INDEX OF PAPERS and POSTERS

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GROUP 1:
PANIC DISORDER, ANXIETY, ASTHMA and HYPERVENTILATION

1.) A New Ambulatory Measure of Hyperventilation in Anxiety Disorders
Alpers, G.W., Wilhelm, F.H., Roth, W.T.

2.) An Internet Survey of Incidences in Changes in Breathing and Dyspnea During Panic Attacks
Anderson, B., Ley, R.

3.) Hyperventilation Syndrome, Asthma and Anxiety Sensitivity
Delvaux, M., Fontaine, O., Bartsch, P.

4.) Are Current Theories of Panic Disorder Verifiable?
Roth, W.T.

5.) Mechanisms of Hyperventilation in Mild Asymptomatic Asthma
Osborne, C.A., Lewis, A., Orr, L.M., Kanabar, V., O'Connor, B.J., Gardner, W.N.

6.) Reactivity to a 35 % CO2 Challenge in Healthy First-Degree Relatives of Patients with Panic Disorder
van Beek, N., Griez, E.

7.) Hyperventilation beyond fight/flight:
The relative importance of affect and action readiness in hyperventilation responses
Van Diest, I., Van de Woestijne, K.P., Devriese, S., Stegen, K., Winters, W., Van den Bergh, O.

GROUP 2:
RESPIRATORY RESPONSES to PHYSICAL and PSYCHOLGICAL TASK PERFORMING and STRAIN

8.) Generalization of Acquired (Respiratory) Symptoms in Reponse to Odors:
A Possible Mechanism for Sensitivity to Multiple Chemicals
Devriese, St. , Winters, W., Van Diest, I., Stegen, K., Van de Woestijne, K.P., Van den Bergh, O.

9.) Breathing Training Allows for Altitude Ascent
MacGregor, R.P., Hille, M.

10.)Emotions and stress in asthma:
Effects on pulmonary function in standard laboratory settings and everyday life of patients
Ritz, T., Steptoe, A.

11.) The Influence of the Background Noise Intensity on Respiratory, Cardiovascular and Electrodermal Activity During Performance of Word Recognition Task
Sohn, J.-H., Sokhadze, E.M., Lee, K.-H., Choi, S.

12.) Induced Emotions and Airway Resistance -
Measuring the Respiratory Impact of Emotion Laden Movie Clips by Whole Body Plethysmography
von Leupoldt, A., Dahme, B.

13.) The Measurement of Blood Gases Before and After
Incremental Exercise on Bicycle of
Patients and Control Participants: Supporting the Effort Syndrome Hypothesis?
von Scheele, B., von Scheele, I.

14.) Cardiorespiratory Reactivity under Stress and Individual Characteristic of Alexithymia
Umezawa, A., Terai, K., Takeuchi, H.

GROUP 3:
RESPIRATORY SENSATIONS and BEHAVIORAL CORRELATES

15.) Improved Methodology for Threshold Detection Studies in Asthmatic Children
Fritz, G.K., Klein, R.B., McQuaid, E.L., Rosenblum, K., Nassau, J.H., Mansell, A.

16.) Behavioral and Respiratory Muscle Responses to Inspiratory Resistive and Elastic Loads

Harver, A., Flamer, M.

17.) Respiratory Sensations after Respiratory Endurance Training
Spengler, C.M.

A NEW AMBULATORY MEASURE OF HYPERVENTILATION IN ANXIETY DISORDERS

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Many theories of panic disorder and phobic anxiety refer to aspects of respiration , usually hyperventilation. Well established for laboratory environments, non-invasive estimates of arterial PCO2 are a recent addition to ambulatory psychophysiological methodology.

With a transcutaneous method, Hibbert et al. (1988) found that some of their freely ambulatory patients do hyperventilate during panic attacks but others do not. Garssen et al. (1996) observed a decrease of PtcCO2 during only one of 24 panic attacks.

