INDEX OF PAPERS and POSTERS

INTERNATIONAL SOCIETY FOR THE ADVANCEMENT OF RESPIRATORY PSYCHOPHYSIOLOGY ANNUAL MEETING 2000

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VA Palo Alto Health Care System, Palo Alto, California, USA
Paul Gross, Ph.D.
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SYMPOSIUM l
PANIC
Introduction

SYMPOSIUM 1: PANIC

Applying Respiratory Psychophysiology in the Clinic

Chair: Walton T. Roth
Participants: Frank H. Wilhem, Richard N. Gevirtz, Thomas Ritz

Applying respiratory psychophysiology to clinical problems has been stimulated by respiratory theories of anxiety disorders and by the awareness of psychological influences on lung diseases such as asthma .

Recent advances in laboratory and ambulatory recording of respiratory measures have shown that respiration is multifaceted and contains unsuspected psychological information about disease states and traits. The presenters will bring us up to date on four issues that have not been addressed adequately until recently.

- First, what is the relationship between hyperventilation or other respiratory disturbances and the currently accepted classification of anxiety disorders?
- Second, is it re ally possible to make sense of respiratory data gathered in non-laboratory settings?

- Third, is there any clinical value in analyzing heart rate variability, much of which is a function of respiration?
- Fourth, how much does emotional state account for respiratory resistance in normals and asthmatics?

Respiration is Altered in Anxiety Disorders After All

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Frank H. Wilhelm, Ph.D., Walton T. Roth, M.D., Georg W. Alpers, Dipl. Psych.

The hyperventilation syndrome clearly overlaps symptomatically with Panic Disorder (PD), but Garssen et al. (1996), using ambulatory transcutaneous pCO2 measures, found a decrease of pCO2 during only one of 24 panic attacks.The suffocation false alarm theory of panic has not been supported by observations of increases in pCO2 preceding attacks. Yet recent work from our laboratory does point to altered respiration in at least three circumstances.

Firs t, consistent with older reports we found that PD patients voluntarily hyperventilating to equal degrees of hypocapnia showed a slower rise of pCO2 back towards baseline than equally anxious Social Phobics or less anxious controls. This may be another example of PD patients' slower recovery from stress activation.

Second, when quietly sitting, PD patients tend to be hypocapnic compared to equally anxious patients with Generalized Anxiety Disorder or to less anxious controls. Oddly, this hypocapnia i s not related to increased mean minute volume but to an increased frequency of larger sigh breaths. Thus, hyperventilation does not have to be huffing and puffing. Breath-to-breath analysis implied lower, not higher, peripheral chemoreceptor sensitivity in PD.

Third, ambulatory monitoring of end-tidal pCO2 showed that driving phobics even without PD have lower pCO2s when driving; these levels tend to normalize with repeated exposure to the driving situation. Thus, ambulatory monitoring can document extent of clinical improvement and synchrony of change between self-reported anxiety and changes in respiration.

The Use of Heart Rate Spectral Data in the Treatment of Psychophysiological and Anxiety Disorders

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Modern technology has made possible the rapid calculat ion and display of component spectral bands present in the variation of interbeat intervals (IBI). The near simultaneous presentation of this data can allow the therapist or the client to see aspects of autonomic nervous system and respiratory patterns that may clarify possible mediators of symptoms.

Thus high frequency activity (.15-.4 Hz) is known to be modulated by the parasympathetic nervous system in conjunction with respiration, low frequency activity (.08-.14 Hz) by baroreceptor mediated blo od pressure changes , and very low frequency (.003-.07 Hz) by sympathetic visceral pathways.

While the measurement of these data in long epochs is well researched, little is known about the shorter dynamic changes that occur as a result of physiological or psychophysiological processes.

This presentation will illustrate both the promises and challenges of using spectral heart rate data in a dynamic application setting. Case examples will be presented to illustrate the richness of this data so urce and the specific methodological limitations of each frequency range.

Effects of Emotion on Respiratory Resistance in Health and Asthma: Arousal or Valence Modulation?

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Clinical evidence suggests that positive and negative emotion al states are capable of eliciting airway obstruction in asthmatic patients. As most experimental studies have focussed on negative emotional states, it is not clear whether the airways react to general emotional arousal or only to negative valence. Since comparisons with healthy control groups are rare, little is known about the disease specificity of induced airway responses to emotion.

Experimental emotion induction was investigated in five laboratory studies. Participants were healthy adult volun teers and asthmatic patients with mild to moderate disease. Emotions were induced by viewing and imagery of affective pictures, by self-referent statements, or by emotional film sequences. Respiratory resistance was measured continuously using the forced oscillation technique.

Three studies provided evidence for an arousal modulation of respiratory resistance, whereas two further studies showed that resistance increases were limited to negative emotional states.

Ventilatory mediators played onl y a minor role in shaping the observed resistance changes. No consistent relationship of airway response to cardiac vagal control was found across studies. In general, airway response was comparable in direction between healthy and asthmatic participants, with little differences in effect size.

I will discuss these results in terms of clinical significance of the emotion induction material, participants' familiarity with it, and clinical relevance of different parameters of the airway response.

Observing the Breath: Empirical, Interoceptive, and Introspective Reflections

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********** ABSTRACT PENDING **********

Suffocation and Respiratory Responses to Carbon Dioxide in Individuals with Panic Disorder

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Studies repeatedly show that individuals with panic disorder (PD) are highly reactive to inhalations of CO2. However, the reason for this hypersensitivity to CO2 among PD individuals has not yet been elucidated.

Given the primacy of CO2 in regu lating respiratory processes, several investigators have suggested that abnormal processing of CO2 and related mechanisms that underlie the experience of suffocation may be important in the development and/or maintenance of PD (e.g., Gorman et al., 1988, Arch Gen Psych, Vol. 45, 31-39; Klein, 1993, Arch Gen Psych, Vol. 50, 306-317).

Thus, the purpose of the present study was to examine the relevance of abnormal CO2 processing in panic. To this end, we compared the response to CO2 buildup between 40 individuals with PD and 32 healthy controls, using two techniques thought to increase central levels of CO2 (i.e., breathing cessation challenge and the Read rebreathing procedure).

We found that, compared with controls, individuals with PD had a greater ventilatory response to the Read rebreathing procedure, as well as a lower end-tidal CO2 value following the breathing cessation challenge, both of which are indicative of hypersensitivity to CO2 in the PD sample.

Individuals with PD also repo rted a greater experience of continuously measured suffocation and terminated the challenge procedures sooner than healthy controls.

Furthermore, within the sample of PD individuals, we found that those who terminated the rebreathing procedure prematurely had a greater ventilatory response to CO2 than those individuals who completed the entire rebreathing period.

These findings suggest the possibility for etiological heterogeneity within the PD population and a method to subdivide individual s with PD into more etiologically homogenous subgroups. Identifying more homogenous subtypes of PD with etiologically simpler networks might, in turn, simplify the search for clues to etiological factors that underlie the psychopathology of PD.

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Drop in end-tidal PCO2 during phobic exposure corresponds with increase in tidal volume

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We recently demonstrated the feasibility of ambulatory measurement of end-tidal PCO2 in driving phobics during exposure treatment. We observed greater hyperventilation in patients than controls, as evidenced by redu ctions in PCO2, but the source for these reductions was unclear.

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 3 sessions of exposure to freeway driving and 13 controls with 2 driving sessions in the same situations.

Results reported below were statistically significant. PCO2 levels dropped more in patients tha n controls during exposure (35.6 to 30.8 vs. 36.0 to 32.9 mm Hg). Both groups showed incomplete PCO2 recovery after exposure. Exposure PCO2 levels increased in patients from session one to two but not to session three (30.8, 32.8, 31.9 mm Hg) and approached levels of controls (mean of session one and two 33.2 mm Hg).

The inductive plethysmography data demonstrated that greater PCO2 reductions in patients than controls were caused by their greater minute ventilation response to exposure (4.57 to 6.83 vs. 5.49 to 5.37 l/min), which was mainly due to their increase in tidal volume (383 to 444 vs. 384 to 327 ml). Respiratory rate was increased to the same degree in patients and controls (13.5 to 18.0 vs. 16.3 to 18.2 breaths/min), making it unlikely that group differences in PCO2 levels were produced by incomplete measurement of end-tidal PCO2 plateaus due to shortening of the expiratory phase. With repeated exposure minute ventilation in patients approached values of controls (6.89, 6.35, and 5.72 l/min).

These data provide 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.

A new treatment for panic disorder: PCO2 feedback assisted respiratory training at home

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Stanford University and the Palo Alto VA Health Care System

Shortness of breath is a common symptom during panic attacks. Recent results from our and other laboratories indicate persistent respiratory anomalies in panic disorder patients even when they are not having panic attacks.

These include increased frequency of sighing and apneas, increased tidal volume variability, lower baseline pCO2, and slow pCO2 recovery after sighs and voluntary hyperventilation.

We developed a treatment that specifically targets these respiratory anomalies. During five weekly 1-hr treatment sessions, patients experience how their end-tidal pCO2 level, respiratory discomfort, and anxiety are affected by different breathing patterns (for example, combinations o f various depths and rates of breathing, sighing, breath holding, and abdominal versus thoracic breathing). Patients are taught the theoretical connections between anxiety and respiration and how to adopt a relaxed and effortless abdominal breathing pattern.

Between sessions, patients take home a small handheld capnometer for intensive practice of newly learned respiratory patterns. Patients practice breathing 20 minutes twice daily in synchrony with tape recorded tones modulated in pitch to indicate the timing for inspiration, expiration, and pauses. The patients' initial goal is to keep their pCO2 level constant during the paced breathing, and later in the training, to increase it by several mm Hg while maintaining a relaxed state.

Breath-by-breath respiratory rate and pCO2 levels are stored in the capnometer memory and reviewed with the therapist during the weekly treatment sessions. Anxiety, symptoms and breathing patterns (respiratory rate, tidal volume, end-tidal pCO2) are assessed before and after therapy using a 24-hr physiological monitoring system and diaries.

Initial results indicate that patients find the rationale of the therapy convincing, are highly compliant (which is monitored by the capnometer recordings during each exercise), and show systematic increases in baseline pCO2 and decreases in anxiety over the course of therapy.

The Treatment of Panic Disorder:
A Comparison Between Breathing Retraining
and Cognitive Behavioral Therapy

Beth Creager Berger, Ph.D and Richard Gevirtz, Ph.D.

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There has been an increase in interest over the past ten years in the respiratory aspects of panic. It has been thought that breathing ret raining would be effective in the treatment of panic if the respiratory aspect is a primary element in panic. However, few studies have looked at breathing retraining on its own in the treatment of panic.

