturer and clinician working with HVS. In the scenario, a student experiences the complexities of researching her treatment. She begins with a non-specific definition of the phenomenon and then develops a proposal that oversimplifies the inclusion and exclusion criteria. Her choice of patho-physiological markers is limited by technological advancement and available resources and she resolves to use tools which she knows to be suspicious. Having constructed a workable proposal, she is confronted with the problems of obtaining a meaningful sample size. To achieve this, she decides on a multi-centre trial and subsequently has to revisit the entire proposal to take on board her contributors'

opinions. Finally, she considers the intervention and finds that the need for a standardized method betrays her 'real' clinical practice.

The presentation considers the importance of consensual definitions, sensitive outcomes, multi-centre collaboration, more varied research methods, standardised interventions, international guidelines, methodological rigour and the practical applications of rigorous research into HVS. In doing so, the paper raises the following questions: How much does current research influence my practice? Is my practice effective? If so, when, where and how? Who are our principal collaborators? Is the problem with the research or with the principles of our practice? Is patho-physiology really at the root of what we do?

Lying through your lungs: How do we ensure rigour in hyperinflation research?

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This paper explores some of the complexities of undertaking research in people with breathing pattern disorders in the absence of organic lung disease. Outlining an approach taken by a research team in New Zealand, we highlight some of the difficulties encountered establishing the veracity of our claim that a therapeutic intervention could be effective in altering physiological markers of persistent hyperinflation. Physiotherapeutic strategies have been employed in the treatment of breathing pattern disorders for over 40 years. These have concentrated upon the awareness, re-education and restoration of efficient breathing patterns. Therapy aims to restore the diaphragm to normal resting length, reduce accessory muscle loading, alter breathing rate and depth, re-educate muscle imbalance, release neuro/myo-fascial tissues and reduce neuromuscular excitability. There is little direct evidence that physiotherapeutic approaches have a significant impact on symptoms associated with breathing patterns disorders. The proposed study therefore looks to evaluate a targeted physiotherapeutic intervention for subjects with persistent hyperinflation in the absence of organic lung disease. The presentation will outline work to date and debate some of the complexities in arriving at valid, reliable and repeatable physiological markers of persistent hyperinflation. Debate will focus upon the problems isolating variables that are independent of patient effort, whilst retaining 'normal', conscious respiratory function.

Effects of breathing exercises for patients with asthma: a systematic review

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There is clinical indication, but little scientific evidence, regarding the value of breathing exercises or breathing training in the management of asthma. As increasing numbers of patients are seeking to self-manage their condition and are visiting physiotherapists, psychologists and complementary practitioners, it is important to systematically evaluate currently available evidence regarding breathing retraining in asthma. A Cochrane review of randomised controlled trials (RCTs) was undertaken in order to determine the effectiveness of breathing retraining interventions in asthma. Trials with patients of any age having physician-diagnosed asthma recruited to undertake breathing retraining were considered for inclusion. Search terms used for searching electronic databases included: breathing exercises, breathing retraining, breathing re-education physiotherapy, physical therapy, respiratory therapy or Buteyko. From the search of titles and abstracts, 32 trials were identified as relevant and their full texts were obtained for consideration for inclusion. Five trials met our inclusion criteria. Data was abstracted and trial quality assessment was undertaken independently by two reviewers. Statistically significant improvement was seen with breathing retraining in daily PEFR (49 l/min, $P = 0.01$, data from two studies with 131 patients), reduction in bronchodilator use reported by one study (5.82 puffs less per week, $P = 0.00008$, 106 patients) and decreased episodes of exacerbations also reported by one study (1.27 episodes less per week, $P = 0.01$, 106 patients). Owing to the small number of trials and participants in the five included trials, no reliable conclusions could be drawn concerning breathing retraining in asthma. However, some encouraging benefits with breathing retraining were seen in terms of improvements in daily PEFR, bronchodilator use and acute exacerbations. Unfortunately, breathing retraining methods used in the five trials varied considerably, which added to the difficulty in assessing and comparing the trials. Further rigorous randomised controlled trials with larger patient numbers are required to draw definitive conclusions.

The authors acknowledge the assistance of the members of the Cochrane Airways Group at St George's Hospital Medical School, London. These include Paul Jones, Steve Milan, Anna Bara, Jane Dennis, Karen Blackhall and Toby Lasserson.

Experiences of breathing therapy for patients with 'hyperventilation' complaints and healthy students

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Breathing therapy is a combination of awareness of breathing responses to internal and external factors and techniques for breath regulation ('breathing retraining'). It is beneficial to both healthy practitioners of self-regulation strategies and to patients with breathing difficulties. There are dual benefits in the latter group in that therapy may help solve their problem, but also confronts them with it. Taking the experiences of healthy practitioners as a reference group, the questions to be studied are (1) whether the benefits are equal for patients with hyperventilation complaints; and (2) whether the experiences depend on the clinical success for patients. A questionnaire was used that assesses the experiences in four scales: awareness of breathing responses to (1) moments of rest and relaxation (REST), (2) activity and emotion (ACTION) and (3) positive (ADVANTAGE) and (4) negative experiences (DISADVANTAGE) of applying breathing techniques. It was applied to 181 students of a professional course in breathing therapy (41 men and 150 women, 36.4 years of age) and to 146 patients with hyperventilation complaints (48 men and 88 women, 41 years of age) of whom 88 achieved good success of breathing therapy, 44 a moderate success and 14 no success. The results showed that healthy students scored higher in REST and ACTION than all categories of patients; patients with good success scored significantly higher on REST than patients with moderate success. ADVANTAGES were equally high for both healthy students and patients with good success and higher than for patients with no or moderate success. DISADVANTAGES were highest for patients with no success and differed from the patients with better clinical outcomes and from healthy students. It is concluded that breathing awareness is particularly beneficial in healthy students and in patients for whom the treatment was successful; however, for patients without clinical success, the increased awareness is mostly of an unpleasant nature.

A physician's perspective: How can therapists exclude organic disorder?

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Background: The physiological definition of hyperventilation is breathing in excess of metabolic requirements, i.e. CO_2 production and is associated with increase in alveolar ventilation. There is a fixed relationship between CO_2 production, alveolar ventilation and alveolar PCO_2 (alveolar air equation). With constant CO_2 production, hyperventilation implies hypocapnia (end-tidal $PCO_2 < 32$ mmHg) and is always associated with excessive respiratory drive.

General points: Before starting therapy, organic conditions causing hyperventilation and breathlessness need to be excluded. These include most lung and airway diseases (asthma, bronchitis, fibrosing alveolitis, pulmonary embolus, diaphragm weakness, respiratory failure secondary to neurological disease), cardiac disorders

(early heart failure, pulmonary hypertension), thyroid disorders, anaemia, renal failure etc. and sinus disease with post nasal drip. By definition, in someone with a primary presentation of hyperventilation, underlying organic diseases are usually not obvious and are easily missed by most doctors, even specialists.

Not all doctors are the same—you cannot assume that organic disorders have been excluded because the patient has been referred by a doctor (e.g. surgeons have little idea about medical conditions, GPs have a too broad a brush). As a therapist, you need to use your own skills and keep a high index of suspicion. You can largely exclude the common conditions yourself with some basic questions and a few simple tests.

There are some general points that you need to keep in mind. Breathlessness and hyperventilation are not the same thing and breathlessness often causes hyperventilation and panic as the patient struggles to breathe. Women in the second half of the menstrual cycle have a lower arterial PCO_2 than males or women in the first half of the cycle. Physiological factors, such as pregnancy, pyrexia and altitude, all contribute to hyperventilation. Psychiatric and organic causes of hyperventilation often coexist and psychiatric disorders, such as depression, anxiety and panic, are often secondary to organic disorders. Always ask yourself why your patient is breathless or hyperventilating and try to specify the cause or causes. Check for misattribution—what does patient think his symptoms are due to? Check for sighing—it only requires an occasional large breath to maintain chronic hyperventilation.

Pointers from the patient's history: Asthma is suggested by childhood chestiness or bronchitis, hay fever, wheeze or breathlessness on exertion, dry cough or variables symptoms. Shortness of breath (with or without hyperventilation) is the presenting feature of all respiratory disorders. Sudden onset of breathlessness suggests pulmonary embolus or pneumothorax), onset over days or weeks suggests pneumonia, or asthma, onset over months suggests lung cancer and onset over years suggests chronic conditions such as COPD or fibrosing alveolitis.

Additional pointers from history: A recent swollen calf suggests a deep venous thrombosis (DVT) and the possibility of pulmonary embolus (PE) as a cause of breathlessness, especially if there is also a history of recent plane travel, immobility or a recent operation (especially of the hip or knee). Haemoptysis (coughing blood) suggests PE, lung cancer or bronchiectasis and requires urgent referral to chest physician. Breathlessness lying down suggests diaphragm paralysis (needs specialist tests by chest physician to confirm). A smoker with a low peak flow suggests COPD. A blocked nose suggests allergic rhinitis and can be associated with sinus disease and post nasal drip—this is a potent cause of hyperventilation and panic.