We show that PCO2 estimations from the air at th e nostrils at the end of expiration can be done in the field. The main advantage of this method over the transcutaneous one is that it has only minimal time delay and breath-by-breath resolution.

Our data are from patients with fear of driving an automobile, who underwent three driving sessions, and healthy control subjects. Data from the first 10 patients who did not experience a panic attack show that their PetCO2 levels during the first driving session (28.7 mmHg) are lower than those of 12 control subjects (33.2 mmHg). The patients' PetCO2 levels do not recover within the exposure sessions, but mean PetCO2 increases across the three sessions (28.7; 30.2; and 30.3 mmHg).

Thus, repeated exposure leads to a reduction of hyperventilation in anxious patients. In two patients who were monitored while having a panic attack there were marked drops in PetCO2: one from a mean of 36.9 mmHg during a 5 min baseline to a minimum of 21.5 mmH g during the attack, and the other from 40.0 mmHg (baseline) to 15.8 mmHg (attack).

AN INTERNET SURVEY OF INCIDENCES IN CHANGES IN BREATHING AND DYSPNEA DURING PANIC ATTACKS

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This study investigated breathing related symptoms and is a follow-up study of the severity of panic attack symptoms reported by active panickers (Anderson, 1998). The subjects experience naturally occurring panic attacks, and have sought treatment guidance by visiting a web site devoted to this subject.

Two hundred and five respondents (58 male, 120 female, 27 unknown gender) filled out a single page symptom questionnaire which included given name, length of time suffering from panic attacks, DSM-IV criteria for panic attacks, and criteria for grades of dyspnea proposed by Comroe (1965, in Tobin, 1990).

The results showed that 195 respondents (95.1 %) reported breathing changes during panic attacks. While 26 (12.6 %) reported increases in breathing with no unpleasant dyspnea, 169 (81.4 %) reported changes in breathing that indicate dyspnea: (1) 29 reported "a little shortness of breath" (14.1 %); (2) 75 reported "sensation of hindered breathing - it is harder to breathe" (36.6 %); (3) sensation of suffocation with acute need for a deeper breath" (29.3 %); (4) 5 reported "the sensation of being at the breaking point of breath-holding" (2.4 %).

These findings which are in the main consistent earlier studies, and are contrary to Schruers, et al's (1998) findings and their conclusions that only a small number of panickers report "shortness of breath" as one of the symptoms of panic. The current study concludes that outside of the laboratory a large majority of people who suffer from panic attacks experie nce dyspneic symptoms. It also demonstrates the value of a graded system, as proposed by Comroe, when evaluating dyspnea for research or clinical purposes.

Hyperventilation Syndrome, Asthma and Anxiety Sensitivity

Delvaux, M., Fontaine, O., Bartsch, P.

(no abstract)

ARE CURRENT THEORIES OF PANIC DISORDER VERIFIABLE ?

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Current theories of Panic Disorder (PD) include the suffocation false alarm hypothesis, hyperventilation, faulty inhibition of activation, hypothalamic inst ability, and cognitive-interoceptive theories. Some of these theories have generated treatment recommendations or claim support from the efficacy of a treatment.

Recent observations of PD patients in our laboratory and others indicate specific involvement of the respiratory system. End-tidal pCO2 is often lower in PD patients compared to patients with other anxiety disorders and equal self-reported anxiety. Tidal volume variability tends to be higher in PD patients. Other replicate d findings are slower physiological recovery after hyperventilation and slower skin conductance habituation, although their diagnostic specificity is less well documented. However, whether these or other findings are unequivocal enough to demonstrate causal mechanisms, justify a particular treatment, or verify or falsify a current theory is questionable. A persistent difficulty is knowing how much etiological weight

to assign to thoughts and attitudes vs. biological abnorma lities. For example, are "catastrophic cognitions" the cause or a result of panic attacks?

I conclude that current theories of PD have a heuristic function but do not provide mutually exclusive explanations for this disorder. Yet, certain empirical findings stimulated by these theories have changed our clinical understanding of PD.