Accordingly, this research was designed to determine if breathing retraining (BR), would be as effective as cognitive-behavioral therapy (CBT) in decreasing the frequency and severity of panic symptoms

Participants
Participants in this study were between the ages of 18 and 60 years. They responded to local advertisements or were referred by their physicians to the study. Eighty people were assessed with a diagnostic screening tool and 44 people were excluded after the initial screening based on the exclusion criteria. Of those accepted into the study, 14 females and 7 males completed treatment.

Measures.
The self report measures were the: panic attack journal, the panic disorder severity scale (pdss). Beck depression inventory (bdi). Physiological measurements were mad e with the C2SMO ETCO2/SpO2 Monitor model 7100 (capnometer) manufactured by Novametrix Medical Systems, inc., Wallingford, Connecticut.

If patients met criteria for the study after a 4 week baseline, they were randomly assigned to a either 6 weeks of BR or 10 weeks of CBT. At the first session, last session, and 4 week follow up, patients were again given all measures. Panic attack journals were given at the initial screening session, at the first session, and at the last session.

BR. The breathing retraining protocol was taken from DeGuire et al (1992). The BR sessions occurred once per week for approximately 30 to 60 minutes.

CBT. The cognitive therapy protocol used was a revised version of the Therapist's Guide for the Mastery of Your Anxiety and Panic II and Agoraphobia Supplement (MAP II Program) by Craske, Meadows, and Barlow (1994).

Results
The number of total panic attacks, the scores on the PDSS, the BDI, and respiratory rates did not yield any treatment by time interactions but all had a significant main effect for Time. There were no significant post-treatment differences between the treatment groups on any of these measures except the BDI scores for the CBT group, (significantly lower than the BR group).

The present study indicated that breathing retraining was as effective as cognitive behavioral therapy in the treatment of panic disorder.

The Treatment of Panic Disorder: A Comparison Between Breathing Retraining and Cognitive Behavioral Therapy

Beth Creager Berger, Ph.D.
Richard Gevirtz, Ph.D.
California School of Professional Psychology

The high incidence of panic disorder in our society today provides the impetus to study it. According to the National Comorbidity Study, 15.6% of the United States population experience a "sudden experience of unexplained fear over a lifetime" (Eaton et al., 19 94). The actual lifetime prevalence of panic disorder was found to be 3.5%. There has been an increase in studies over the past ten years looking at the respiratory component of panic(i.e. Hibbert & Chan, 1989; Papp et al., 1997; Roth, Wilhelm, & Trabert, 1998).

It has been thought that breathing retraining would be effective in the treatment of panic if the respiratory aspect is a primary element in panic. However, few studies have looked at breathing retraining on its own in the treatment of pan ic. Accordingly, this research was designed to determine if breathing retraining (BR), which affects respiratory functioning, will be as effective as cognitive-behavioral therapy (CBT) in decreasing the frequency and severity of panic symptoms. Additionally, it is thought that BR can be taught in a shorter amount of time than CBT thus reducing cost and treating symptoms in a more efficient manner.

METHODS

Participants

Participants in this study were between the ages of 18 and 60 years-old who were residents of the Greater Augusta, Georgia area. They responded to local advertisements or were referred by their physicians to the study. 80 people were assessed with a diagnostic screening tool and 44 people were excluded after the initial screening based on the exclusion criteria. Of those accepted into the study, 14 females and 7 males completed treatment.

Measures.

Panic Attack Journal. This was used to measure panic attack frequency. Each journal contained i nformation regarding the frequency of panic attacks, as well as the severity and number of symptoms for each panic attack.

The Panic Disorder Severity Scale (PDSS). The PDSS measures the frequency of panic attacks, distress during panic attacks, anticipatory anxiety, and interference in work and social functioning due to panic disorder symptoms. It assesses the overall severity of panic disorder. It ha good reliability and validity (Shear et al., 1997).

Beck Depression Inventory (BDI). T he BDI is an indicator of severity of depression. The BDI is a reliable and well-validated measure of depressive symptoms (Beck & Steer, 1993).

C2SMO ETCO2/SpO2 Monitor model 7100 (capnometer) manufactured by Novametrix Medical Systems, inc., Wallingford, Connecticut. The capnometer measures the amount of end-tidal CO2 (ETCO2) and respiratory rate. The samples of exhaled air were taken from a disposable mouthpiece.

Procedure and Design

At the initial screening procedure, informed co nsents were obtained and the patients were given the Research Participant Bill of Rights. Patients were also given a demographic questionnaire to fill out and all outcome measures were given. If patients met criteria for the study after a 4 week baseline, they were randomly assigned to a either 6 weeks of BR or 10 weeks of CBT. At the first session, last session, and 4 week follow up, patients were again given all measures. Panic attack journals were given at the initial screening session, at the first

session, and at the last session. After the 4 week follow up session, all participants were given the option of receiving the alternate treatment.

Treatment

BR. The breathing retraining protocol was taken from the DeGuire et al. study (1992). In that study, six sessions of breathing retraining were shown to significantly decrease respiratory rate and increase end-tidal CO2 in participants with functional cardiac disorder. The BR sessions occurred once per week for approximately 30 to 60 minu tes. Breathing retraining sessions consisted of the following:

1. The first session consisted of reviewing facts concerning respiratory physiology. A handout was given to each participant explaining the relationship between hyperventilation and panic disorder. Diaphragmatic breathing was then demonstrated by the therapist and practiced by the participants.
2. The next five sessions were used to practice correct diaphragmatic breathing. The sessions also focused on learning to breathe correctly while speaking and in other situations in which maintaining diaphragmatic breathing may be difficult.

CBT. The cognitive therapy protocol used was a revised version of the Therapist's Guide for the Mastery of Your Anxiety and Panic II and Agoraphobia Supplement (MAP II Program) by Craske, Meadows, and Barlow (1994).

The treatment package includes: (a) education about the nature and causes of panic; (b) self monitoring and awareness of cues; (c) identification of different res ponse components; (d) understanding the physiology of panic and learning physical control such as breathing retraining or relaxation training; (e) self statement analysis and cognitive restructuring; (f) prediction testing; (g) interoceptive and naturalistic exposure to feared physical sensations; and (h) how to maintain progress. The present study slightly modified the Craske et. al. treatment protocol by utilizing progressive muscle relaxation instead of breathing retraining. Additionally, instead of t hirteen sessions, the current treatment was given in ten sessions by combining some of the sessions together. The CBT sessions occurred once per week and lasted approximately 45 to 90 minutes.

Therapists.

There was a total of nine therapists who provided treatment to the participants. Three therapists provided breathing retraining, four provided cognitive behavioral therapy, and two led breathing retraining and cognitive therapy. The therapists' level of formal training included six master 's level clinical psychology students, one undergraduate psychology major, one psychiatrist, and one clinical psychology doctoral student. The primary investigator of the study trained each of the therapists and met with each therapist for 2 hours per week during the treatment phase to review the previous session and preview the following session. Additionally, the CBT therapists brought a script into each session with them in order to ensure thorough deliverance of the treatment.

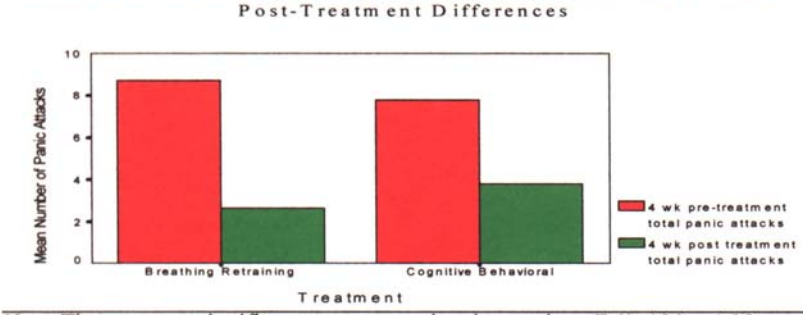
Results

Pr e-test differences. None of the differences between the two groups on demographic variables or dependent measures were significant. However, the ETCO2 levels in the CBT group were significantly lower at pretest than the BR group, which had within normal ETCO2 levels.

Panic frequency. The number of total panic attacks, the scores on the PDSS, the BDI, and respiratory rates did not yield any treatment x time interactions but all had a significant main effect for Time. There were no significant pos t-treatment differences between the treatment groups on any of these measures except BDI scores for the CBT group, which were significantly lower than the BR group. There was a significant treatment x time interaction for ETCO2 levels, and despite not receiving BR, the CBT group significantly increased their ETCO2 levels to the within normal range.

Figure 1

Figure 1

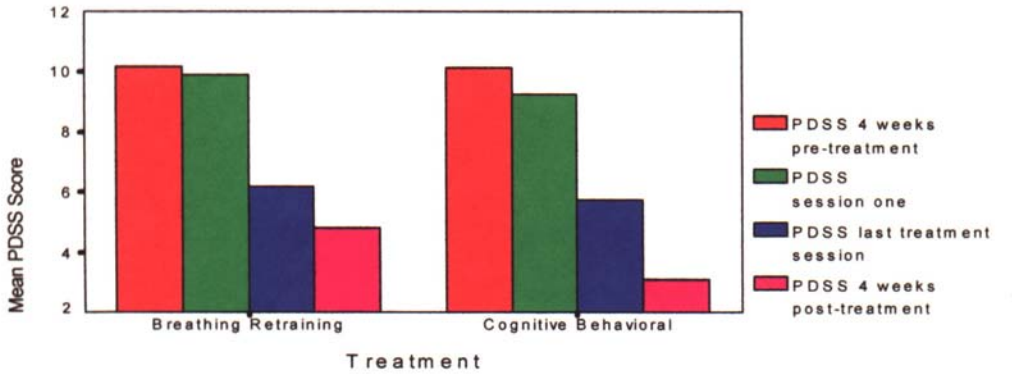


Note: There was no significant treatment x time interaction, $F(1, 19) = 1.38$, $p = .254$; There was a significant main effect for Time, $F(1, 19) = 32.18$, $p < .001$. There were no significant post treatment differences between the treatment groups in the total number of panic attacks, $F(1,19) = 1.61$, $p = .220$

Note: There was no significant treatment x time interaction, F (1, 19) = 1.38 , p = .254; There was a significant main effect for Time, F (1, 19) = 32.18, p < .001. There were no significant post treatment differences between the treatment groups in the total number of panic attacks, F (1,19) = 1.61, p = .220

Figure 2
Panic Disorder Severity Scale (PDSS): 4 Week Pre-Treatment, Session One, Last Session, and 4 Week Post-Treatment Scores

Figure 2
Panic Disorder Severity Scale (PDSS): 4 Week Pre-Treatment, Session One, Last Session, and 4 Week Post-Treatment Scores



Note. There was no significant interaction between the two treatment groups, F (3,19) = .57, p = .640. A main effect was found for Time, F(3,19) = 40.17, p <.001. There were no significant post treatment differences between the treatment groups when measured at the last treatment session or 4 weeks post-tre atment, F (1,19) = .067, p = .799; F (1,19) = .350, p = .561, respectively.