Essential tests: Chest X-ray is essential. Only asthma, pulmonary embolus or pulmonary hypertension can be present with a normal CXR. A report of normal lung fields and a small heart reassures that there is no interstitial lung disease or major cardiac disorder. *Peak flow* (plastic meter can be bought cheaply at a chemist). Check the predicted value for your patient—depends on age, sex, height (normal range is $> 80\%$ predicted). Males are usually between 550 and 650, females between 350 and 450 l/min. It is helpful to get your patients to monitor their own

peak flow at home twice daily over 2–3 weeks. A reduction in the early morning reading is the best diagnosis of asthma (it should be same as in the evening—asthma is suggested if it is >50 l/m lower in morning than the evening, or varies from day to day). *Routine blood tests* are essential—as a minimum, blood count, biochemistry, thyroid blood tests, ESR should be normal—these can be carried out by any GP or the patients can be referred directly to a private laboratory.

Optional tests: An echocardiogram (Cardiac Department) excludes heart failure and pulmonary hypertension. A fine cut CT scan of lungs is becoming the gold standard to finally exclude early interstitial lung disease (in which the CXR can be normal), lung cancer and emphysema, but is expensive and involves a lot of radiation exposure. A ventilation/perfusion scan (Nuclear Medicine Department) can exclude pulmonary embolus (but is also abnormal in airway disease). Cerebral symptoms and/or headache require a CT scan of head to exclude tumours etc. A sinus X-ray (and possibly a CT scan) excludes sinus infection. An MRI scan of the brain stem and cervical spine excludes tumours in the respiratory centres or spinal respiratory pathways (rare but occurs).

On the basis of long experience with patients with disproportionate breathlessness and hyperventilation, I believe that most of the above investigations should be performed before therapists can be reasonably happy that they are not dealing with an organic cause of breathlessness in the patients they treat. The presence of organic causes of hyperventilation and breathlessness do not necessarily negate the need for therapy but a successful outcome for therapy will be unlikely unless these organic disorders have been identified and treated.

Upper limb function and hyperventilation syndrome (HVS): a clinical phenomenon

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This case study presentation considers whether HVS and associated breathing pattern disorders can be related exclusively to upper limb function. It considers whether there is a psychophysiological basis to the clinical scenario and analyses the evidence for a link between upper limb function and breathing pattern disorders. Patient T presented with a long history of multi-focal musculoskeletal disorders that had not responded to conventional therapy. An anxiety/stress disorder had been identified, but no successful intervention had been employed. An approach, which focused on her breathing pattern disorder, yielded immediate results, but residual symptoms remained. These focused on the direct and indirect use of her upper limbs (hanging out washing, writing on a chalk board, aerobic exercise). Conventional sub-maximal aerobic testing demonstrated no dysfunction, yet a sub-maximal upper limb test provoked profound breathing dys-synchrony, a sensation of suffocation and a significantly disordered breathing pattern. All symptoms

were fully reversible and resolved immediately when the test was voluntarily terminated. Treatment focused upon dissociating normal upper limb function from the patient's breathing pattern. The presentation will illustrate some of these findings, which include some unexpected clinical outcomes. In addition, the presentation will include an overview of the research to date that illuminates the psychophysiological basis of a link between upper limb function and breathing and explore how this may become deranged. The presentation will close with recommendations for further research in this area that may inform practice. These will focus on the identification, evaluation and isolation of suspected upper limb involvement in breathing pattern disorders.

Persistent hyperinflation in the absence of organic lung disease: a case report

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This case study presentation considers the manual intervention required to reverse persistent hyperinflation in a 35-year-old male who presented with persistent hyperinflation in the absence of organic lung disease. The patient, a 35-year-old trained tiler, presented upon referral from a respiratory physician. Patient B had been experiencing 'shortness of breath' for a number of years that had been gradually getting worse. He presented to the respiratory physician who ruled out any organic disease and diagnosed hyperventilation/disordered-breathing syndrome. Treatment initially focused on education and reassurance of the absence of an organic disorder. The symptoms initially improved but then returned, increasing in severity. A biomechanical approach was then introduced, using a combination of inspiratory muscle training, postural balance and manual hands on techniques. Many of the studies to date have concentrated on neuro-physiological changes evident in disease and few have recognised changes that take place in people with persistent hyperinflation. The drive to maintain higher lung volumes may be related to neuro-muscular stimulation of stretch receptors in the upper costovertebral joints. Physiotherapeutic strategies have concentrated upon the mechanical imbalance inherent in inefficient breathing patterns. Yet, to date little work has been carried out to evaluate whether these approaches have a significant impact associated with persistent hyperinflation, hyperventilation syndrome and other abnormal breathing patterns. This case history follows a path of practice and leads to further questions to be asked regarding research in this area.

A case of a reinforced hyperventilation panic

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Hyperventilation and biopsychological organization often have complex interactions. That hyperventilation can have a considerable impact on psychological functioning has been demonstrated many times. However, except for the direct influence of anxiety, little has been written of the impact of psychological factors on the hyperventilation itself. The present case is a clinical example of such an interaction. A 30-year-old woman was treated for severe hyperventilation panic with agoraphobia. Within a 2 and half year period, she was cured of this panic four times and each time correct diaphragmatic breathing was established. With each relapse, there emerged a completely different pattern of hyperventilation-inducing breathing. Initially, her breathing rate while relaxed was 35/bpm with much upper thorax involvement. At one relapse, her breathing was much slower but consisted entirely of mouth breathing with extremely long exhalations. At another relapse, her breathing was extremely shallow with almost no observable movement. Among her initial symptoms were great difficulty in concentration, sweating, crying, trembling and a high level of anxiety. An intense fear of suffocating was the core cause of her agoraphobia. Following breathing retraining and other behavioral interventions, these symptoms almost completely disappeared. With each relapse, most of these symptoms returned with a somewhat lowered intensity. Behavioral investigation of the precipitant of each relapse suggested that the panics served as a phobic avoidance of certain internal stimuli. The hyperventilation symptoms distracted her from tender thoughts and feelings concerning her father. Once she was desensitized to those phobic stimuli, the relapses ceased. It was the reinforcement provided by phobic avoidance that caused her to learn different methods to bring on the panics. She did have many other severe problems, which were treated by a dynamically oriented therapist. A 6-year follow up showed no return of the panic or of the hyperventilation-induced symptoms.

Inspiratory muscle training in elite athletes, hyperventilation disorder and chronic airflow limitation

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This paper presents an evaluation of the psychophysiological basis behind the application of inspiratory muscle training (IMT) in three distinct populations: (1) Elite athletes—who demand maximal cardiorespiratory work capacity whilst having normal lungs and normal perception of breathing. (2) Hyperventilation disorder—where there is an abnormal perception of breathing in the absence of organic

lung disease, perception of dyspnea and the likelihood of disordered breathing. (3) Chronic airflow limitation (CAL)—where there is an associated obstructive lung disorder.

There has been a significant interest in IMT as a tool for achieving targeted respiratory training in elite athletes and some authors have argued that the benefits obtained could be applicable to people with a perception of dyspnea. There is growing evidence that newly developed devices that incorporate physiological training principles and biofeedback (e.g. RT_2, DeVilbiss Healthcare UK Ltd.) offer advantages over more traditional threshold loading approaches. Despite the evidence supporting the use of IMT in elite athletes and CAL, little work has been undertaken to explore the merits of IMT in hyperventilation disorders. This paper explores some of the scientific and theoretical bases for claims in favour of IMT and questions whether it offers clinicians any solutions to the problems of hyperventilation.

Dysfunctional breathing in a woman in wheelchair

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A woman of 47 years had a partial lower cervical lesion since a car accident 5 years ago. After intense rehabilitation, she had a part-time administrative job for 20 h a week as a receptionist. She was on sick leave for 1 month because of fatigue, palpitations and dyspnoea, which the GP labelled as hyperventilation. She had excessive use of the auxiliary respiratory muscles, sat slumped and did not feel her lower body. In her youth, she stuttered and had fainting attacks. The score on the Nijmegen questionnaire (NQ) was high (32). The response to breathing instructions was very positive. In three sessions, a remarkable change occurred with the use of several instructions, such as closing and opening the fingers of both hands with exhaling respiration. Inhaling resulted in mental relaxation leading to an increased sense of body weight and more relaxed inhalation. Raising both shoulders while continuing breathing helped her to differentiate shoulders and ribcage and resulted in an increased awareness of the respiratory motion around the trunk. She now sits more on her pelvis and is aware of respiratory movement. The auxiliary respiratory muscles are no longer overactive. At the 2-month follow-up, the complaints on the Nijmegen questionnaire were reduced to a normal level (16). After 6 months, the effect has become stable and she has returned to work. Thus, the complaints were mostly due to dysfunctional breathing because restoring both proper body awareness and functional breathing movement helped sufficiently.

Breathing retraining in mild asthma: What are the advantages?