MECHANISMS OF HYPERVENTILATION IN MILD ASYMPTOMATIC ASTHMA

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The mechanisms of hyperventilation (HV) in asthma are uncertain and have been attributed to a combination of physiological and psychological elements. The mechanisms may be different and more complex in severe compared to mild chronic stable asthma. Even the presence of HV in the latter group is uncertain, but is of relevance to the aetiology of hyperventilation syndrome.

We measured ventilatory variables (Vt, Ti, Te, Tt, Freq.), radial arterial blood gases for arterial PCO2 (PaCO2) and PaO2, end-t idal PCO2 (PetCO2), standard lung function (FEV1, VC, PEF, gas transfer, TLC, RV), airway responsiveness to methacholine (Pc20), airway inflammation assessed by induced sputum eosinophilia, and psychiatric state assessed by Speilberger STAI-Y and Beck Depression Inventory in 17 normal subjects (C) and 23 mild asymptomatic asthmatics (A) (occasional salbutamol, normal lung function, positive Pc20).

In A PaCO2 and PETCO2 were significantly than C (PaCO2: mean _SD ; 37.2 _ 3.6 mmHg verses 39.5 _ 2.9 mmHg. $p < 0.02$) related to a small but insignificantly higher respiratory frequency. The reduced PaCO2 in A significantly correlated with Pc20 ($r = 0.55$, $p < 0.01$) but not with any aspect of lung function, airway inflammation or psychiatric morbidity.

In summary, even mild asymptomatic asthma is associated with a significant decrease in both arterial and end-tidal PCO2 and this relates to airway hyperresponsiveness rather tha n mucosal inflammation. Psychological factors cannot be implicated.

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REACTIVITY TO A 35 % CO2 CHALLENGE IN HEALTHY FIRST-DEGREE RELATIVES OF PATIENTS WITH PANIC DISORDER

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Introduction: It has been established that panic attacks can be experimentally elicited by 35 % CO 2 i nhalation in panic disorder (PD) patients. CO 2 seems to activate specific mechanisms and that may be what predisposes PD patients to the development of panic. It is known that PD runs in families and that PD patients have a certain tendency to react hypersensitively to CO_2 . If there is a link between familial loading for PD and hypersensitivity to CO_2, first-degree relatives may also have a tendency to be hypersensitive to CO_2. The aim of the present study, therefore, was to determin e whether first-degree relatives of PD patients are, in fact, more reactive to a 35 % CO_2 // 65 % O_2 challenge than healthy control subjects.

Method: The effects of a single inhalation of 35 % CO_2 and one of compressed air (placebo) were examined in 50 healthy first-degree relatives of PD patients and in 50 healthy controls matched for age and gender. At the time of the challenge, all subjects were healthy and none had taken any medication that could influence their m ental state. Questionnaires that the subjects filled out included the State-Trait Anxiety Inventory (STAI-1) for anxiety state, the Visual Analogue Scale for Anxiety (VAS-A) for subjective anxiety, and the Panic Symptom List, DSM III-R (PSL III-R) for panic symptoms.

Results: The first-degree relatives were more reactive to the 35 % CO_2 challenge than the controls.

Conclusion: The present findings indicate that being a member of a family with a PD patie nt is, in itself, an important factor in CO_2 hypersensitivity, even for subjects who have never experienced a panic attack.

Hyperventilation beyond fight/flight: The relative importance of affect and action readiness in hyperventilation responses

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(no abstract)

GENERALIZATION OF ACQUIRED (RESPIRATORY) SYMPTOMS IN RESPONSE TO ODORS: A POSSIBLE MECHANISM SENSITIVITY TO MULTIPLE CHEMICALS

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Aim: (Respiratory) somatic symptoms in response to odors can be acquired in a Pavlovian conditioning paradigm. In the present study it was investigated 1) whether learned symptoms can generalize to new odors; 2) whether the generalization gradient is linked to the affective or irritant quality of the new odors; and 3) whether the delay between acquisition and test modulates generalization.