Discussion

The present study indicates that breathing retraining may be as effective as cognitive behavioral therapy in the treatment of panic disorder. Analyses showed that both treatments were successful in significantly decreasing the frequency and severity of panic attacks as measured by the Panic Disorder Severity Scale and as measured by a self report panic attack journal. Furthermore, both g roups showed significant decreases in symptoms of depression as measured by the Beck Depression Inventory. There were no significant differences between the groups on any of the panic attack outcome measures at post treatment or at a four week follow up and effect sizes were essentially the same for both groups. The large treatment effects paralleled previous studies (i.e., Arntz & Van Den Hout, 1996, Clark et al., 1999). The CBT group did have lower BDI scores than the BR group at four weeks post-treat ment indicating that CBT may be more effective at decreasing depressive symptoms than BR.

Having a better understanding of what mechanisms are primarily responsible for panic attacks allows for more effective treatment of panic. If it is accepted that the respiratory component of panic disorder is essential and breathing retraining is effective early in treatment, patients can experience greater relief in a more rapid manner than with cognitive behavioral therapy or even medication therapy. Patien ts may also be more accepting of breathing retraining as a method of treatment because they may understand the disorder in a more physiologic manner rather than feeling as if there is something psychologically wrong with them. Subsequently they may practice the breathing techniques more than they would engage in CBT homework. It may be that medication and/or a cognitive component of treatment could add benefits in addition to the breathing retraining. But again, the breathing retraining could be used to help provide immediate relief and then other components of treatment could be followed.

It is our position that panic may be due in part to some faulty breathing pattern and that breathing retraining serves to correct this. Therefore, instead of merely being a distracting coping mechanism, breathing retraining may actually change breathing patterns which may affect respiratory physiology, such as stabilization of the autonomic nervous system, ETCO2 levels or stability of respiratory rate. Subsequen tly panic attacks are less likely to occur in the future.

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Resonant Frequency Heart Rate Variability Biofeedback: Effects on Cardiovascular and Baroreflex Function

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It is known that respiratory activity continually perturbs cardiovascular hemodynamics. Respiration modulates the activity of most sympathetic and parasympathetic efferents. The autonomic efferents in turn mod ulate heart rate (HR), arterial and central venous pressures, and peripheral vascular resistance with respiratory periodicities.

The changes in arterial pressure, arterial PCO2, PO2, and pH, which result from the changes in respiratory activity, modulate the activity of both the autonomic and central nervous systems through afferent impulses from baro and chemoreceptors. These complex and constant effects of respiration actively affect human condition.

We found previously that human car diovascular system has resonant features. Each person has a specific resonant frequency in the range of .055 - .12 Hz. Breathing at resonant frequency causes high amplitudes of both heart rate (HR) and blood pressure (BP) oscillations.

We hypothesized that breathing at resonant frequency trains the reflexes of cardiovascular system, in particular, the baroreflex.

We taught ten healthy subjects to increase the amplitude of HR oscillations using breathing at resonant frequency and HR bio feedback. Each subject took ten twenty- minute biofeedback sessions over a 1.5 2 month

period and practiced for forty minutes daily at home (two sessions of twenty minutes each) using a home trainer biofeedback device. Four of these sessions involved complete physiological assessments, as described below.

An additional seven subjects constituted a control group. They received four twenty- minute record sessions within a period of 1.5 2 months. During each of the four recording sessions, we rec orded respiration, HR and beat-to-beat BP during each session and 5 minute before each session (base line).

We found the following significant changes among subjects receiving biofeedback between the baseline of the first session and the baseline of the last: systolic BP decreased from 110.3±11 mmHg to 99.7±10 mmHg ($P<0.01$); diastolic BP decreased from 66.3±7.9 mmHg to 59.6±8.7 mmHg ($P<0.03$); respiration total spectral power decreased from 59.25 arb2 to 42.60 arb2 ($P<0.05$); HR total spectral power i ncreased from 3910 ms2 to 5378 ms2 ($p<0.02$); systolic BP total spectral power increased from 26.1 mmHg2 to 36.3 mmHg2 ($P<0.01$); baroreflex gain increased from 12.0±4.0 ms/mmHg to 14.0±5.9 ms/mmHg ($P<0.05$).

Averaged across the 10 subjects, baroreflex gain during biofeedback training was 17.9±6.8 ms/mmHg. None of these measures changed significantly for subjects in the control group.

In conclusion, we found evidence that breathing at resonant frequency, assisted by biofeedback for increasing amplitude of respiratory sinus arrhythmia, increased variability of cardiovascular function, increased baroreflex gain, and decreased BP.

Psychophysiological Effects of Hyperventilation on Children and Adolescents at Different Ages

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********** ABSTRACT PENDING **********

A Breathing Pattern Training and Portable Activation System: Use in the Rescue Breathing Pattern in COPD

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A visual biofeedback breathing pattern training system has been described at the 1995 ISARP meeting. Briefly, by menu selection of pulmonary parameters an infinite variety of breathing patterns may be generated. The display is auto corrected to a full volume-time display, and a cursor flashes across the programmed analog in the right time domain to prompt patient performance. In real time the patient sees their breathing performance and thereby has a visual biofeedback signal to correct performance.

It is theorized that the auto scale display is a particularly effective training tool as it forces the patient to focus on feeling the desired performance.

In a COPD pulmonary rehabilitation program patients were given a copy of their desired breathing analog with instructions to practice twice daily for five minutes, and to "put the picture in your mind" while practicing breathing exercises. With this minimal program most patients were following the desired breathing pattern within two to three m onths. Part of the program was visual instruction on lung deflation to correct air trapping and overinflation. Long term retention of the learned breathing pattern was variable, and use of the learned breathing pattern was likewise variable and anecdotally successful.

Provision of audio prompting at beginning inspiration ("beep" high pitched) and expiration ("beep" low pitched) provided a prompt which enabled patients to reproduce the desired breathing pattern without the visual prompt. It is theo rized this simple audio prompt is activating the complex visual learned breathing pattern.

A portable audio and simple LED prompting device has been developed.

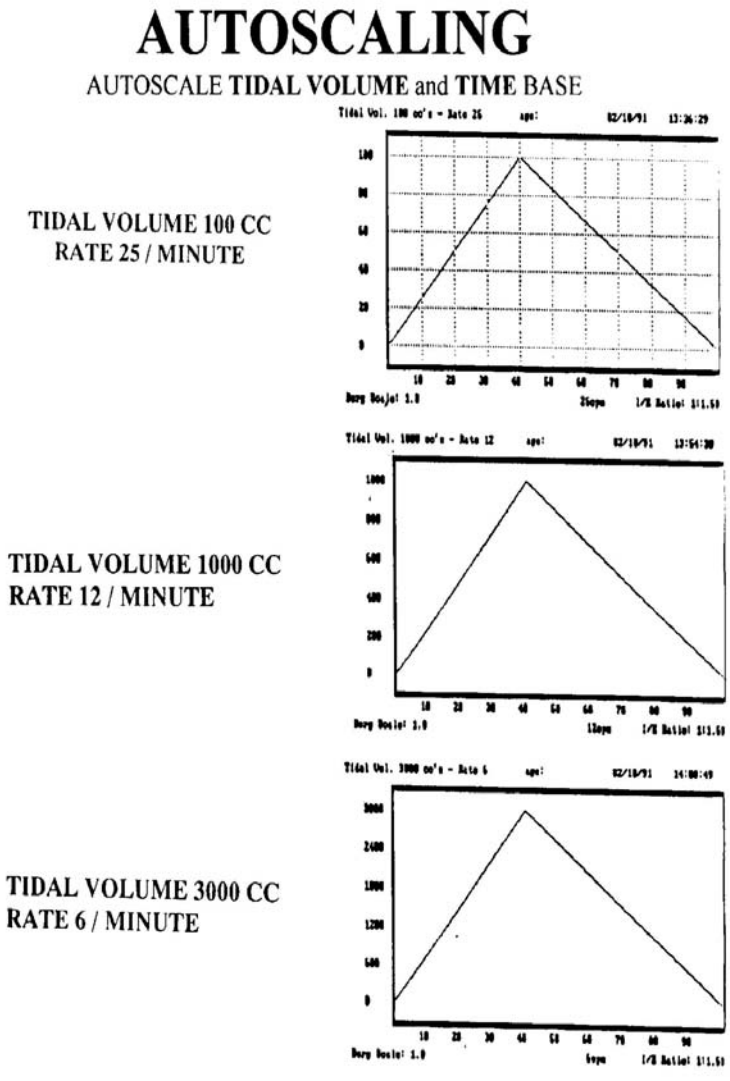
Conclusion: A breathing pattern training and portable activation system has been developed for COPD, and has implications for Hyperventilation Syndrome control.

EXPANDED ABSTRACT

At the 1995 ISARP meeting in Toronto the setup and u se of a computer based visual biofeedback training system was presented, and therefore will not be elaborated on in this presentation (Copies of 1995 handout available - See: Hillsman, ISARP 1995 or http://www.sierrabiotech.com in the Biofeedback Incentive System (tm) section).

The 1995 presentation featured a version achieved in an IBM XT computer. Briefly, by menu selection an infinite variety of inspiration and expiration breathing patterns may be displayed on a computer CRT. A cursor blinks along the programmed line, and the patient attempts to match their real time performance with the programmed line.

Performance deficiencies, such as the slow inspiration rate shown in the diagram, are immediately apparent and the patient is therefore given a visual biofeedback s ignal to correct their training performance.



AUTOSCALING

Note the three breathing patterns are identical, despite the fact that the Respiratory Rates and Tidal Volumes are very different.

This is due to the autoscaling display.

Autoscal ing forces the patient to focus on internal sensing of the biofeedback experience.

It is theorized this is the reason why this type of visual biofeedback

breathing training is particularly effective.

THE RESCUE BREATHING PATTERN

The Rescue Breathing Pattern is a cognitive universal response to acute dyspnea, o r a dyspnea exacerbation of chronic dyspnea. It is a modulator response superimposed on the physiologic reflex breathing control mechanisms.