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Hyperventilation is a common occurrence in asthma. It may result from over breathing when bronchoconstriction occurs and may be caused by emotion. Hyperventilation is also accompanied by breathing relatively dry and cool air, which is a common asthma trigger. We made the hypothesis that some mild asthmatic subjects present with hyperventilation and that the treatment of hyperventilation will have an effect on the physiology and emotions in asthma. We measured the $PetCO_2$, the FEV_1, the respiratory rate and the heart rate of 120 subjects: 40 were asthmatic subjects who then received a breathing retraining, 40 were also mild asthmatic subjects, but received no intervention and the 40 others were healthy control subjects. The mean age of the three groups was the same. The baseline FEV_1 was also the same in the three groups. The heart rate at rest was also the same, but the $PetCO_2$ of the control group (mean (S.D.) 34.8 (3.1) mmHg) was statistically higher than the $PetCO_2$ of the asthmatics (mean (S.D.) 32.6 (3.3) mmHg) ($P < 0.001$). The respiratory reeducation caused no change in $PetCO_2$; however, it caused a slower respiratory frequency ($P < 0.01$). The asthma symptoms (measured by a daily diary) of the asthmatic group who received a breathing retraining decreased significantly as compared to the other group of asthmatics. The impact of the respiratory reeducation on psychological aspects (anxiety sensitivity and quality of life) will also be discussed.

Respiration during voluntary pain and bleeding control

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Many yogic adepts demonstrate conscious control over mind and body. This study explored the psychophysiological correlates of piercing the forearm of a male subject with a non-sterile metal skewer. Physiological signals monitored were: surface electromyography (sEMG) from the upper right trapezius, respiration from the thorax and abdomen, electroencephalography (EEG) from the right occipital-parietal area, skin conductance level (SCL) from the left hand, and blood volume pulse from the left thumb. During the pre-baseline, the subject sat quietly with his

eyes open, then with eyes closed, and was guided through slow breathing. A clean non-sterilized skewer was pierced through his skin and muscle of the right forearm. After 3 min, the skewer was removed. The subject continued to sit quietly for the post-baseline. There was no bleeding during the insertion or removal of the skewer. The subject reported no pain. Breathing rate decreased from 16 breaths per minute (brpm) from pre-baseline eyes open condition to 6 brpm during guided breathing relaxation, skewer insertion and removal, and post-baseline. There was no breath holding during the skewer insertion or removal. SCL and trapezius sEMG increased during the skewer insertion and decreased after the removal of the skewer. α EEG increased and predominated throughout the slow breathing baseline, skewer insertion and removal and post-baseline. Heart rate showed a significant in-phase respiratory sinus arrhythmia (RSA) during the guided breathing, skewer insertion and removal, and post-baseline. The subject appeared to use slow breathing to keep his attention passively focused and not to react to the pain stimuli. The slower breathing rate, combined with the corresponding increase in RSA and predominant α EEG activity, may encourage the development of an anabolic state that would promote rapid regeneration and may be a useful pain reduction strategy.

The physiological correlates of two breaths per minute by a yoga master

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Control of breathing is the bridge between voluntary and autonomic control in many yogic practices. This study explored the physiological correlates of breathing two-breaths per minute (bpm) by a 61-year-old Japanese Yogi. Physiological signals monitored were: respiration from the abdomen and thorax; EEG from the occipital-parietal; blood volume pulse from the middle finger; skin conductance level (SCL) from the right palm; transcutaneous SpO_2 from the right index finger; and end tidal CO_2 from the right nostril. The subject sat quietly with eyes open for three-minute pre-baseline period, then breathed 2-brpm without feedback for 18 min with eyes closed, followed by two post-baseline periods for 3-min period with eyes open and closed. Respiration rate decreased significantly from 18 bpm during pre-baseline to 1.9 bpm for 18 min to 11.8 bpm during post baseline with eyes open and 7.3 bpm during post-baseline with eyes closed. End tidal CO_2 increased significantly from a mean of 37.8 mm during pre-baseline HG to 43.7 mmHg and during 2 bpm breathing and decreased during post-baseline to 36.3 mmHg. SpO_2 saturation did not change significantly although there was a significant increase in SpO_2 variability during the 2 bpm period. During the 2 bpm period compared to the pre-baseline, there was a significant increase in α and θ EEG and no change in

δ EEG activity, a slight increase in SCL, no change in mean heart rate and a significant increase in heart rate variability although not phase-locked with respiration. The subject felt comfortable and reported that this breathing felt effortless and that he could have continued breathing at 2 bpm. The yogi demonstrated that he could breathe at 2 bpm without effort and without compromising O_2 saturation. Discussed are the clinical and meditative uses of slow breathing.

Reducing computer mousing symptoms with biofeedback respiration and work-style training

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Many people are unaware of the physiological changes that occur when working at a computer. An increase in respiration rate and upper trapezius and deltoid muscle tension may lead to the onset of pain and injury. The resulting pain syndrome is often called repetitive strain injury (RSI), computer related disorders (CRDs), or by the descriptive localized problem (e.g. carpal tunnel syndrome or thoracic outlet syndrome). This controlled study explored the physiological changes that occurred during computer mousing and the efficacy of a three-session training intervention protocol. Twenty-seven volunteer subjects (mean age 27.6 years) were randomly placed into experimental or control groups. After completing a history questionnaire, sensors were attached to monitor respiration with strain gauges from the abdomen and chest; surface electromyography (sEMG) from upper trapezius, anterior deltoid, forearm flexor and extensor muscle groups; and temperature from the non-mousing forefinger. All Ss performed pre-assessment mousing tasks (tracing and word substitution) followed by a symptom questionnaire. Experimental subjects participated in three weekly training sessions that focused on respiration and sEMG biofeedback with somatic awareness, work-style training and generalization. The control group received no training. Final assessment for all subjects was identical to the pre-assessment. The experimental group showed a significant decrease in respiration rate from 19.4 to 13.5 breaths per minute as compared to the controls, 17.7–18.5 bpm ($P < 0.01$); significant decrease in upper trapezius sEMG activity, 18.0–2.6 μV compared to the controls 12.5–18.3 μV ($P < 0.001$); and a significant decrease in mean symptom rating compared to the controls ($P < 0.01$). This study demonstrates that training subjects to breathe slower and diaphragmatically, practice somatic awareness and implement healthy work habits while mousing, reduces CRDs. To stay healthy at the computer, we recommend that all people learn these simple preventative self-regulation skills.

Mobilizing health following a kidney transplant

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This case report explores how breathing training may reduce anxiety, lower sympathetic arousal and enhance quality of life. The subject was a 39-year-old female who, 6 months previously, underwent her second kidney transplant after 20 years on haemodialysis. The six-session training program included discussion about the relationship between arousal, homeostasis and immune competence; exploration of factors that inhibited abdominal movement during breathing tactile and kinaesthetic coaching and imagery to encourage lower and slower; breathing and respiratory sinus arrhythmia (RSA) biofeedback training; and transferring the learned skills to the home and work environment. After training, the subject lowered her habitual thoracic breathing rate of 27 breaths per minute (brpm) with an absence of RSA and a heart rate between 80 and 85 beats per minute to predominantly diaphragmatic breathing of 8 brpm with increased RSA and a slower heart rate (78–82 beats per minute). She lowered her blood pressure and reported significant improvement in her ability to relax, fall asleep and inhale pentamadine without any difficulty. She generalized the biofeedback breathing skills into many settings, such as waiting to be seen by the doctor, driving to the Medical Centre and before going to bed at night to relax. As she states, 'I am amazed at how effective this is. Over the years, I have tried meditating and watching my breathing with minimal, if any, results. Biofeedback made me feel in control of my body and its reactions to unexpected events'. Upon an 8-month follow-up, she continues to do well and reported that, 'I like living life slower. I seem to get as much done with less stress.' This approach should be investigated as a complementary technique for transplant patients, as well as for anyone needing to inhale medications that evoke coughing, gagging and panic.

2. Psychological factors in asthma

Emotional factors in bronchial asthma

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There is an extensive literature relating emotional factors with bronchial asthma, with at least some well controlled studies indicating that emotional distress can trigger severe episodes of bronchoconstriction. This paper focuses on the nature of

the emotions that provoke airway constriction and on the mechanisms underlying these effects. Early work centred on the role of autonomic nervous system pathways, but more recent studies emphasize the significance of neuroendocrine and immune-mediated processes. Recent laboratory and naturalistic studies are described which help to establish the relevance of emotional factors in clinical conditions.