Method: Conditional odor stimuli (CS) were (diluted) amm onia and niaouli. One odor was mixed with 7.4 % CO_2-enriched air (unconditional stimulus) during 2 min breathing trials (CS+ trial), while the other odor was presented with air (CS— trial). Three CS+ and three CS— trials were run in a semi-randomized order (acquisition phase). The test phase implied one CS+ Only (i.e. CS+ without CO_2) and one CS— test trial, followed by three trials using new odors (butyric acid, acetic acid, and citric aroma). Half of the subjects (n = 28) were tested immediately after the learning phase, the other half were tested after one week. Ventilatory responses were measured during and somatic symptoms after each trial.

Results: Participants had more symptoms to CS+ only exposures, but only when ammonia was used as a CS+. Also generalization occurred: participants reported more symptoms to butyric acid and acetic acid and not to citric aroma, but only when they had been conditioned successfully before. Both the selective conditioning and generalization effect were mediated by Negative Affectivity (NA) of the participants. The delay between acquisition and test phase had no effect. Conditioning did not affect respiratory responses.

Conclusions: Symptoms to odorous substances can be learned and generalize to new substances, especially in persons with high negative affectivity. The findings further support the plausibility of a Pavlovian perspective on multiple chemical sensitivity.

BREATHING TRAINING ALLOWS FOR ALTITUDE ASCENT

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The purpose of this study was to evaluate the benefits at high altitude of proper breathing training and improve the overall response to altitude using a n oximeter as a biofeedback measuring tool.

The Sherpas of Nepal and Tibet tend to have an altered breathing response to low oxygen and make it to altitude with little difficulty in comparison to trekkers. We hypothesized that this might be a function of their breathing pattern and in reviewing the literature on mountain climbing noted that this had not been investigated. We looked at their bent-over posturing, their pursed-lip whistle, their elongated exhalations, their ability to sing and talk while carrying heavy loads and climbing, and their diaphragmatic movement as opposed to chest expansion as significant for their ability to adjust to high altitudes.

Two research trips over two years were conducted with 21 hikers trained for a three month period of time to breathe diaphragmatically and exhale slowly at rest and while exercising. Three trekkers were untrained. Both an oximeter and capnographer were used as biofeedback measuring devices at sea level and at altitude. Fat analysis and psychological profiling were also performed. The groups ranged in ages between 19 and 51.

The three untrained and youngest dropped out at 11,200 feet, one dropped out because of the flu and subsequent cerebral edema and three others because of sinus, respiratory problems, and general poor physical conditioning prior to leaving. The remaining 14 hikers maintained O2 levels in the 90's at 18,000 feet, were able to breathe away night-time headaches and exh ibited no Cheyne-Stokes breathing at night. There were no injuries and no muscle complaints.

The over all results were positive for those unable to take high altitude medications such as Diamox, but more so to explain how man can survive more positively at high altitude using proper breathing.

EMOTIONS AND STRESS IN ASTHMA: EFFECTS ON PULMONARY IN STANDARD LABORATORY SETTINGS AND EVERYDAY LIFE OF PATIENTS

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Objectives: It is well known from clinical reports that a variety of affective states c an precipitate asthmatic symptoms. However, little experimental work is available which compares direct effects of different emotional states on the airways, and nothing is known about the relevance of this experimental work for everyday life of patients. A study is presented in which we induced a number of different emotional states under controlled laboratory conditions, and studied the relationship of laboratory airway responses to the mood-related lung function changes of asthmatic patients in daily life.

Method: Participants (24 asthmatic, 24 nonasthmatic participants) viewed short film sequences pre-evaluated to induce anxiety, anger, depression, elation, happiness, contentment or neutral affective states. Oscillatory resistance (R os), heart rate, systolic and diastolic blood pressure, baroreflex sensitivity, skin conductance level, respiration rate, tidal volume and minute volume, and affective self-report were measured throughout the se ssion. Following the session, patients recorded their mood, shortness of breath, activities, and spirometric self-assessments three times per day for at least three weeks. Measurements of Forced Expiratory Volume in the first second (FEV1) were compared for episodes of strong negative, positive and neutral mood.