Simply stated, it is to "pump air in and out of the lungs as hard and as fast as possible" in an conscious attempt to relieve dyspnea distress.

The Rescue Breathing Pattern is as follows:

- Increased Respiratory Rate
- Increased Tidal Volume (Rate limited)
- Increased air Flow
- Forced breathing
- Focus on In spiration
- Shortened Expiration Time

The RBP leads to Air Trapping and functional Overinflation due to:

- Necessary long time constant for expiration
- Increased dynamic bronchial compression

Overinflation places the chest wall and respiratory muscles in a position of mechanical disadvantage, and this will acutely exacerbate dyspnea.

In COPD / Emphysema / severe Asthmatic the patient must breathe:

- Slowly
- Large Tidal Volume
- Gently
- Focus on Expiration
- Increased Expiration Time

Unless these patients breathe in this manner they will develop Air Trapping and functional Overinflation.

These requirements are 180 degrees out of phase with the RBP, and therefore the Rescue Breathing Pattern in these conditions is a pathological and corrupt reflex action.

The RBP **is** an appropriate response in Restrictive Lung Disease.

<C

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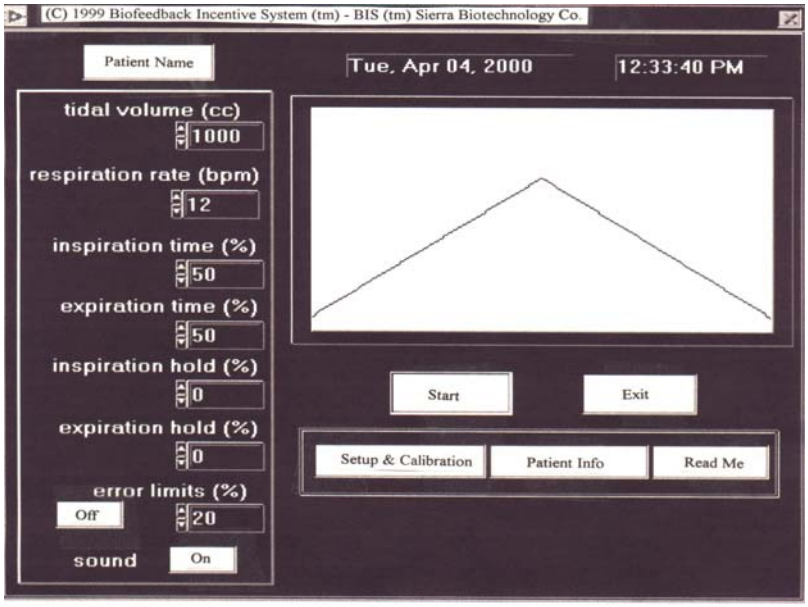
Return to ISARP HomePage ENTER>**LabVIEW VERSION OF VISUAL BIOFEEDBACK TRAINING SYSTEM**

A copy of this program may be downloaded



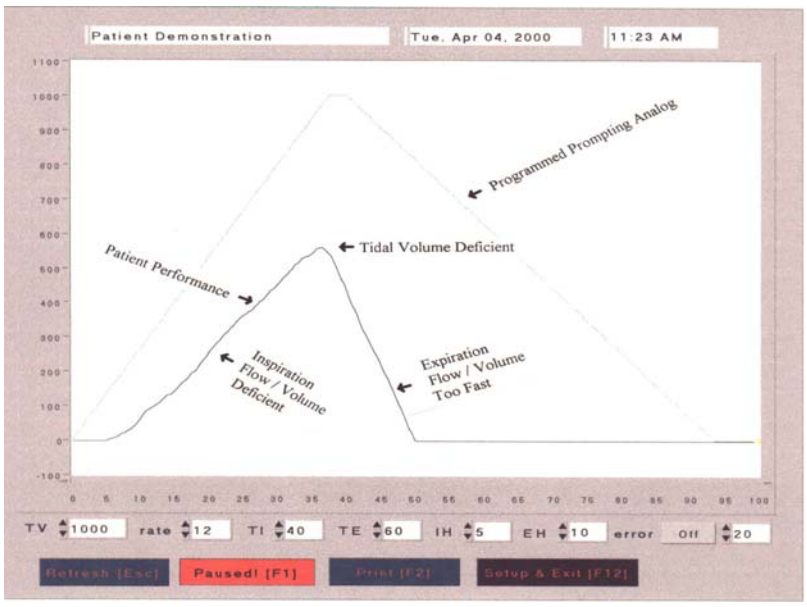
This is the setup screen.
As the various parameters are changed by using the up or down indicators, or entering a number, the waveform changes appropriately. Note the patient's name may be e ntered. Date and time are automatic.

When ready to proceed to the working displays click on the "Start" button.

Note the Tidal Volume (y axis) always appears at the same height, and the Respiratory Rate (x axis) is always the same at 100 units.

An Inspiration Hold a nd Expiration Hold may be selected.

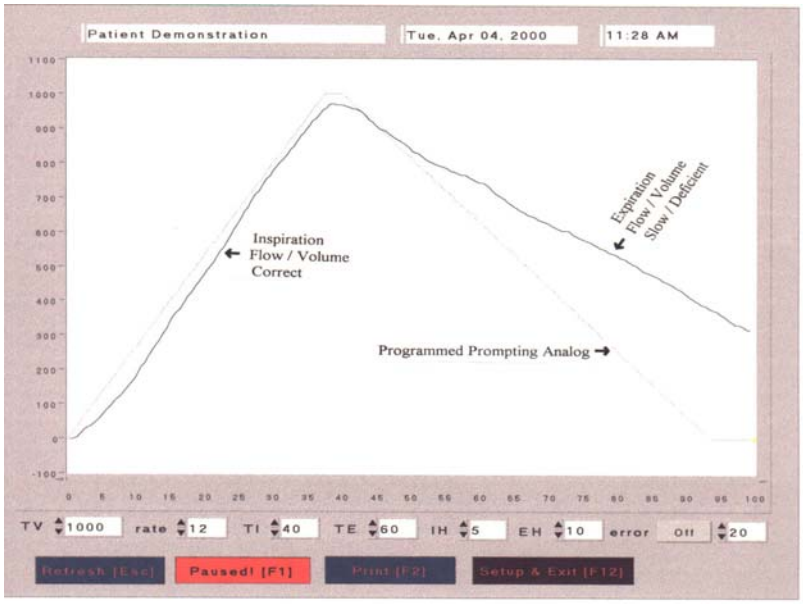
Note the abbreviated controls at the bottom permit dynamic waveform adjustment during the training session.



Note the patient performance line contrasted to the programmed line. Inspiration is deficient and Tidal Volume was not achieved.

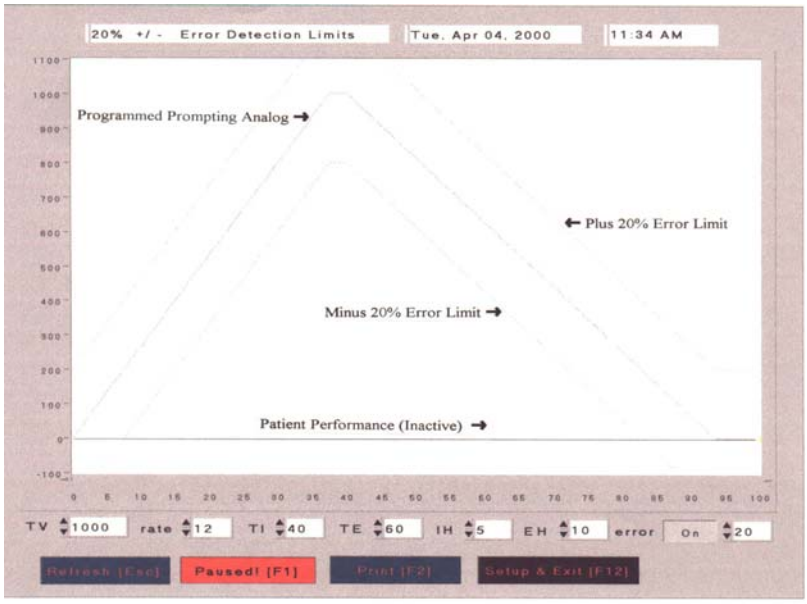
Note the expiration slope is too steep,

i.e. expiration was too fast.



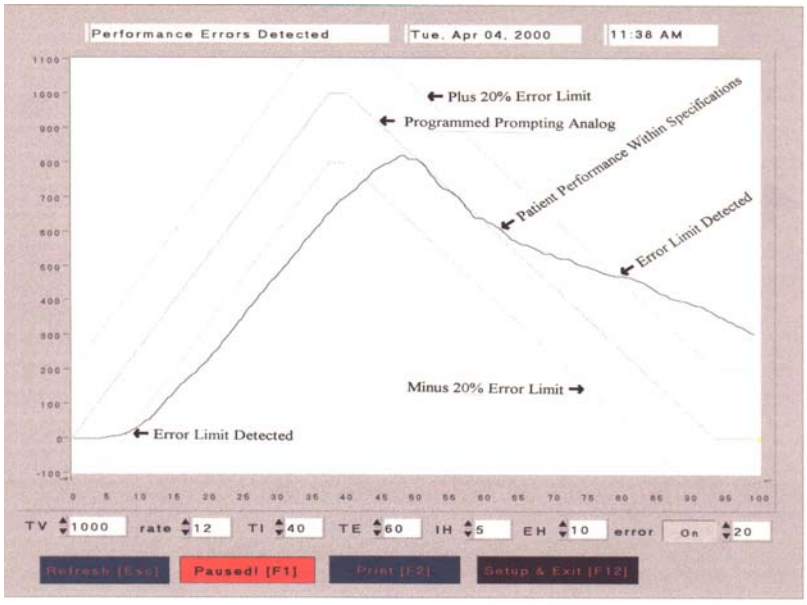
Note that inspiratory flow is almost perfect, and Tidal Volume has been achieved.

However, expiratory flow is too slow, and the patient has not exhaled back to resting Functional Residual Capacity.
This is Air Trapping.