Event-related potentials to airflow obstruction in children with asthma

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Increases in asthma morbidity and mortality have been related, in part, to 'underperception' of acute bronchoconstriction. Event-related potentials (ERPs) to respiratory stimuli may prove useful clinically in the identification of central correlates of abnormal perceptual responses in pulmonary patients. ERPs are time-locked cortical responses that are recorded non-invasively and that reflect the timing of sensory and perceptual brain responses. We previously demonstrated that ERPs to added loads (i.e. to increases in the resistance to breathing) vary predictably as a function of age, attention and stimulus intensity. The present study examined the effects of task difficulty on P300 to respiratory loads in children with asthma and in children without asthma. Data were obtained in nine patients ($M = 13.9$ years) and in 11 controls ($M = 13.0$ years) and for comparative purposes, in 13 young adults without asthma ($M = 20.5$ years). Percent predicted FVC, FEV_1 and FEV_1/FVC values in children with asthma (109, 104 and 97, respectively) were comparable to similar values recorded in children without asthma (108, 111 and 102, respectively). ERPs were recorded at Fz, Cz, and Pz to two levels of airflow obstruction. On 'target' (rare) inspirations subjects breathed against loads presented briefly (for around 100 ms), once every two to ten breaths without warning. In separate experimental periods, containing either only large (occlusion) or small (≈ 10 cm H_2O/l per second) obstructions, subjects made a button-press response immediately upon sensing a load. Button-press latencies were equivalent in patients and controls but were significantly longer to the small than to the large obstruction [$F(1,18) = 21.9$, $P < 0.001$]. P300 latencies decreased reliably as load size increased [$F(1,18) = 5.21$, $P < 0.05$], but ERP differences between groups were not statistically significant. We conclude that ERPs to acute increases in inspiratory resistance are readily recorded in children either with or without asthma and that both cortical and behavioral responses to increases in airflow obstruction are more similar than dissimilar in these groups.

Affective picture effects on respiratory resistance in health and asthma: arousal, negative valence or disgust?

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Prior research from our laboratory has yielded conflicting results on airway effects of affective picture presentations. While earlier findings suggested an effect of valence-nonspecific emotional arousal, a subsequent study yielded evidence for airway responses restricted to pictures of a negative valence. Two further studies are presented that employed the picture presentation paradigm in healthy and asthmatic participants. In the first study, 36 pictures were presented to 42 non-asthmatic students, in three affectively homogeneous blocks of positive, neutral, and negative pictures. Each picture was viewed for 20 s, with an interstimulus-interval of 30 s. In the second study, 30 asthmatic and 30 non-asthmatic participants viewed 40 affective pictures in a random order. Pictures had been pre-selected for eliciting mainly anxiety, depression, disgust, happiness, contentment, erotic feelings or neutral affect (depicting neutral objects and persons). In both studies, respiratory resistance (Rrs) was measured continuously by forced oscillations. In addition, respiratory indices (time, volume and flow parameters; and pCO_2 in study two), cardiac activity (including ventilation-corrected respiratory sinus arrhythmia) and electrodermal activity were measured. Results in both studies showed a monotonic increase in Rrs from positive to neutral to negative pictures. Analysis of inspiratory versus expiratory fractions of Rrs revealed that effects were restricted to expiratory Rrs. Rrs increases were most prominent in pictures preselected for eliciting disgust. Little evidence for changes in vagal tone or respiratory mediators of Rrs was found. We conclude that airway response to static affective pictures is restricted to material eliciting negative valence, and possibly to very specific affective contents. Predominance of responding in expiratory Rrs suggests a stronger involvement of the upper airways, with changes in glottic opening or laryngeal tone due to disgust.

Suggestion, negative affectivity and symptom perception in patients with asthma

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Asthmatics with high negative affectivity (NA) report more symptoms. We investigated the effect of suggestion on subjective and objective asthma symptoms as a

function of NA of the patients. Asthmatics (ATS criteria, $N = 32$) took two puffs from three separate inhalers. Inhaler 1 (with white mark) was said to contain an inert substance. Inhaler 2 (marked red) was said to contain a bronchoconstrictive agent that would trigger asthma symptoms. Inhaler 3 (marked with brand name Ventolin®) was said to contain a bronchodilator. FVC, FEV1 and ResFR were measured at baseline as well as the intensity of asthma symptoms (ASC), negative affectivity (PANAS) and social desirability (SDS). Following each trial of two puffs, V_{Ti}, V_{Te}, Ti, Te, $ETCO_2$ and HR were registered for 2 min, followed by measures of ResFR, ASC and the probability to take medication with a given symptom level (VAS %). High NA subjects had overall more subjective symptoms than low NA. They reported more respiratory (obstructive) symptoms after suggested bronchoconstriction and less obstructive (and dyspneic) symptoms after suggested bronchodilation. Other symptom categories were not affected by these manipulations. Low NA subjects did not show such effects. Respiratory symptoms correlated significantly with intended medication intake ($r = 0.26$; $P < 0.01$). Neither NA-status nor suggestion influenced lung function and only breathing parameters under voluntarily control changed as a function of suggestion. The suggestion effects on symptoms were unrelated to social desirability. In conclusion, self-report of symptoms in asthmatic patients with high levels of NA is based on other cues than symptom perception in patients with low NA.

Prior experience with symptom exacerbations, learning and symptom perception in patients with asthma

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Objective: We investigated symptom perception in newly diagnosed patients with asthma ($N = 30$; eight males) as a function of prior experience with an (induced) asthmatic symptom episode.

Methods: A standard histamine-provocation test (Cockroft's protocol) on Day 1 was followed in patients testing positive (FEV1 drop of 20% or more) by an identical test protocol on Day 2 with saline. Symptoms (asthma symptom checklist, ASC) were measured pre- and post-test on both days. Physiological measures were FEV1, FVC and ResFR.

Results: Symptom scores for ASC subscales obstruction (OBS) and fatigue (FAT) increased significantly from pre- to post-saline inhalation, whereas none of the physiological parameters changed. The increase in OBS and FAT was significantly related to negative affectivity (NEM) of the subjects ($r = 0.50$, $P < 0.01$; $r = -0.47$; $P < 0.01$, respectively), but not to changes in ResFR, FEV1, VC or provocative dose of histamine causing a 20% drop in FEV1.

Conclusion: The induction of symptoms in a specific set of contextual cues may be sufficient to subsequently induce similar symptoms when confronted with these contextual cues only. Asthmatic subjects with high negative affectivity were more vulnerable to these effects.

Breathing retraining improves for dysfunctional breathing in asthma

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Dysfunctional breathing (DB) may complicate asthma and impair quality of life. A randomised trial comparing breathing retraining with control (asthma education) was undertaken on asthmatic patients in the community with possible DB (Nijmegen questionnaire (NQ) score >22) (van Dixhoorn et al., 1985). Some 28.8% of responding patients had positive screening scores. A total of 33 patients were randomised to breathing retraining by a physiotherapist or asthma education with a nurse to control for professional attention. Health status was measured pre and 1 month post intervention using the AQLQ questionnaire (Juniper et al., 1992). Significant improvements were seen in the median AQLQ scores in the intervention over the control group in the overall score (0.60 vs. 0.09, $P=0.018$), symptoms domain (0.42 vs. 0.09, $P=0.042$), the activities domain (0.0.52 vs. 0, $P=0.007$) and the environment domain (0.50 vs. -0.25, $P=0.018$), but not in the emotions domain (0.80 vs. 0.25, $P=0.205$). The number needed to treat (NNT) to produce a clinically significant improvement in health status was 1.96. These data indicate that the NQ can detect patients who may benefit from breathing retaining from the general asthmatic population treated in primary care.

Juniper et al., 1992. Thorax 47, 76–83.
van Dixhoorn et al., 1985. J. Psychosom. Res. 29 (2), 199–2063.

3. Emotion and breathing

Bodily sensations and anxiety: the role of interoception

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A significant proportion of patients presenting to primary care or specialist medical services are worried about their health, although they are organically healthy. The health worries can be very persistent despite repeated medical reassurance. Typical examples are patients with panic disorder who repeatedly present to the emergency services thinking that they are experiencing a heart attack or patients with hypochondriasis who remain convinced that they have a heart condition although medical tests have been negative. The lecture will first review evidence that individual differences in the ability to perceive bodily changes and in the selective attention to bodily cues contribute to the patients' health anxiety. Studies on heartbeat perception and the perception of respiratory resistance in panic disorder, hypochondriasis and patients with benign palpitations will be reviewed. The implications of the findings for treatment will be discussed. This includes the necessity of giving patients a positive explanation for their bodily sensations. Results from controlled treatment trials will be reviewed.

Anxiety sensitivity modulates classical conditioning of respiratory distress during a CO_2 inhalation paradigm

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Anxiety sensitivity (AS) is a psychological trait of overconcern about bodily sensations that has been linked to panic disorder. Consistent with this idea, panic disorder patients are often psychologically hyperreactive to respiratory provocations, such as CO_2 inhalation. To extend these findings, the current study evaluated reactivity to repeated single-breath vital capacity CO_2 inhalations in 52 university students selected for high and low anxiety sensitivity. The sequence of inhalations was as follows: two room air, eight CO_2/O_2 (20%/80%), and two room air, with an inter-inhalation (recovery) interval of 240 s. Participants were fully informed about the sequence of gases to minimize expectation effects. We recorded self-reports of anxiety and breathlessness at critical times during the procedure. A comparison of reactivity during the first 15 s following the two sets of room-air inhalations allowed us to assess differential (high- versus low-AS) susceptibility to classical conditioning of respiratory distress (UCR) to the vital capacity inhalation (CS) during the eight CO_2 inhalation trials. We hypothesized that respiratory distress would be more conditionable in high than in low AS subjects. Results from a preliminary analysis of self-reported breathlessness indicate that high AS subjects habituated more slowly than low AS subjects to the CO_2 trials, but that the recovery after individual CO_2 trials did not differ between groups. The hypothesized conditioning effect was confirmed: Breathlessness ratings immediately following the second set of room air inhalations were much higher in high than in low AS

participants. This supports the idea that traumatic learning during suffocation episodes contributes to anxiety-related symptoms in individuals with high AS, some of whom suffer from panic disorder.