Results: During the laboratory session, Ros increased significantly during all emotional films compared to the neutral film in both groups. There was little relationshi p between changes in R os and other autonomic and ventilatory indices. FEV 1 measurements during daily life of asthmatic patients revealed a pattern similar to the laboratory, with a decline in lung function during strong negative and positive mood states as compared to neutral states. The decline in lung function during negative mood episodes was correlated with Ros increases elicited by negative films, particularly the depressive film, in the laboratory.

Conclusion: Experience of various emotional states can reduce pulmonary function of asthmatics under controlled laboratory conditions and in daily life. There is little evidence for a single explanation of this response characteristic of the airways in terms of ventilatory or autonomic adjustments. Responses to laboratory emotion induction by films can partly predict the mood-induced decline in lung function during everyday life of asthmatics.

THE INFLUENCE OF THE BACKGROUND NOISE INTENSITY ON RESPIRATORY, CARDIOVASCULAR AND ELECTRODERMAL ACTIVITY DURING PPERFORMANCE OF WORD RECOGNITION TASK

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Combination of mental stress task with noise background is a traditional tool employed in cardiorespiratory psychophysiology. However, intensity of background noise is a factor significantly affecting both subjective rating of experienced stress level during test and physiological responses associated with mental load in noisy environment.

In this study on 27 subjects we analyzed the influence of the background white noise (WN) intensity (55, 70, and 85 dB) on psychophysiol ogical responses during word recognition test.

Recorded were respiratory activity, such as respiration rate (RESP-R) and inspiration wave amplitude (RESP- A); electrodermal activity (skin conductance level - SCL, skin conductance response - SCR magnitude, number of SCR - N-SCR), and cardiovascular activity (heart rate - HR, HF/LF ratio of HRV - index of heart rate variability, respiratory sinus arrhythmia - RSA, pulse transit time - PTT, finger pulse volume - PV, skin tempera ture - SKT) during 40 s baseline resting state and 40 s long performance on 3 similar Korean word recognition tests with different background WN.

Electrodermal responses (SCL, SCR-M, N-SCR) and vascular responses (PV) demonstrated increment of reactivity with increased intensity of WN, and maximal magnitudes were characteristic of the highest intensity of noise (85dB). However, some cardiac and respiratory responses did not exhibit the same tendency of reactivity inc rease along intensity dimension, namely HR, HF/LF ratio of HRV, as well as RESP-R showed decrement of response magnitudes during 85dB background noise. RSA index significantly increased, normalized HF/LF ratio decreased in 85dB condition, whereas RSA decreased and HF/LF index increased in 55 and 70dB.

Important finding in terms of physiological reactivity was similarity of response profiles to 55 and 70dB WN, but 85dB WN resulted in significantly different profile of rea ctions (lower cardiorespiratory, but higher electrodermal and vascular responsiveness). Subjective rating of experienced stress during test clearly differentiated conditions with different intensity noise background, but stress ratings demonstrated significant negative correlation only with RESP-R and RESP-A, while failed to correlate with any other physiological variables.

Obtained results support the special value of respiratory variables as the indicators of stre ss and negative emotional states. Potential autonomic mechanism involved in mediation of observed physiological responses are sympathetic activation with parasympathetic withdrawal during mild 55 and 70dB noise, and simultaneous activation of sympathetic and parasympathetic systems during intense 85dB noise. Parasympathetic activation in this case might be a compensatory effect directed to prevent sympathetic domination and to maintain optimal arousal state for successful performance on mental stress task. Since 85dB noise was associated with higher subjective stress rating, it should be suggested that modeled acute stress episodes in our study were featured by _- and __adrenergic sympathetic arousal accompanied by parasympathetic activation and this finding is extremely important in understanding the roles of both autonomic branches and not only traditionally recognized sympathetic inputs contribution to development of stress syndrom e. Furthermore, obtained results partially supported Gellhorn's "tuning" phenomenon (Gellhorn, 1970) as a possible mechanism underlying unresolved stress.