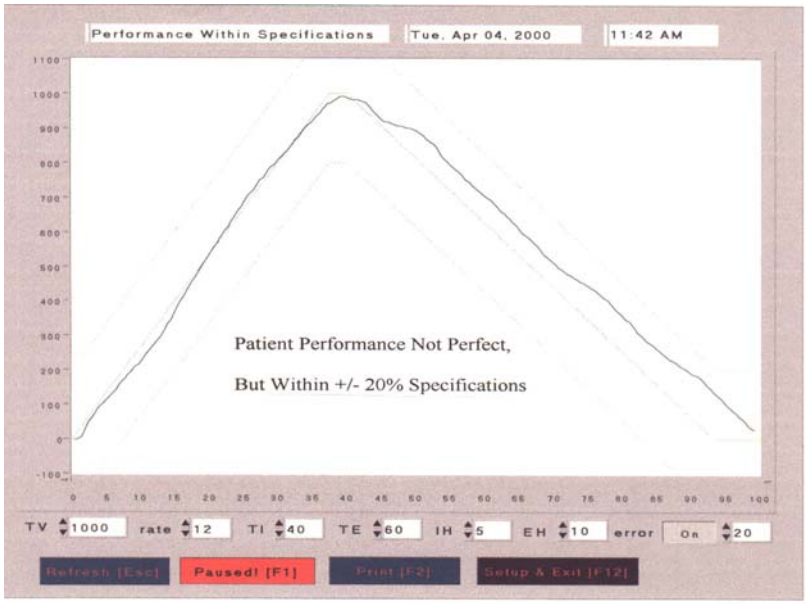
Note that plus and minus error limits may be defined, above and below the programmed line. These limits may be adjusted.

This is a quality c ontrol means to grade patient performance, or assure data input in experimental situations.



Note the error detection (which optionally may be indicated by a beeping sound) in early inspiration.

Briefly the patient is on track, but then falls outside error limi ts on expiration due to slow exhalation.

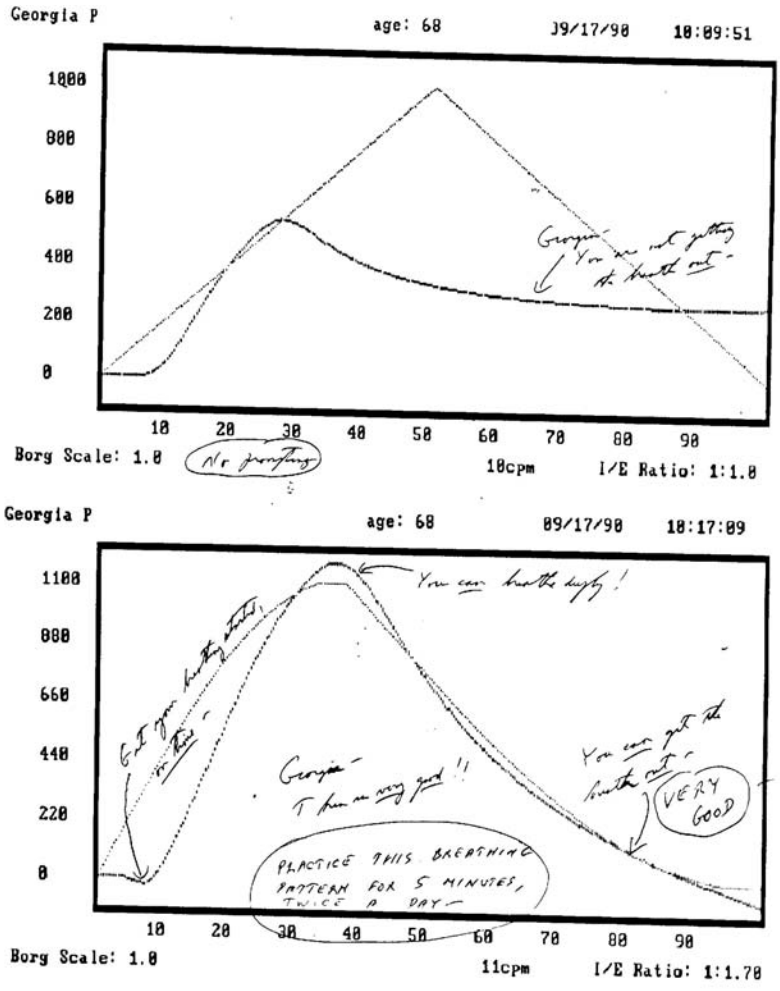


Note that inspiration performance and achieved Tidal Volume are almost perfect.

Expiration performance is not as perfect, but is within the +/- 20% error limits as defined.

For research purp oses with stringent control needs a +/- 10% or less error limit might be set.

PATIENT SELF TRAINING



These pictures are "screen dumps" in the original training system of an actual patient training session, in this case the patient's first session.

The upper record was obtained with the patient's screen blinded, in order to obtain a record of the natural breathing pattern. For comparison purposes the Tidal Volume is fixed at 1000 cc and Respiratory Rate at 10 breaths per minute.

Note the Tidal Volume of about 500 cc, and despite a prolonged expiratory phase, the patient not exhaling down to baseline FRC. This is Air Trapping.

The lower record obtained 8 minutes later shows the patient's breathing prescription, and other than for a slight delay in starting inspiration, a good match with the programmed analog breathing prescription. Tidal Volume is now about 1150 cc and lung deflation to FRC has been achieved. Annotations have been made to point out deficiencies and desired performance, and encouragement.

The patient is given a copy of their record, and with the following instructions:

Practice your breathing twice a day, for five minutes (but no more, to avoid fatigue and bore dom). Sit in a comfortable chair. Relax. Concentrate. While doing your breathing exercises, **put the picture in your mind.**

Usually most patients demonstrate substantial alteration of their breathing pattern toward the breathing prescription within two to three months. By about the third or fourth month many spouses report patients breathing in the programmed manner while sleeping. Retention of the learned breathing pattern is variable, usually requiring two or three reinforcement sess ions per year, though some patients retain their learned breathing pattern for a year or longer.

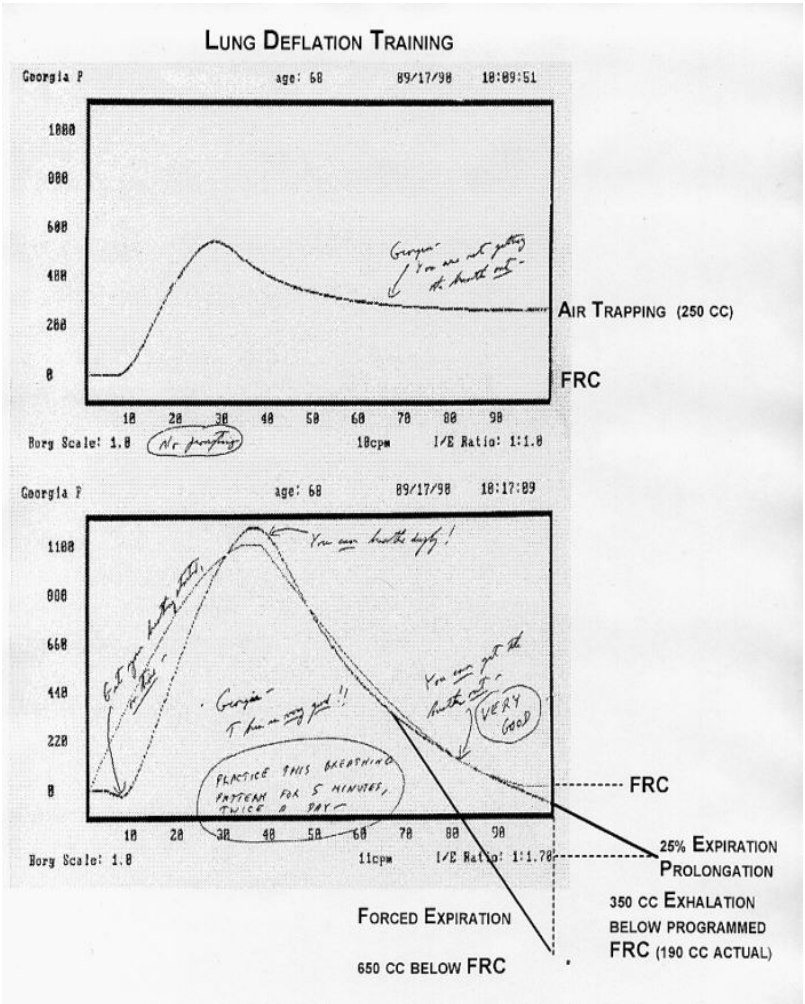
LUNG DEFLATION TRAINING

The top record shows the same patient demonstrating Air Trapping.

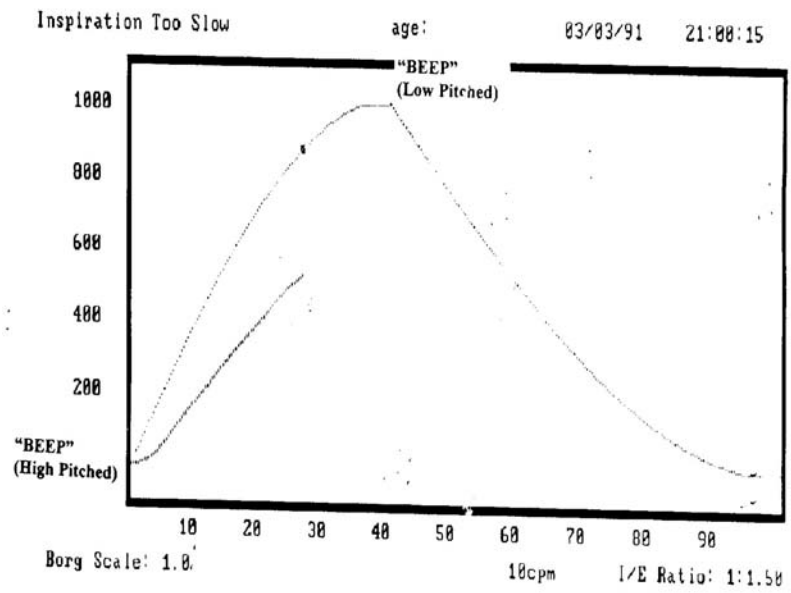
The bottom record shows two methods of lung deflation training.

The first technique is an approximate 25% prolongation of the programmed expiratory time. The example shows about 350 cc exhalation below the FRC. This technique is good to gradually deflate the lung over several breaths. It can be used to advantage at the onset of a dyspnea attack.

The second technique is a rescue technique for patients acutely distressed. The same respiratory timing is used, but approximately 1/2 to 2/3 of the way through expiration the patient forces expiratio n to a point below FRC (up to this time, patients are trained to relax as much as possible with passive exhalation). Note the expiratory forcing must be a controlled gentle action, in order to minimize the dynamic bronchial compression problem and further exacerbation of airway collapse. Done correctly, usually within 5 to 10 breaths the patient feels substantial, though not complete relief of their dyspnea attack. They can then use their regular breathing pattern to regain complete comfort over the next fe w minutes.