Respiratory variability in panic disorder

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Disordered breathing may play an important role in the pathophysiology of panic disorder. Several studies have now indicated that panic disorder patients have greater respiratory variability than normal controls. In this study, we examine baseline respiratory measures in four diagnostic groups to determine if greater respiratory variability is specific to panic disorder and whether effective anti-panic treatment alters respiratory variability. Patients with panic disorder, major depression or premenstrual dysphoric disorder, and normal controls subjects) underwent two respiratory exposures (5 and 7% CO_2 inhalation), while in a canopy system. Panic disorder patients returned after 12 weeks of either anti-panic medication or cognitive behavioural therapy, and were re-tested. Normal control subjects were also retested after a period of 12 weeks. Panic disorder patients had significantly greater respiratory variability at baseline than normal control subjects and patients with major depression. The premenstrual dysphoric patients also had greater variability that the normal control group. Panic disorder patients who panicked to 7% CO_2 inhalation had significantly greater baseline variability than panic disorder patients who did not panic. Anti-panic treatment did not significantly alter baseline respiratory variability. Our data suggest that increased respiratory variability may be an important trait feature for some panic disorder patients and may make them more vulnerable to CO_2-induced panic.

Measurement of respiratory and cardiac function by the LifeShirt™: initial assessment of usability and reliability during ambulatory sleep monitoring

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An accurate ambulatory monitor of breathing is needed to observe acute respiratory changes in patients with medical or psychological disorders outside the clinic (e.g. hyperventilation during panic or apneas during sleep). Significant limitations of existing monitors have been their size, troublesome operation and difficulty in keeping chest and abdomen bands in place during 24-h recordings. Recently, a garment with embedded inductive plethysmography sensors for continuous ambulatory monitoring of respiration, ECG, inductive cardiography, motility, postural changes and other functions has been developed. The signals are displayed and stored on a handheld computer (Visor) and then analyzed offline, extracting over 40 clinical parameters relating to cardiorespiratory function (e.g. tidal volume, heart rate, peak-valley RSA, stroke volume, pre-ejection period, apnea–hypopnea index, thoraco-abdominal coordination, sighing). In addition, patient information regarding specific symptoms and moods can be elicited. We report here on the first pilot study using the LifeShirt system. Twelve students were monitored for 12 h, immediately before, during and immediately after sleep. The setup of the shirt and recording system, and calibration of the bands involved minimal effort. Subjects rated the device as easy to operate and reported minimal interference with sleep comfort. Raw data were uploaded via Internet to the VivoMetrics Data Processing Center (Ventura, CA) and results for all parameters were retrieved the next day in spreadsheet format, containing breath-by-breath and median minute-by-minute results for all parameters. In addition, computer generated clinical reports about apneas, periodic breathing and heart function were provided. Initial evaluation of several parameters (heart rate, respiratory rate, tidal volume, minute ventilation, duty cycle) showed that changes during the night were consistent with changes described during sleep in the scientific literature. We conclude that this advanced system may open a new era in ambulatory monitoring for both clinical practice and scientific research.

The association between resting end-tidal PCO_2 and negative affectivity

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Objective: Two studies aimed to clarify the controversies concerning the relationship between dispositional negative affectivity and resting end-tidal fractional concentration of CO_2 ($FetCO_2$) in normal subjects.

Methods: In the first study, 110 participants (83 women) completed the trait and state scale of the PANAS and a checklist on psychosomatic symptoms in daily life. In addition, they reported on their menstrual phase and use of oral contraceptives. $FetCO_2$ was measured non-invasively during 5 min via a nose cannula connected to a capnograph. In a second study, $FetCO_2$ of high ($n = 20$, ten men) and low ($n = 20$,

ten men) NA participants was measured twice (6 min each) in the following conditions: (a) rest; (b) completion of the NEO personality questionnaire; and (c) completion of a verbal knowledge test. During one of the two measurements per condition, the participant was wearing a mouthpiece in addition to the nose cannula.

Results: After partialling out the effects of gender, menstrual phase and use of oral contraceptives, no association between NA and resting $FetCO_2$ was found in the first study. In the second study, there was no main effect of NA, but $FetCO_2$ was elevated significantly in the high NA group when they filled out the questionnaires, regardless of its type.

Conclusions: After proper control for hormonal status, no relationship between trait-NA and resting $FetCO_2$ was found when using a non-invasive measurement device. Dispositional NA was associated with higher levels of $FetCO_2$ responses to a mental task.

Respiratory variability and psychological state in schoolchildren

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Among the relations between respiration and psychological state, the associations with respiratory variability have been contradictory. Some authors found that persons with fear and anxiety have increased variability in respiration rate and volume (Stevenson and Ripley, 1952; Wilhelm and Roth, 2001), while others found that variability increased with experimentally induced positive emotion (Boiten, 1998). In this study, respiration was measured noninvasively during 5 min in 162 children: 88 girls, 74 boys), 11 years of age (9–13). They completed a battery of psychological tests. Principal component analysis revealed that respiratory parameters could be reduced to three: respiration rate, variability and T_i/T_{tot} (duty cycle), where variability (S.D. of total cycle time) represented S.D. of inhalation time, exhalation time and exhalation pause time. Structural equation modelling (SEM or LISREL) revealed a model that fitted the data well ($\chi^2 = 88.201$; dof = 79; $P = 0.224$): variability was positively related to anger-in and negatively to negative fear of failure and neurotic complaints; respiration rate was positively related to positive fear of failure and duty cycle was positively related to the latent variable of negative affect. Thus, variability in resting time components of respiration was higher among children with less fear of failure and less complaints. The findings are discussed in terms of a model of healthy respiration showing responsivity and a complex regulation through multiple determinants (Lehrer, 2000). By contrast, low responsivity showing the dominant influence of a single determinant or over responsivity showing the influence of conflicting determinants are less healthy respiratory characteristics.

4. Hyperventilation and breathing disorders

Hyperventilation: issues

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The physiological definition of hyperventilation is breathing in excess of metabolic requirements i.e. CO_2 production. This implies increase in alveolar ventilation. There is a fixed relationship between CO_2 production, alveolar ventilation and alveolar PCO_2 (alveolar air equation) and, with constant CO_2 production, hyperventilation implies hypocapnia (end-tidal $PCO_2 < 32$ mmHg). It is always associated with excessive respiratory drive. There is considerable uncertainty and lack of consensus about many aspects of this disorder and the average physician automatically equates hyperventilation with an anxiety state. This is probably far too simplistic. Is it a disorder in its own right and justifying a title, or is hyperventilation merely a marker for other conditions?

The first issue concerns the diagnosis of either increased ventilation or a low arterial PCO_2. What is the role of symptom questionnaires such as the Nijmegen questionnaire? Should we be trying to measure ventilation directly (difficult up to now because of lack of a normal range and big variability)? What is the future role for arterial, transcutaneous, and end-tidal measurements of PCO_2, either static or ambulatory? Can we make more use of various stress tests (e.g. exercise, voluntary overbreathing, Nixon's Think test etc.) to uncover a tendency to hyperventilate?

The second and most important issue is the cause or causes of hyperventilation. Is there a single syndrome that qualifies for the term 'hyperventilation syndrome'—if so, what is it? It seems that we all have our own views but there is little consensus. Should we stop worrying about this issue? It seems to me that part of the uncertainty about this disorder is that we all study different patient groups and I think in future we need to be more obsessional about defining our patient groups and where they come from (not all doctors are equal and you cannot assume that just because a patient has been referred by a 'doctor' then all organic causes of hyperventilation will have been excluded). Should hyperventilation be regarded as a disease per se or a clinical finding indicating other underlying disorders? Are patients with hyperventilation simply the extreme of the normal range (many normal subjects have resting arterial PCO_2 values in the low 30s (mmHg) or high 20s? If so, what causes these patients to become symptomatic—is this related to misattribution and the way these patients interpret their symptoms of hypocapnia? Is there a difference in patient's tolerance of hypocapnia? Can hyperventilation be explained entirely by a combination of known physiological, organic and primary or secondary psychiatric disorders? If not, what are the additional factors—habit? misattribution? abnormalities of control of breathing? bad breathing? Is there such a thing as abnormal breathing patterns which are susceptible to correction or is the

breathing pattern manifested by patients with hyperventilation the natural consequence of the automatic control mechanisms optimising breathing to account for all the conflicting metabolic and behavioural demands? Should we identify separate initiating and maintaining factors? Should we distinguish acute and chronic hyperventilation?