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INDUCED EMOTIONS AND AIRWAY RESISTANCE - MEASURING THE RESPIRATORY IMPACT OF EMOTION LADEN MOVIE CLIPS BY WHOLE BODY PLETHYSMOGRAPHY

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Strong emotions are suspected to provoke an airway obstruction in asthmatic p atients. In a specific experimental context we tried to test this popular psychosomatic hypothesis.

It is a common experimental technique to induce short affective or neutral states by pictures or movie-/video-clips. In this study negative (sadness) and positive (amusement) affective states were induced by movie-clips lasting about 3 minutes selected according the results and recommendations of Gross and Levenson (1995). A screen saver was used to induce a neutral state.

Airway resistance was measured by whole body plethysmography (Jaeger Body Test): Airway resistance (Raw), specific airways resistance (sRaw) and thoracic gas volume (TGV) during a baseline prior to the movie presentations and after each presentation, sRaw immediately before and during movie presentation.

SRaw rised during all 3 mood inductions Mean Diff. (sRaw) = .032 kPa*s; ANOVA: $F = 11.00$, $p = .004$, Greenhouse-Geisser corrected. There was no effect of mood inductio n on Raw (Baseline: M = 0.28 kPa*l-1*s) and TGV (Baseline: M = 1.76 l). According to subjects' ratings the relevant target emotions (joy vs. sadness) were realized (joy M = 6.8, maximum = 8, s = 1.15, sadness M = 5.16, maximum = 8, s = 2.43). No specific affective state had been provoked while looking at the screen (mean of all affective ratings: M = .91, s = .81).

The results show an unspecific effect of looking at movie clips on airway obstruction. This is in con trast to the more specific effects we got in earlier studies, measuring continuously total respiratory resistance (TRR) by the forced oscillation technique.

THE MEASUREMENT OF BLOOD GASES BEFORE AND AFTER INCREMENTAL EXERCISE ON BICYCLE OF PATIENTS AND CONTROL PARTICIPANTS: SUPPORTING THE EFFORT SYNDROME HYPOTHESIS?

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Von Scheele & von Scheele (in press) reported a significant drop in patients but not in "normals" in blood gases, above all in Base Excess and HCO3- after exercise (Effort-Blood Gas-test) indicating Hyperventilation (HV) and depleted alkali system. HV "occurring out of appropriate context, under some circumstances, can be potentially harmful to health" (Fried, 1993, Nixon, 1994, von Scheele & von Scheele, p. 1, in press).

Method: Participants: 14 patients with various kinds of stress related problems received the test by clinical reasons and because of predictions about depleted alkali systems.

Result: reported here based on new measurement confirms von Scheele & von Scheele (in press) findings - pronounced change in patients' alkali buffering system. A "side-effect", EBG-data have influenced co-operations with referring physicians constructively.

Discussion: Reported data highlight the need for recovery and anabol process. Recovery in metabolic buffer systems can take hours to days to years if ever, depending on their actual state and treatment efficacy. Recovery means "back to" steady-state, but unfortunately, steady-state might have been reset to a biological dysfunctional level (von Scheele, 1987).

We regard patients' breathing behavior/physiology as a key and a tool for a better understanding of stress related dysfunctions.

It provides an out of system theory (Schwartz, 1982) perspective, base for diagnosis and training both on-line and overtime. Respiration is both the independent and dependent variable (Ley, 1994) depending on where you are positioned - and so is the patient (Kelly, 1955) (von Scheele & von Scheele, in press).