AUDITORY PROMPTING



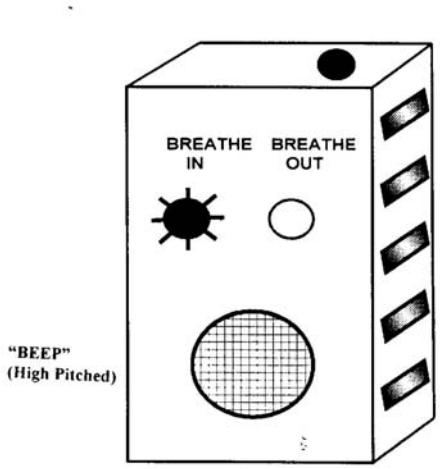
An optional audio prompt is available in the visual biofeedback training system. This consists of a "beep" (high pitched) at the beginning of inspiration, and a second "beep" (low pitched) at the beginning of expiration.

If the patient tr ains with both visual and audio prompts, and the visual prompt is turned off, the patient will use this simple prompt to reproduce the desired breathing pattern. This is an adjunct for patients having difficulty using, or retaining their breathing patterns.

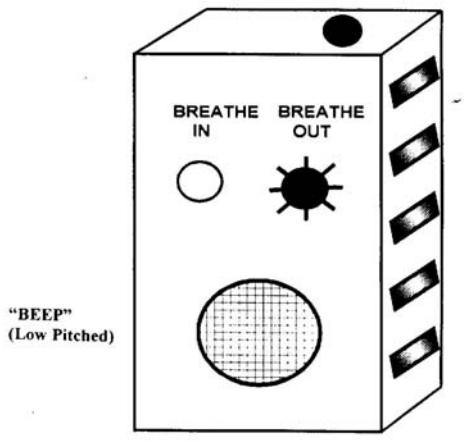
It is theorized that patients learn complex breathing patterns from the visual biofeedback training system, and that these complex breathing patterns are activated by the simple audio prompt.

PORTABLE PROMPTING DEVICE

A portable prompting device is in development. A schematic design is as noted.



Inspiration and expiration LEDs activate with the appropriate "beep" sounds. A series of control switches set the appropriate timing signals, set sound level, and turn off the sound. Separately there i s a manual override button which causes a 25% prolongation of the expiratory time for lung deflation. During this time the expiratory signals blink and "beeps" continuously, to focus attention on expiration.



When activated the prompting device works as noted.

Th e inspiratory LED activates with a high pitched "beep."

The expiratory LED activates with a low pitched "beep."

CONCLUSION: A comprehensive system to train patients in therapeutic breathing patterns, and to activate those complex learned patterns under operative conditions in the field with a simple portable device has been achieved.

Diagnostic Tools for Analysis of Dysfunctional Breathing and its Effects on Psychophysiological Systems

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Introduction
During cognitive appraisal of threat, hyperventilation (HV) might directly be triggered (von Schèele & von Schèele, 1999) which in turn directly might lead to cerebral vascular constriction reducing available oxygen and thereby hypoxia (Fried, 1993, Lum, 1984).

Probably HV can also be triggered by the orienting response in some patients, at least patient reports indicate this.

In some of our patients we not only observe hypocapnea (low ETCO2) and oxygen compensation (SaO2 99-100%) but also depletion of alkalical systems (von Scheele & von Scheele, 1999)) as well as dysfunctional intra cellular bu ffering (measured indirectly extra cellular).

The observations above indicate that for adequate stress profile analysis and proper diagnosis in severe, patients suffering from stress chronic related disorders not only electro-physiological but also respiratory and metabolic parameters are of crucial importance in the analysis - and this is not only for disorders related to brain functioning, e.g. panic and epilepsy but the whole field of stress related disorders. The dysfunctional effects from the sy stems involved may even be synergistic.

Recently also interest is developed concerning insulin resistance (related to influence from low intra cellular Mg and increase in Ca as well as food intake and absence of exercise) and glucose sensitivity.

In von Scheele & von Scheele (1997) hypoglycemia and anxiety was discussed referring to Lum (1994) and Guyton (1991) and its relation to dysfunctional breathing.

To combine effective diagnostic which gives concrete guidelines about treatment st rategies we have developed a measurement system which is easy to use and which also analyze data and give interactive suggestions which can increase of the effect of a patients training.

Results: Data from clinical testing is review from different patient population from tortured victims to patients with mild stress related dysfunctions where diagnostics suggest different treatment program and where post test clearly show the result of the treatment.

Respiratory and Auditory P300 Amplitudes are Diminished in Asthmatics

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Ian M. Colrain, Ph.D.
Department of Psychiatry and Behavioral Sciences, Stanford University and
Department of Psychology, The University of Melbourne.

The present study investigated the respiratory-related evoked potential (RREP) in 16 asthmatics and 16 matched controls. The goal was to evaluate the relationship between respiratory sensitivity and the P3 component.

It was hypothesised that asthmatics would be less sensitive to respiratory stimuli and have reduced P3 amplitudes.

Additionally, auditory evoked potentials (AEPs) were recorded from an oddball paradigm in both groups and compared with the RREP results.

The perceptual sensitivity of subjects was assessed by means of a load magnitude estimation task. Four ma gnitudes of resistive loads and no-load were applied for a single breath 10 times each in random order. Respiratory sensitivity was estimated using a modified Borg scale.

Results showed little difference in perceptual sensitivity between the groups and in both groups resistive load was linearly related to magnitude estimation (asthma, R2=0.96; control, R2=0.99).

For RREP assessment, inspiratory occlusions were presented for 500ms. For AEP assessment, 60dB 1000 and 2000Hz pure tones were presen ted for 100ms, with the 2000Hz tone presented with a 20% probability (target). Scalp EEG was measured from 29 channels.

Results showed that both the respiratory and auditory P3 components were markedly reduced in the asthmatic group compared to the control group (reduction of »30%). All other components were similar between the groups.

These results suggest an intrinsic reduction in P3 amplitude for asthmatic patients. This may relate to differences in processing perceptual information. Import antly, whatever the mechanism of the P3 deficit, it is not specific to occlusion stimuli.

Respiratory responses to emotional film viewing and paced breathing in panic disorder and major depressive disorder

Wilhelm, Frank H., Rottenberg, Jonathan; Roth, Walton T.; Gotlib, Ian H., & Gross, James J.

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Recent experiments demonstrated respiratory regulation anomalies in panic disorder (PD) patients during baselines and anxiety provocations. Specifically, tidal volume instability was greater in PD patients than in an equally anxious clinical control group or healthy controls, indicating that this measure might be a diagnostic marker of PD.

In the present study we sought to elucidate the interactions of diagnosis and emotional state by experimental induction of several moods (neutral, fear, sadness, amusement) using film scenes.

We compared PD patients (N=16), major depressive disorder patients (MDD, N=69), and psyc hiatrically healthy controls (N=29). In another task, we asked participants to breathe at fixed rates of 9, 13.5, and 18 cpm. Respiration, recorded unobtrusively with inductive plethysmography, was submitted to a detailed analysis of its volume and timing.

Instability of tidal volume and respiratory rate was assessed by complex demodulation. At baseline, anxiety levels in PD and MDD patients were only moderate (2.0 on a 0-8 scale), but significantly higher than in controls (0.6). Groups did not di ffer in any of the respiratory measures including tidal volume instability.

In response to the film scenes, all groups showed similar increases in fear, sadness, or amusement, depending on the scene. Varying the speed of paced respiration produced changes in several respiratory measures, but these changes did not differ between groups.

We conclude that tidal volume instability is not a definitive diagnostic marker for PD. Mood changes in PD patients lead to respiratory changes that are equiva lent to those in MDD patients and controls.

Respiratory anomalies in PD patients may be present only when suffocation fears are triggered by the experimental condition or measurement devices, at certain levels of anxiety or hypocapnia, or after subjects have been sitting for extended periods.

The Role of Hyperventilation in Asthma

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********** ABSTRACT PENDING ********** **

Hyperventilation and hypoventilation during emotional experiences

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Our recent studies on respiration and emotion revealed that inspiratory flow rate (respiratory drive) significantly increase during psychological stressors such as mental arithmetic, a cold pressor test and video-games, and also that duty cycle (timing) and gas exchange (transcutaneous pCO2) maintain stable during stressors.

Psychophysiological detection of deception is a method of determining when an individual is lying. It has been studied for a long time. It is well known that deception is accompanied by large electrodermal responses reflecting increased sympathetic nervous system activity. Because lie detection is stressful, it could be hypothesized that an index of r espiratory drive should increase significantly during this task.

To test this hypothesis, we conducted one experiment to compare the differences between respiratory changes under detection of deception in a mock-crime situation and ones under laboratory stress.

Contrary to our expectations, results showed that breathing patterns under the detection of deception situation were quite different from ones under stress. Expiratory volume and minute ventilation in response to critical questions were significantly smaller than ones in response to non-critical questions. An index of respiratory drive did not show any significant increments.

The results of this experiment suggest that respiration during deception is characterized by inhibitory breathing. Our results also suggested that emotional experience can induce hypoventilation as well as hyperventilation. Additional research will be necessary to clarify which feeling induces facilitation of breathing and which feeling induces inhibito ry breathing.

EDITING CRITERIA FOR COMPUTERISED ANALYSIS OF END-TIDAL PCO2 MEASURED BY AN AMBULATORY CAPNOGRAPH

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End-tidal me asurements of PCO2 are widely used as substitutes for arterial PCO2 in normal subjects but there is little systematic data comparing the two.

End-tidal measurements can be easily invalidated by poor capnograph response time and shallow breathing with failure to clear the dead space. Editing by eye is often perfunctory and tedious or is not attempted, especially over the long runs produced by ambulatory capnography with data collection either by telemetry on-line or by a data logger off-line.

We have developed a computer program sampling a PCO2 trace at 50 Hz which detected a breath if PCO2 was below a variable threshold of about 8mmHg for more than 1/3 second, measured end-tidal PCO2 (PETCO2) by a peak reading algorithm, simultaneously measured two values 0.25 and 0.5 sec back from the end-tidal value as measures of alveolar plateau slope, and approximately measured inspiratory (TI) and expiratory times (TE) as the times between the up and down legs of the trace.

Breaths were rem oved if the slope values were more than 2.5 and 5 mmHg respectively below PETCO2, if TI or TE were less than 0.9 sec or greater than 10 sec, if TI was more than 50% greater than TE, and/or if PETCO2 was more than 5 mmHg below adjacent values. All of these thresholds could be 'fine tuned' for individual subjects.