The third issue concerns treatment. How much emphasis should we put on treatment of the hyperventilation per se rather than the underlying aetiological issues? Is hyperventilation 'treatable'? Does relaxation, stress management, CBT etc. have a role? Do we need to treat hyperventilation or should we just worry about treating the misattribution and secondary psychiatric factors? Should all patients with hyperventilation be reviewed by a specialist physician and a psychiatrist?

Panic and respiratory dysfunction: a review with new data and analyses

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The suffocation false alarm theory of panic disorder has stirred controversy, prompted new investigations and led to revised analyses of old data yielding new conclusions. The concordance of panic disorder, separation anxiety and CO_2/lactate hypersensitivity in panic disorder and CO_2/lactate hypersensitivity premenstrual disorder led to the hypothesis of endorphinergic underactivity.

Non-fearful panic induction in normals with naloxone pretreatment of lactate infusion. This study investigated whether induction of an acute opioid receptor. blockade by naloxone pretreatment renders normal controls, who are generally unresponsive to lactate, sensitive to its panicogenic effects. Thirteen normal controls received i.v. naloxone followed by lactate infusion. Four of these subjects also received naloxone followed by saline. Subjective symptoms were assessed by the acute panic inventory (API), an anxiety scale, and Borg breathlessness scale. Tidal volume was measured throughout. Eight of 12 subjects had an episode meeting the acute discomfort criteria for DSMIV panic attack. This was accompanied by significant increases in API, Borg scale and tidal volume, but not on measures of fear. Naloxone infusion alone did not elicit any significant increases on any measure. Dose effects raise questions about role of δ opioid receptor. The naloxone lactate protocol may yield a laboratory model of nonfearful panic. Endorphinergic dysregulation may underlie separation anxiety, CO_2 and lactate sensitivity, a suffocation false alarm and panic disorder.

The lack of HPA activation in acute panic: The other heuristically baffling area was the fact that the acute panic attack was not associated with HPA activation or defecation and it was saliently associated with acute dyspnea. All of these features are inconsistent with the belief that the panic attack is simply misreleased fear.

Klein hypothesized that catabolic hyper-oxidative HPA activation would be counterproductive if the organism was reacting to acute suffocation, so perhaps some immediate HPA inhibition was a feature of the specific acute suffocation alarm system. A review of physiological data elicited by both experimental and clinical acute hypoxia will be presented.

Drop in end-tidal PCO_2 during phobic exposure

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Background: We recently demonstrated the feasibility of ambulatory measurement of end-tidal PCO_2 in driving phobics during exposure treatment. We observed greater hyperventilation in patients than controls, as evidenced by reductions in PCO_2.

Methods: To further advance our understanding of respiratory regulation during acute anxiety we measured volume and timing characteristics of breaths by inductive plethysmography. The data set was extended to include 18 driving phobics with three sessions of exposure to freeway driving and 13 controls with two driving sessions in the same situations.

Results: PCO_2 levels dropped more in patients than controls during exposure (35.59–30.76 vs. 35.97–32.90). Both groups showed incomplete PCO_2 recovery after exposure. Exposure PCO_2 levels increased in patients from session one to two, but not to session three (30.76, 32.80, 31.87 mmHg) and approached levels of controls (mean of session one and two 33.19 mmHg). The inductive plethysmography data indicated that respiratory rate was increased to the same degree in patients and controls, making it unlikely that group differences in PCO_2 levels were produced by incomplete measurement of end-tidal PCO_2 plateaus due to shortening of the expiratory phase.

Conclusions: This data provides converging evidence for the role of respiratory factors in the pathophysiology of phobic disorders. Repeated exposure leads to a reduction of hyperventilation in anxious patients.

Strained respiration: a distinct phenomenon with subject and task specificity

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Straining is a familiar phenomenon in psychology and medical practice and it is recognized by many in association with heavy or difficult mental tasks. Nevertheless, scientific studies of the circumstances evoking a strained breathing pattern were lacking. We found theoretical indications that strained respiration may be characterized by a complete or partial closing of the glottis during the first stage of expiration, thus inducing an increase in intrathoracic pressure and a prolongation of the expiratory phase (Fokkema, 1999). Inspiration may be quick and 'unrestrained'. In view of the reflexes associated with such a respiratory pattern, an association with circumstances of attention and information uptake and processing has been suggested, rather than stressors that require a quick response. The present study was designed to test this difference in response to different tasks.

Ten elementary school children (age 11 years) performed arithmetic and computer game tasks and nine of them performed a visual search task, after completing a state anxiety test in their school. Respiratory movements were recorded continuously with chest and abdominal straps, while blood pressure and heart rate were monitored. Expiratory time fraction (Exp/cycle, complement of duty cycle) and the relative volumes at 1/3 and 2/3 of the expiratory phase (V% expir. 33 resp. V% expir. 67) were the main parameters to measure the degree of straining. Subjects appeared to differ in baseline breathing pattern. At rest, breathing cycle period was 3.0 ± 0.05 s (mean $\pm$ S.D.), expiratory time fraction was 0.64 ± 0.09, V% expir. 33 was $53.2 \pm 1.1\%$ and V% expir. 67 was $84.8 \pm 0.7\%$. On top of the individual differences, subjects respond in individually different ways, some with a hyperventilatory tendency, others with strained breathing characteristics. This tends to mask the overall task effects, which for the arithmetic and search tasks consisted of a longer breathing period ($\Delta = 0.19 \pm 0.55$* resp. 0.06 ± 0.29* s) and an increase in the parameters for straining (e.g. less volume exhaled at 1/3 of the cycle: Δ V% expir. $33 = -7.6 \pm 35.0$* resp. $-2.6 \pm 7.6\%$ ns). The responses to the computer game were a shorter breathing period ($\Delta = -0.25 \pm 0.60$ s*) and decreased straining with respect to the expiratory time fraction ($\Delta = -0.01 \pm 3.6$ s, ns), but also increased straining (ΔV% expir. $33 = -4.1 \pm 6.7\%$*). Of the arithmetic task, the strong responses were measured during the first five respiratory cycles, during which no responses were made. A subject's response type, which may differ from the general tendency, is consistent over arithmetic and search tasks: The responses to the maths and search tasks (compared to pre-test resting) are used as replications in the Anovas (ΔV% expir. 67 $P < 5\%$; ΔV% expir. 33 $P = 7\%$; ΔExp/cycle $P = 1\%$; ΔExp. period $P < 1\%$, ΔInsp. period $P < 5\%$; ΔAmplitude $P < 1\%$). Such a test is not significant when responses to the computer task are included as replications of the subject's response.

Common tasks appear to induce changes in breathing pattern which are different for individual subjects and which probably are dependent on the type of mental effort. The suggested parameters for strained breathing are simple and adequate to measure these changes in breathing. Possible physiological correlates of strained respiration, such as hemodynamic variability might contribute to the explanation of individually different susceptibility to stress-related maladies like cardiovascular disease.

Fokkema, D.S., 1999. The psychobiology of strained breathing and its cardiovascular implications: a functional system review. Psychophysiology 36, 164–175.

Effects of mental workload on $ETCO_2$ in computer work

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The data reported here are preliminary findings on changes in respiration taken in connection with a program of research designed to address the question of how psychosocial stress in computer work contributes to musculoskeletal disorders of the upper extremities, neck and shoulder. The specific purpose of the present study was to determine the effects of high and low mental workload (stress) on end-tidal CO_2 ($ETCO_2$), one of several physiological measures taken concurrently. Fourteen subjects who could type at least 40 words per minute participated in the study. Using a within-subjects design, each subject completed an 18-min mental task under high and low stress conditions. The order presentation of the mental task conditions was counterbalanced. Under the high stress condition, the subjects counted backwards by units of seven from three-digit minuends (e.g. 903–7 = 896, 896–7 = 889, etc.) as rapidly as they could and keyed the remainders in α form into a computer. Under the low mental-stress condition, no mental calculations were performed. In this condition, the study participants read backward-sevens subtractions presented on a video-display and keyed the remainders in α form into a computer. The mean drop in $ETCO_2$ from resting baseline to the 18-min task was significantly greater for the high mental-stress condition (-3.84 mmHg) than the low mental-stress condition (-2.21 mmHg). These results support the hypothesis that $ETCO_2$ is a sensitive index of mental workload.