POSTER: CARDIORESPIRATORY REACTIVITY UNDER STRESS AND INDIVDUAL CHARACTERISTIC OF ALEXITHYMIA

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A principal characteristic of alexithymia represents the difficulty to express emotion verbally and personality trait observed in classical psychosomatic patients (Sifneos, 1972). There were some confusion in previous studies concerning the relationship between physiological reactivity to stress and characteristic of alexithymia. Although some studies indicated that high alexithymic subjects were characterized with high physiological arousal, other studies pointed out that high alexithymic subjects showed low arousal.

The present study was, therefore, designed to clarify the relationship between cardiorespiratory reactivity to laboratory stressors and individual characteristic of alexithymia.

Following a 20-min baseline rest, thirty healthy male and female undergraduates subjects worked on three presentations of mental arithmetic (MA) and of a cold pressor test (CPT) in a supine position. Continuous pneumotachography, fractional end-tidal CO 2, inductive plethysmography, arterial blood pressure, and ECG recordings were carried out with subjects in the supine position. All subjects completed the 20-item Toronto Alexithymia Scale (TAS-20) (Bagby et al., 1994) and the Minnesota Multiphasic Personality Inventory (MMPI).

Subjects were divided into a high alexithymia group and a low alexithymia group, based on the median value of TAS-20. Minute ventilation, mean flow rate, heart rate and mean blood pressure from baseline to MA and CPT conditions, whereas an index of respiratory t iming did not show any significant changes. The high alexithymic subjects scored significantly higher on the MMPI F, D, Pa, Pt, and Sc scales. However, changes in respiration rate and heart rate from baseline in high alexithymic subjects under MA and CPT were significantly less than in low alexithymic ones. There was no difference in mean blood pressure between two groups.

These results suggested that high alexithymic subjects were characterized with high subjective arousal and low cardiorespiratory reactivity.

IMPROVED METHODOLOGY FOR THRESHOLD DETECTION STUDIES IN ASTHMATIC CHILDREN

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Blunted perception of respiratory changes has been shown to be associated with near fatal asthma episodes. Threshold detection of added resistive loads offers a way to quantify a patient's perceptual ability, but methodologic issues complicate the interpretation of results. Although asthma and its management are especially important in the pediatric population, resistive loading studies need to account for large between - subject differences in airway size.

This paper compares two methodologies for thresh old detection of added resistive loads in children, reporting technical improvements that make the approach more reliable and clinically meaningful.

In both studies, resistive loads were presented as percentages of the subject's own intrinsic resistance according to Weber's Law, thereby controlling for variations in airway size between larger and smaller children.

The apparatus in Study I utilized laminar flow screens arranged in series to present various total resistanc es, while in Study II the apparatus utilized a servo-controlled cone that occluded an aperture to varying degrees (both systems are illustrated). Two protocols (systematic tracking and random presentation) resulted in two detection thresholds for each subject. Modifications in Study II included forced choice, attentional enhancements, and larger increments of added resistance. Subjects in Study I were 103 asthmatic children (mean age 10.8 yrs), and in Study II, 48 asthmat ic children (mean age 12.1 years).

Results showed the method in Study II to be superior. Tracking and random thresholds were achieved by 90 % and 83.3 % respectively of the subjects in Study II compared to 68 % and 48 % in Study I. The correlation between the mean tracking and random thresholds was .66 in Study II compared to .44 in Study I. Raw thresholds were highly correlated with intrinsic resistance in both studies (r =.49 - .89), supporting the importance of considering W eber's Law in resistive loading studies of children.

Standard methodology will enhance the comparability between studies. The high rates of children achieving thresholds in Study II show that children as young as 7 years of age can understand and complete resistance loading protocols. The high correlation between tracking and random thresholds in Study II supports the reliability of the method. Future research directions will be discussed.

BEHAVIIORAL AND RESPIRATORY MUSCLE RESPONSES TO INSPIRATORY RESISTIVE AND ELASTIC LOADS

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The mechanisms underlying load perception in humans remain in doubt although muscle, upper airway, and lun g proprioceptors likely contribute to the pool of sensory information used by humans to judge changes in the mechanics of breathing. Zechman and colleagues (J. Applied Physiology, 1985, 58, 236-243) showed that transdiaphragmatic pressure patterns exhibited graded increases during loading, and that the time at which such pressure changes significantly exceeded control levels decreased as detection latencies decreased with graded increases in either resistive or elastic lo ad intensities.