We validated this with two protocols of resting breathing and 15 minutes of formalized speech as representing the severest test of editing. We studied 11 normal subjects and compared 121 ar terialized venous PCO2 (PavCO2) samples with the computer detected end-tidal data with and without automated editing.

Without editing, PETCO2 was significantly different ($p<0.001$) from PavCO2 (mean ; 30.1 (SD 6.4) mmHg and 34.7 (SD 4.2) mmHg respectively) and the limits of agreement were wide; - 6.6 to 15.8mmHg. With computer editing, PETCO2 estimated PavCO2 to within 0.5 (SD 1.0) mmHg with limits of agreement of -2.5 to 1.5 mm Hg and there was a 99.8 % agreement with editing by eye over a tota l of 6684 breaths with a rejection rate of 49%. Valid measures could be obtained in speech as long as more than 6 valid end-tidal values remained per minute after editing.

Thus, sophisticated editing of end-tidal traces is essential and can be performed with a high degree of accuracy by an automated computer system, even during speech.

THE RANGE OF END-TIDAL PCO2 IN NORMAL SUBJECTS AND MILD ASTHMATICS USING COMPUTERISED AMBULATORY CAPNOGRAPHY.

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The range of arterial PCO2 (PaCO2) is generally quoted as 35-45 mmHg for normal subjects. This range has been determined from measurements recorded under controlled conditions at rest in the laboratory and should therefore only be used as a reference for these situations.

Most previous measurements of PaCO2 or equivalents involved either arterial puncture or use of uncomfortable apparatus such as mouthpiece or masks which may influence the breathing being measured. End-tidal PCO2 (PETCO2) record ed from expired air is regarded as equivalent to PaCO2 in resting subjects with normal lungs.

We measured PETCO2 by mass spectrometer sampling relatively uninvasively via a fine nasal catheter (1) and reported that the lower limit of the normal range in the laboratory should be 31 -32 mmHg (4.13 - 4.27 kPa).

We have consistently argued that the lower limit of the normal range should be revised downward for patients during activities of daily living outside the laboratory (3). This is of relevance in the study of patients with hyperventilation related disorders.

We previously showed that the outside range of PETCO2 within which symptoms of hypocapnia can occur in normal subjects was 14-29 mmHg (2). It could therefore be argued that if normal subjects during activities of daily living routinely reduced their PaCO2 below 29 mmHg for periods of time during the day, they may be prone to hypocapnic symptoms, which, if misinterpreted as more serious disease, could lead to a vicious spiral of increasing anxiety, hyperventilation and invalidism(3). It is conceivable therefore, that subjects who naturally keep their PaCO2 at the lower end of the normal range may be the subjects who are particularly vulnerable to hyperventilation disorders, but this need to be established.

Hyperventilation is well recognized as a concomitant of acute asthma, and studies in which PaCO2 was related to the severity of acute asthma showed clear evidence of significant hyperventilation with only modest reductions of FEV1(4). We have also argued that a major aetiological factor in symptomatic hyperventilation is very mild and often previously undiagnosed asthma, which can lead to a vicious cycle of panic and disability(3, 5, 6, 7).

For example, we described a patient who presented with severe hyperventilation and tetany who was subsequently found to have asthma as the sole underlying aetiological factor for hyperventilation (6).

In another study(7), we investigated a group of pat ients presenting to our accident and emergency department with a primary diagnosis of hyperventilation. Approximately 80% had good evidence of underlying asthma, which had been previously unrecognized in a half. The association between symptomatic hyperventilation and asthma has been emphasized by others. It would therefore be reasonable to propose that mild chronic asthma may be associated with even more marked hyperventilation and hypocapnia than has been described in severe acute asthma.

However, we recently showed that mild asthma was associated with only a small (but significant) reduction of both arterial and end-tidal PCO2 which correlated with airway hyperreactivity and not with measures of lung function or airway inflammation(8), but this study was not able to confirm that mild asthma is invariably associated with more marked and clinically significant hyperventilation.

In the present study we wished to determine whether some normal subjects demonstrated levels of PETCO2 below the accepted normal lower limit during activities of daily living, and whether more dramatic hypocapnia may occur under some circumstances in mild asthmatics under these conditions.

We used our new ambulatory capnograph sampling from a fine nostril catheter with data collected by either on-line by telemetry or off-line by a data logger to study PETCO2 in 23 normal subjects and 11 mild asthmatics over 4 hours during activities of daily living within the workplace and at home (n=8).

For the normal subjects, the mean PETCO2 was 37.3 (SD 2.8) mmHg and the lowest limit of the range (mean minus 2SD) was between 30 and 35 mmHg in 15 subjects and below 30mmHg in 4 (1 male, 3 females). There was no correlation between PETCO2 and lung function, psychological scores, gender or between work place and home. The responses of the asthmatics were not significantly different from those of the normal subjects.

This study therefore confirmed that the majority of both normal subject s and mild asthmatics when measured uninvasively during activities of daily living had end-tidal PCO2 values well below the accepted lower limit of 35mmHg and within the range at which symptoms of hypocapnia could occur.

The subjects with the more extreme reductions of PETCO2 may well be more vulnerable to developing clinically significant hyperventilation, but this requires further study. We were not able to confirm that mild asthmatics have more dramatic hyperventilation during activities of d aily living that normal subjects. The hyperventilation in our previously studied patients with mild asthma may have had an additional or alternate etiology.

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A Tool for Analysis of Dysfunctional Breathing and its Correction in Patient Populations

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Long-termed stress might repeatedly trigger hyperventilation (HV)(von Schéele & von Schéele, 1999) to become automatic, which in turn might lead to a varity of different disorders, e.g. cerebral vascular constriction reducing available oxygen and thereby hypoxia and depletion of buffering systems with e.g. "burn out"-effects on cellular levels.

In some of our patients we not only observe hypocapnea (low ETCO2, below 4,5%) and oxygen compensation (SaO2, 99-100%) but also, as a result of log-termed depletion of alkalical systems hypercapnea.

Data from our clinics (von Schéele, et al in press) indicate that for adequate stress profile an alysis and proper diagnosis in patients suffering from chronic stress related disorders analysis of respiratory and metabolic parameters are of crucial importance as well as a platform biobehavioral treatment.

Method: To combine effective diagnostic and treatment where the patients can do most of the "work", we have developed a system, www.air-pas.com (Artificial intelligence based, Interactive Respiratory Psychophysiological Analysis System), which measure (a) Exhalation carbon dioxide, (b) Oxygen sa turation, (c) Heart rate and (d) Blood volume pulse.

The AIR-PAS contents different diagnostic and treatment protocols and generate diagnosis reports and interactive suggestions during biofeedback training directly followed by a report.

Results: Among 37 patients with stress related problems during treatment initially 6 had normal ETCO2, 21 low, 10 high (above 5,5%) and 6 so-called masked acidosis. Patient with severe organic cardiac diseases showed a similar dysfunctional pattern as those with functional.

Treatment results from an increasing number of patients are promising and controlled studies will come.

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Emotion, Stress, and Airway Resistance in Asthma

Thomas Ritz, Ph.D., Bernhard Dahme, Ph.D.

********** ABSTRACT PENDING **********

Emotion, stress and airway resistance:
How much more do we know now ?

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Clinical reports have listed a variety of affective states as potential precurso rs of asthmatic symptoms. Alexander (1950) suspected that states such as anger, sadness, fear of separation, jealousy or sexual arousal would provoke asthma attacks. Prior experimental or empirical studies mainly lacked control groups, so it was hard to judge whether emotion induced airway obstruction is specific for asthmatics.

A number of recent experimental psychophysiological studies tried to gain more insight into the mechanism of bronchoconstriction due to provoked emotions or induced stre ss situations.

The results were as follows:

(1) Uniform increases in oscillarory resistance (Ros) were observed in all emotional states in asthmatics and healthy controls.

(2) Negative emotions, mainly sadness, produced higher and more consistent Ros increases than positive emotions.

(3) In a systematic laboratory field comparison the laboratory results could be replicated in the field with Ros values due to induced sadness in the laboratory being the best predictor of FEV1 de creases during the experience of negative mood states in the field.

(4) Even mild mental and emotional stress are associated with reliable Ros increases, sometimes more pronounced in asthmatics than in healthy controls.

(5) Bronchoconstriction during mental and emotional stress seems to be associated with a lack of vagal withdrawal observed only in asthmatics but not in healthy controls.

(6) Even if the emotion and stress induced Ros increases are associated with considerable effect sizes (ranging about from 1.00 to 1.40), the underlying airway obstruction is only near to the threshold of just noticable differences, and therefore of little clinical significance. Further research would profit from specific dose-effect studies of emotion and stress induced bronchoconstriction in asthmatics and healthy controls.

Defensiveness as a risk factor for stress-induced bronchoconstriction in asthma

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Objective: Previous literature has shown that the psychological trait of defensiveness is related to elevated sympathetic reactivity to stress and to several cardiac risk factors. The aim of this study was to examine if asthmatic patients with this trait show elevated laboratory stress-induced asthma exacerbation.

Methods: Defensiveness was measured by the Marlowe-Crowne Social Desirability Scale using a quartile split with defensive asthmatic patients defined as the upper 25%. Sixty-six non-defensive and 22 defensive participants with asthma were exposed to laboratory tasks (initial baseline rest period, reaction time, and a shop accident film), as described in a previous paper.

Results: Prior to the initial baseline period, no between-subjects differences were present on pulmonary function measures. After exposure to the tasks, defensive patients with asthma showed a decline on spirometry test measures, in comparison with nondefensive asthmatic patients who displayed an increase. During the tasks, there was evidence of greater respiratory sinus arrhythmia amplitudes and lower skin conductance levels among defensive patients with asthma.

Conclusions: These data suggest that defensive patients with asthma may be at greater risk for stress-induced bronchoconstriction and they may show a stereotypical difference in stress reactivity from defensive people without asthma: stress-induced elevations in parasympathetic versus sympathetic activity.

Emotionally induced asthma: Fact or fiction?

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Although a certain percentage of asthmatic patients report various emotional experiences as precipitants of asthmatic symptoms, these reports seem to have little predictive validity for psychologically induced airway responses in labo ratory studies.

We suspected that two factors contribute to this poor outcome, (1) problems in the measurement of perceived trigger, and (2) the choice of laboratory tasks with little relevance to emotional triggers in daily life.

The proness to psychological triggers has previously been assessed by clinical interviews or rating scales of unknown reliability and validity. In addition, prior studies have preferred stress-induction or bronchoconstrictive suggestions as experimental stimuli.