Control of ventilation in subjects with idiopathic hyperventilation: physiological and psychological considerations

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Sustained arterial and alveolar hypocapnia (not uncommonly to levels < 30 mmHg) with no discernible aetiology, such as arterial hypoxaemia or metabolic acidosis, has been characterised as idiopathic hyperventilation (IH). Howell (1997)

suggested that this condition maybe associated with both psychological and physiological abnormalities, with a high prevalence of anxiety, some evidence of depression and also disproportionate breathlessness reported in his group of patients. In an attempt to clarify the mechanism(s) of this hyperventilation in patients with well documented IH, we utilized: (a) the Hospital Anxiety and Depression scores to assess psychological status and the Nijmegen questionnaire to assess symptomatology and (b) the ventilatory and pulmonary gas exchange responses to exercise (to the limit of tolerance), to hypoxia and to hypercapnia to assess ventilatory control. The anxiety and depression scores were 10.5 (4.6 S.D.) and 6.6 (2.6), respectively, with a Nijmegen score of 46.3 (14); i.e. consistent with increased anxiety, depression and symptoms suggestive of hyperventilation. The patients' arterial acid-base status at rest was consistent with a compensated respiratory alkalosis (pH 7.42 with a base excess of -4.5 mEq/l, $PaCO_2$ 28 mmHg and PaO_2 119 mmHg). The patients also reported disproportionate breathlessness, both at rest and during exercise. Compared to an age-matched control group, the IH group manifested an augmented ventilatory (V_E) response during the moderate-intensity domain of incremental cycle ergometry, mediated chiefly through tachypnoea. At 40 W V_E was 38.6 (11.5) l/min in the IH group compared with 20.7 (5.6) in the controls. The further hyperpnoea, however, was proportional to the reduced 'set point' of PCO_2, such that at 40 watts end-tidal PCO_2 ($P_{ET}CO_2$) was regulated at resting levels (i.e. 28 (5.3) and 27 (5.7) mmHg, respectively). The lactate threshold (θ_L: estimated by standard ventilatory and pulmonary gas exchange criteria) was evident at the same fraction of the maximum O_2 uptake (VO_{2max}) in the IH group as in the normal subjects, consistent with good effort at the limit of tolerance. At θ_L, the ventilatory equivalent for CO_2 (V_E/VCO_2) averaged >40 in the IH group, compared to normal values of <30. Interestingly, however, there was little or no respiratory compensation for the metabolic acidosis above θ_L in the IH group. The increment in V_E/VCO_2 between θ_L and VO_{2max} was -2.5 in the IH group compared with $+6$ in the controls and the associated $P_{ET}CO_2$ decrements -0.5 and $+10$ mmHg, respectively. As this suggests reduced peripheral chemosensitivity, we performed an isocapnic hypoxic rebreathing test (Rebuck and Campbell, 1974) with PCO_2 maintained at the spontaneous level of 32 mmHg. The responses in the IH subjects were markedly attenuated, averaging -0.96 (0.7) l/min/% O_2sat. However, when PCO_2 was increased to 40 mmHg and maintained at this value, the response was normal at -2.0 (0.58) l/min/% O_2sat. The CO_2 sensitivity at rest, as assessed by the rebreathing technique (Read, 1967), averaged 1.3 (0.6) l/min/mmHg, i.e. the lower limit of normal; during moderate exercise, this increased to 2.15 (1.4) l/min/mmHg within the normal range. The CO_2 'control point' as assessed by the constant CO_2 inflow technique (Cummin, 1986) was significantly lower during exercise than at rest (39.6 (2.2) and 43.1 (2.9), respectively). Our results suggest increased chemosensitivity is not contributory to the primary hyperventilation in patients with IH. In fact, the hypocapnia is likely to have suppressed peripheral chemosensitivity, as evidenced both by the functional absence of the compensatory hyperventilation at high work rates and the markedly low eucapnic hypoxic ventilatory responsiveness (normalised by the increased PCO_2). The mechanism of the primary hyperventila-

tion in patients with IH remains to be elucidated. A behavioural mechanism seems unlikely, however, in that the results of polysomnography demonstrated that in slow-wave sleep the hyperventilation was maintained (Lowe, 2001).

The ventilatory response to eucapnia at rest, during CO_2 rebreathe and on exercise in idiopathic hyperventilators

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It has been postulated that the chemoreceptor response to CO_2 is altered in idiopathic hyperventilators (IH). IH patients have a normal central CO_2 sensitivity, however during eucapnic exercise ventilation is increased to a significantly higher level than during hypocapnic exercise (Jack, 1997). We studied 16 IH patients ten females mean age 55 (12) years. Patients performed hypocapnic and eucapnic cycle ergometer exercise tests, CO_2 rebreathe (Read, 1966) and CO_2 replacement at rest. VE increased during replacement at rest by 4.9 (7) l/min and by 5.1 (6) l/min during rebreathe $P = \text{NS}$ (both at $PetCO_2$ 40–42 mmHg). On freewheel VE increased by 17 (9) and 16 (9) l/min on freewheel with CO_2 replacement $P = \text{NS}$. At 40 W exercise however VE increased by a further 19 l/min with the addition of CO_2 (hypocapnic exercise at 40 w VE 34 (13) to 53(17) l/min at 40 w with CO_2 replaced $P < 0.01$). These data would suggest that there is an interaction between performing work and the addition of CO_2 that causes a disproportionate rise in VE. Endogenous CO_2 production on exercise in association with a complex interaction with the sensory input from muscles performing work may be responsible for the disproportionate rise in VE seen in patients with IH on exercise.

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The effect of hypocapnia and eucapnic on the symptomatology of idiopathic hyperventilators during hyperventilation provocation tests (HPT)

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There has been recent controversy as to the mechanisms of symptom production in idiopathic hyperventilators (IH) and the role of HPT in diagnoses of IH (Hornsveld, 1996). It has been documented that hypocapnia is mainly responsible

for the cause of symptoms in IH. We performed hypocapnic and eucapnic HPT (McKell, 1947) on 39 IH patients (23 females 16 males mean age 53 (12) years). Patient symptomatology was documented at test termination in three main symptom groups in terms of dizziness (D), chest tightness (CT) and shortness of breath (SOB). The data was analysed using χ^2-tests.

Symptoms	HPT	HPT + CO_2	P value
SOB	11	6	NS
CT	10	13	NS
D	25	13	<0.05
Total	46	32	NS

Dizziness was significantly reduced when $PetCO_2$ was maintained at 40–42 mmHg during HPT, however chest tightness and SOB remained, total symptoms reported was also not reduced. These data suggest that hypocapnia contributes to symptoms of hyperventilation as this is known to cause vasoconstriction and reduced cerebral blood flow. The mechanical effort of overbreathing may also be responsible for symptomatology of IH and the effect of this on muscle spindles and stretch receptors may also be important in the understanding of this complex condition.

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A placebo controlled randomised study for treatment of patients with idiopathic hyperventilation

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Idiopathic hyperventilation causes significant morbidity consumes healthcare resources and effective treatment modalities have yet to be found. We have performed a randomised placebo controlled study using hypnotherapy, physiotherapy, hypercapnic hyperpnoea training and supportive educational training as placebo treatment. The results have demonstrated some changes in objective and subjective measures. We have treated 17 patients (mean age (S.D.) 55 (10) years, 14 females, three males with hypnotherapy (three 1 h sessions)). Some 15 patients with physiotherapy (mean age (S.D.) 52 (12) years seven females, eight males (six

half-hour sessions): seven patients with hypercapnic hyperpnoea training (HHT) (mean age (S.D.) 55 (7) years two females, five males. HHT training consisted of five half-hour sessions per week over a 4-week period. We have also completed four placebo treatments to date mean age 63 (7) years three females for six half-hour sessions. Patients in all four groups completed Hospital Anxiety and Depression score (HADs), Nijmegen questionnaires, St George's Respiratory Questionnaires (SGRQ), they also performed cardiorespiratory exercise tests and breath hold tests on room air and 100% oxygen. The questionnaire results in all four treatment groups demonstrated that anxiety was reduced, there was also a significant decrease in symptoms reported following treatment with a significantly reduced Nijmegen score after treatment in all groups. SGRQ total score was also reduced in all four treatment groups. The exercise data post treatment showed that breathing pattern was more appropriate and there was less hyperventilation with significantly less hypocapnia. The exercise tests post hypnotherapy demonstrated decreased hyperventilation at rest and 40 W with significantly reduced ventilation (l/min) (pre and post testing at rest (14.9 (6) vs. 12.2 (4) and at 40 W 36 (13) vs. 29.3 (11) $P < 0.05$) respectively. $P_{ET}CO_2$ mmHg was also significantly increased at all work rates (rest and maximal watts (pre and post testing at rest (26.6 (4) vs. 31.1 (3) and at maximal watts 28.7 (4) vs. 31.2 (5) $P < 0.05$). This pattern was also observed post physiotherapy at 40 and maximal watts ventilation was significantly reduced (pre and post testing at 40 W 40.7(14) vs. 31.5 (12) and maximal watts 61.8 (19) vs. 51.4 (14) $P < 0.05$). $P_{ET}CO_2$ mmHg was also significantly increased at most work rates (rest 28.5 (5) vs. 31.8 (3) and maximal watts 27.8 (4) 31.5 (4)). Breathing frequency was significantly reduced at 40 and maximal watts following physiotherapy $P < 0.05$. HHT however did not demonstrate altered physiology on exercise, although $P_{ET}CO_2$ was significantly increased at rest (pre and post 27.7 (3) and 30.1(3) $P < 0.05$). Tidal volume (V_T) at rest was also significantly higher post training (0.55 (0.04) vs. 0.66 (0.12)) and the ventilatory equivalent for oxygen V_E/VO_2 was also significantly reduced at 75% and maximal work rate. Pre and post testing at 75 W V_E/VO_2 41.9 (5) vs. 38.3 (3) and maximal watts 48.4 (6) vs. 43.8 (5) $P < 0.05$. The placebo group showed similar findings post exercise with reduced ventilation, increased $P_{ET}CO_2$ and decreased V_E/VO_2. Breath hold times pre and post physiotherapy were 18 (7) vs. 19 (9) on room air and 27.5 (21) vs. 34.9 (30) on 100% O_2 $P = \text{NS}$. BHT pre and post hypnotherapy were 20.7 (15) vs. 20.5 (11) room air, and 21.7 (21) vs. 22.5 (18) on 100% O_2 $P = \text{NS}$. BHT was significantly increased following HHT training on room air and on 100% O_2. BHT on room air pre and post HHT was 29.6 (19) vs. 41.8 (28) and on 100% O_2 BHT was 28.1 (20) vs. 42.9 (30) $P < 0.05$. Maximal inspiratory mouth pressures (MIPs) and maximal expiratory mouth pressures (MEPs) were also recorded pre and post HHT. The results showed no change in MIPs (pre 77.6 (34) post 75.6 (34) $P = \text{NS}$), however MEPs were significantly higher following HHT (72.4 (17) vs. 89.2 (15) $P < 0.01$). The questionnaire data pre and post testing suggests that all treatment groups produced a reduction in anxiety and depression and the symptomatology reported. This pattern was also observed in the placebo group where no active treatment or breathing retraining was applied. These data suggest that the changes in subjective