In this study, we recorded behavioral and respiratory muscle responses to inspiratory resistive and elastic loads to determine if the pattern of respiratory chest wall activity varied reliably with detection latencies.

Eight adult males ranging in age from 19-43 years ($M = 27.63$) participated in a single experimental session lasting two hours. Initially, subjects completed a health survey and performed spirometry. (All subjects were healthy and demo nstrated normal lung function: mean FVC, FEV1, and FEV1/FVC percent predicted scores were 100, 106, and 107, respectively.)

Subsequently, subjects participated in each of four experimental periods created by the combination of load type (resistive or elastic) and load size (small or large). In each period, subjects breathed on a mouthpiece and one of the four loads was delivered, once every to two five breaths without warning, for around 500 msec. Subjects were instructed to press a button as quickly as possible immediately upon sensing the load. Equivalent numbers of loads (about 40) were delivered in each experimental period. Peak mouth pressures were greater to larger loads than to smaller ones [$F(1,7) = 12.89$, $p<.01$] but were comparable for resistive and elastic loads.

As expected, resistive loads were detected more quickly than elastic loads [$F (1,7) = 25.78$, $p <.001$]; and, larger loads were detected more quickly than were smaller ones [$F(1,7) = 6.39$, $p<.05$].

EMG activity was recorded from pairs of surface electrodes positioned bilaterally on the chest wall near the 8TH intercostal space and on the non-dominant (usually left) forearm. The rate-of-rise (slope) in muscle activity was measured, in each subject, from the onset of loading to the onset of peak pressure, and was determined for both chest wall and forearm muscle groups. In each experimental period, levels of EMG activity recorded from right and le ft chest wall sites were significantly related; and, activity at both sites was related reliably to the rate-of-rise in peak inspiratory pressure. EMG activity levels were greater to larger loads than to smaller ones, but the ANOVA testing effects due to load size (large or small), load type (resistive or elastic), or electrode site (right or left), failed to yield a reliable effect for load size ($p = 0.19$). On the other hand, average chest wall EMG patterns exhibited graded increases during loading that varied in rate with increases in load size.

These latter observations are not inconsistent with the hypothesis that changes in respiratory muscle activity vary with load perception responses in humans.

RESPIRATORY SENSATIONS AFTER RESPIRATORY ENDURANCE TRAINING

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We have shown in several studies that respiratory endurance training (isocapnic hyperpnea) does not only incre ase respiratory endurance more than 6-fold but it also increases cycling endurance at a constant, submaximal load (65-85 % of peak power) by 28-50 % in healthy sedentary as well as in physically trained subjects. We could exclude changes in heart rate, stroke volume, blood lactate concentration and hypoxic drive (frequently occurring with 'normal' physical training) as being responsible for the improvements in endurance performance.

Thus, it seems likely that changes in the respiratory system itself are responsible for the improved physical endurance. This hypothesis is supported by the finding that the perception of respiratory exertion (PE resp) during exercise is significantly reduced (from 7 to 4.5 pts on a 10-pt modified Borg scale) after forty training sessions of 30 min duration while no change is observed (7 vs. 6 pts) in a control group without training.

This change in perception is not a result of concomitant respiratory strength trai ning (no change in maximal inspiratory and expiratory pressures).

Also, perceived effort while breathing through external inspiratory and expiratory resistances is not altered.

Thus, respiratory endurance training seems to decrease perceived respiratory exertion during high-velocity respiratory tasks only, i.e. voluntary isocapnic hyperpnea or exercise hyperpnea, while respiratory muscle strength and perception of resistive loads remain unchanged in healthy subj ects. The specific mechanism(s) underlying the changes in respiratory sensations and in endurance performance need to be investigated in further studies.