measurements maybe placebo effect. At rest there were some improvements post treatment with increased $P_{ET}CO_2$ and decreased ventilation, suggesting a more appropriate breathing pattern at rest. The exercise data shows the same pattern with less hyperventilation and significantly less hypocapnia at some work rates. This altered physiology was observed in all active treatment groups and post-placebo treatment, these data suggest that the altered physiology may also be placebo effect. The completed treatment study may address this and show that educational training is as effective as active treatment.

The role of physiological linkage in cardio-respiratory activity in marital satisfaction

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Compelling research has demonstrated the role of physiological processes in the marital relationship. This study examined the extent to which patterns of physiological responding between spouses accounted for variance in marital satisfaction. Earlier research (Levenson and Gottman, 1983) demonstrated the power of physiological linkage to account for variance in marital satisfaction; however, parasympathetic nervous system activity was largely ignored. Replication of the Levenson and Gottman (1983) study was attempted with the addition of utilizing spectral analysis of heart period data to account for activity from both branches of the autonomic nervous system. Participants were volunteers who answered advertisements placed in newspaper and advertising publications. All participants were screened over the telephone and excluded from study participation for alcoholism, domestic violence and/or usage of prescription drugs with cardiovascular effects and if they had been married < 1 year. The first 30 couples to meet study criteria and complete laboratory session were included in the study. Couples arrived at a medical clinic after not seeing each other for at least 8 h. Each spouse completed the Marital Adjustment Test (MAT); a measure of marital satisfaction, the average of which comprised the measure of marital satisfaction. Physiological measures were explained and attached to each spouse. Physiological monitoring included heart rate (HR), skin resistance level (SRL), facial EMG, respiration rate, finger temperature and spectral analysis of heart rate. Spectral analysis of heart rate was incorporated to investigate relative contributions of the sympathetic nervous system (SNS) and the parasympathetic nervous system (PNS). High frequency, an index of cardiac vagal tone, primarily mediated by the PNS, was observed at 0.15–0.40 Hz. Low frequency and index of primarily SNS activity, was observed at 0.10–0.15 Hz. The ratio of LF/HF indexed the relative contribution of the SNS and vagal activity (Malik et al., 1996).

Spouses participated in a vanilla baseline procedure (Jennings et al., 1992), which consisted of spouses rating their preference for one of two scenic photographs each

minute for 10 min. Couples then participated in a 15-min discussion of the events of their day, followed by a 5-min recovery period. Recovery periods consisted of the same procedure as the baseline segment. Spouses then completed the Knox Problem Inventory, a questionnaire listing common areas of conflict for couples. A discussion followed with the couple regarding the topic listed most conflictual by both partners eliciting each spouse's thoughts and feelings regarding the area of difficulty. This was followed by another 5-min recovery period. Couples were then reminded of their endorsed area of disagreement and instructed to spend the next 15 min working on a solution to the problem. This resulted in the second discussion period of the study and was followed by a final 5-min recovery period.

The average of the couples' MAT score was used as the measure of marital satisfaction. Physiological variables were analyzed continuously and averaged into 10-s intervals. Physiological data was screened for values two or more S.D. above or below the mean of the preceding and following value and replaced with the mean of the preceding and following value. Physiological variables were analyzed for patterns of relatedness between spouses utilizing bivariate time-series analysis. No support was found for a relationship between physiological linkage of spouses and marital satisfaction, failing to replicate the earlier research of Levenson and Gottman (1983).

Possible differences between the findings of Levenson and Gottman (1983) and the present study may result from the sample of participants selected. The present study eliminated $\approx 50\%$ of the couples where one or both of the spouses endorsed being unhappily married due to a reported episode or more of violence in their marriage. No such screening for domestic violence was conducted in the original research of Levenson and Gottman (1983). The proposed absence of fear or threat during a discussion of a problem area in the marriage may result in the differences between study findings. In addition to the hypothesis of physiological linkage, the relationship between vagal tone and marital satisfaction was investigated. Vagal tone was analyzed during study conditions by computing period means for HF heart period data. Results indicate a pattern for happier couples to experience increased vagal activity following discussion periods. A significant correlation was found for the recovery period following the couples' discussion of the events of their day ($r = 0.461$). The recovery period following the couples' conflict discussion approached significance ($r = 0.356$). The same pattern of vagal activity was found for couples while they discussed the events of their day ($r = 0.318$). Physiology that accompanied happier couples reflected vagal nerve regulation of heart rate during non-conflict discussion periods. This finding supports Steven Porges (1992) theory of complex human communication being mediated through regulation of vagal tone.

A general hypothesis regarding SNS activation and marital satisfaction was investigated through analysis of period means of SRL and LF/HF heart period data. No significant finding resulted from analysis of period means for SRL, with Pearson Product Moment Correlations ranging between $r = -0.024$ and $r = -0.272$. However, he study found a significant relationship between SNS activation, as indexed by LF/HF, and marital satisfaction during a couple's discussion of the

events of their day ($r = -0.390$). This finding indicates that during discussions of relatively neutral material, the greater the influence of SNS activity and the lower the level of PNS influence, the less happily married the couple. General results of the study demonstrate the importance of the relationship between marital satisfaction and the complex interplay between SNS and PNS activity, during relatively neutral discussions of couples.

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Association of perceived stress with hypopnic breathing pattern in women

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The physiological pathways by which behavioral stress and high sodium diet contribute to the development of hypertension remain to be clarified. Previous studies with laboratory animals associated behavioral stress with sodium-sensitive hypertension via a suppressed breathing pattern. Studies with humans have linked elevated resting blood pressure in women (but not men) who inhibit anger expression to high resting end tidal CO_2 ($PetCO_2$). In the present study, perceived stress, resting breathing frequency, resting $PetCO_2$ and resting blood pressure were recorded in 278 healthy adults. For both men and women, slow breathing pattern was an independent correlate of higher resting $PetCO_2$. For women (but not men), high perceived stress was an independent correlate of slow breathing pattern and high resting $PetCO_2$ was an independent correlate of systolic blood pressure. These findings are consistent with the view that high perceived stress could potentiate sodium-sensitive hypertension in women via effects on breathing pattern and blood gases involved in sodium regulation.

Toward the clinical application of symptom perception data in pediatric asthma

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Accurate symptom perception is an important aspect of asthma management. Technological advances have made it possible to obtain multiple assessments of patients' subjective estimates compared to corresponding objective pulmonary function data. In theory, an individual's pattern of symptom perception can be determined from these multiple assessments. Dangerous under-recognition of clinically meaningful symptoms, accurate symptom perception, and over sensitivity to mild or minimal changes are examples of potentially meaningful patterns. This presentation will discuss operationalizing for clinical practice data from our study of children's symptom perception. Quantification of multiple assessments and an approach to summarize an individual's data are essential steps in the process. The Asthma Risk Grid's latest modification incorporates the three levels of clinical compromise from the NHLBI guidelines. Nine categories and several ratios summarize the data for an individual patient according to levels of accuracy and potential for underestimates of symptoms. Use of the grid will be presented succinctly. The perceptual data on four illustrative, 'pure culture' cases from our pediatric sample will be presented using the Grid to collapse the data graphically. The individuals' asthma morbidity data for the previous year will be compared to their grid classification. This presentation illustrates the application and clinical relevance of symptom perception assessment for childhood asthmatics.