We therefore developed an Asthma Trigger Inventory (ATI) for a psychometrically sound measurement of perceived triggers of asthma. Scores of the emotional trigger subscale were correlated with airway responses to emotional film clips in the laboratory. Patients completed a pilot version of the inventory before the laboratory session. They then viewed a number of short movie sequences pre-evaluated for eliciting distinct positive, negative, and neutral emotional states, and completed two classical stress tasks, mental arrithmetic and medical slides viewing.

Airway response was measured by oscillatory resistance throughout film clips and tasks. The emotional trigger score of the ATI was positively correlated to resistance increases during all films, in particular positive films.

In contrast, a single item rating scale for psychological trigger factors from a questionnaire on clinical history of asthma showed no significant associations with resistance increases during films. No relat ionship between the emotional trigger score and resistance increases during stress tasks was found.

We conclude that patients' reports of emotional asthma triggers, if measured adequately, can predict actual responding of the airways to emotional stimulation. The relevance of trigger reports to symptom aggravations or asthma attacks remains to be shown.

Cognitive and Emotional Factors in Relation to Symptom Perception in Childhood Asthma

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Symptom perception is increasingly recognized as a key link in the chain of variables connecting airway compromise and functional morbidity. This study examined the relationship between symptom perception and psychological variables in children with asthma.

Subjects were 64 children (58% male) with a mean age of 11.6 years (range:7-17 years) who had mild-intermittent (7.8%) to severe (14.1%) asthma.

Symptom perception was assessed with the AM2 programmable, hand-held spirometer, used at home at least daily for one month (mean assessments per child = 53). Subjective quantification of symptoms prior to each spirometric assessment (via a visual analog scale and a guess of peak expiratory flow rate) was entered and locked in by subjects immediately before each "blow".

Corresponding subjective and objective values for each assessment were plotted o n the vertical and horizontal axes of a grid. Perceptual accuracy was defined in 2 ways: 1) the percentage of values in the "accurate" zone," i.e. subjective estimate within +/-10% of objective value and 2) the percentage of values in the "danger zone," i.e. subjective estimate > 10% higher than objective value, and objective compromise >20% from personal best spirometry.

An intelligence estimate was derived from selected scaled scores (Vocabulary and Block Design) of the WISC - III. Visual attention was measured by the Conners' continuous performance task (CPT), and auditory attention was measured by the Arithmetic and Digit Span subtests on the WISC-III.

Parents completed the Connors Parent Rating Scale (CPRS), which provides parent report of attention, impulsivity, etc. Children completed the Children's Depression Inventory (CDI), and the Multidimensional Anxiety Scale for Children (MASC).

The AM2 device worked well to collect and store longit udinal data from children as young as 7. For the whole group, 12.3% of the subjective/objective points fell in the danger zone and 53.4% in the accurate zone. Perceptual accuracy was associated with older age ($r = .26$, $p < .05$), higher IQ estimate ($r = .43$, $p < .001$), better auditory attention ($r = .36$, $p < .01$), and lower parent rating of inattention ($r = -.33$, $p < .01$) and oppositional behavior ($r = -.30$, $p < .05$). Dangerously poor perception was associated with poorer visual attentio n as measured by the CPT ($r = -.31$, $p < .05$). Depressive and anxiety symptoms were not related to perceptual accuracy.

These data support the feasibility of measuring real-world respiratory perception in asthmatic children in a clinically meaningful way. Intelligence and attentional variables were predictive of perceptual ability. Children with attentional difficulties (e.g., ADHD diagnosis), may be at risk for poor perception of asthma symptoms.

ASTHMA SELF MANAGEMENT: AN EMERGING OPPORTUNITY

Thomas Creer, Ph.D.

********** ABSTRACT PENDING **********

RESPONSE TO CO2 CHALLENGE IN POST-TREATMENT, RECENTLY ABSTINENT, ALCOHOL DEPENDENT INDIVIDUALS.

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Our goal was to determine whether alcohol dependent individuals are hypersensitive to a CO2 challenge.

This question addresses a putative mechanism linking anxiety disorders to alcohol use disorder, two conditions that frequently co-occur. Pathological alcohol use may perturb one or more systems in the CNS that increase one's risk for the development of anxiety disorder.

We have speculated that one such process is the effect of alcohol dependence and withdrawal on the control of breathing, especially as mediated by the central CO2 chemoreceptors. This view is based upon two separate observations.

First, dysregulated respiratory responses (e.g. hyperventilation, hypersensitive suffocation response) are common among individuals with anxiet y disorders and have been linked directly to the etiology of anxiety disorder.

Secondly, acute effects of alcohol include the suppression of the normal ventilatory response to CO2, raising the possibility that compensatory upregulation via tolerance would manifest as ventilatory hypersensitivity upon withdrawal.

If this were true, it might help to explain the high rate of anxiety disorder among alcohol dependent individuals via the anxiogenic consequences of respiratory dy sregulation associated with alcohol withdrawal.

To partially test this view, we applied two well known methods to quantify CO2 sensitivity (Read's rebreathing challenge and breath-holding challenge) in:
1) 15 recently detoxified alcohol-dependent individuals (alcoholic group); and,
2) 32 never alcohol disordered social drinkers (control group).

Contrasts (ANOVA) showed that the alcoholic group exhibited significantly greater respiratory responses to CO2 c hallenge in both challenge methods (ps < .004 and .002). Hypersensitivity to CO2 shown by alcohol dependent subjects indicates a vulnerability to both hyperventilation and exaggerated suffocation responses which, as described above, have been implicated in the development of anxiety responses.

These findings offer support for the view that alcohol dependence and withdrawal may serve to promote anxiety problems among alcoholics when centrally mediated respiratory systems become dysregulated.

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Specific Inspiratory Muscle Training

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Research has been carried out since the late 1800's on the effect and importance of respiratory muscle strength. (1) Anecdotally it has been reported that in the First World War soldiers we re encouraged to exercise their muscles of respiration by breathing through cotton wool causing increased resistance with the aim to improve inspiratory muscle strength leading to improved cardiovascular performance.

Can this be possible?
Does Respiratory muscle fatigue exist ?
If so can respiration effect performance?
If it does, will strengthening of these muscles make a difference?
How do we strengthen them?

This presentation will answer the above questions.
I will :
1 Give a brief review of the literature on the topic respiratory muscle fatigue.
2 A brief review of the literature on inspiratory muscle training both in the fields of research and practice.
3 Present three case histories looking at the effectiveness of strengthening the respiratory muscles.
These will be presented to examine the effect and results of Specific Inspiratory Muscle Training (SIMT)

* The first case is a 42-year-old female who presents with life long asthma.
* The second a 56 year old female with chronic occupational overuse syndrome, (RSI)
* The third case is that of a 26-year-old ex-competitive triathlete presenting with fatigue at rest and extreme SOBOE. (shortness of breath on exertion)

All three subjects were followed over an eight-week period in which time they carried out SIMT.

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Symptomatic Chronic Breathing Disorder in a Post-Coronary Bypass Graft Patient

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This study examines the results achieved by physiotherapy, first at the time of the surgical procedure, and the subsequent long-term effects it had on this particular case 4 months later. It also examines the role physiotherapy had to play in the patient†s full recovery.

It is of particular interest given the current climate of quick turn-over of surgical patients, especially those having their surgery away from their own base hospital (if they still have one) who may miss out on discharge checks, or cardiac rehabilitation courses.

Key points: improvement in awareness and skills in recognition and differential diagnosis of breathing pattern disorders/hyperventilation syndromes, in both in-patient and out-patient contexts.

The Rescue Breathing Pattern:
Physiologic and Psychologic Consequences in COPD

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The Rescue Breathing Pattern (RBP) is a universal cognitive response to dyspnea distress. Simply stated, the RBP is to "pump air in and out of the lungs as fast as you can."

For patients with Restrictive Lung Disease the RBP is a proper physiologic response, and the breathing pattern is dictated by a reduced and inelastic pulmonary reservoir.

In Obstructive Lung Disease, and particularly with Emphysema and loss of lung elastic recoil, the RBP is an improper response as it will lead to Air Trapping and Dynamic Hyperinflation. Overinflation places the chest bellows mechanism in a position of mechanical disadvantage and will independently exacerbate the dyspnea sensation. These physiologic principals may be understood by a consideration of the Flow-Volume Loop and in particular the static Pressure-Volume Curve.

To prevent and correct overinflation the COPD patient must breathe slowly, with a larger Tidal Volume, a prolonged expiratory phase, and breathe gently. This corrective breathing pattern is 180 degrees out of phase with the RBP. The RBP is therefore a corrupting reflex in COPD.

The commonest complaint in COPD is dyspnea, usually after an event of exertion or uncontrolled coughing. Frightened patients commonly activate the RBP which will in turn further exacerbate their dyspnea. This leads to a feeling of loss of control, and will compound their psychologic problems. When COPD patients learn to control the RBP they usually regain a significant measure of psychologic control.

Conclusion: The RBP is a universal response to dyspnea, and in COPD is a corrupt response leading to further dyspnea and a sense of psychologic loss of control. Control of the RBP in COPD will improve dyspnea and help to restore confidence and loss of control.

EXPANDED ABSTRACT

THE RESCUE BREATHING PATTERN

DEFINITION OF THE PROBLEM

The Rescue Breathing Pattern ("RBP") is a cognitive universal response to acute dyspnea, or a dyspnea exacerbation of chronic dyspnea. It is a modulator of the usual regulatory feedback mechanisms of breathing control.

Simply stated, the RBP is to "...pump air in and out of the lungs as hard and as fast as possible..." in an attempt to relieve dyspnea distress.

The response is easily observed in normal subjects, both deconditioned and well conditioned athletes. It may also be seen in diseased patients, though the signs may be more subtle. It is seen in all varieties of lung disease, both airway *obstructive* and pulmonary *restrictive* diseases. It is also seen in all types of cardiac diseases, or any other miscellaneous conditions that produces dyspnea.

The only partial exception to this RBP rule is the general class of *Anxiety Psuedodyspnea* and/or *Hyperventilation Syndrome*. Note that these conditions are also cognitively driven, and freq uently productive of erratic or bizarre breathing patterns. However, I submit that even the most anxious Hyperventilation Syndrome patient, if obliged to run 500 meters as fast as possible, or if submerged in their swimming pool for two minutes and coming up for their first breath of air, will exhibit the RBP at least initially.

The RBP is therefore a cognitive **modulator response** that is superimposed on the many reflex feedback breathing control mechanisms, such as the oxygen and CO2 sensors, chest wall and lung pressure and proprioceptive receptors, and many other receptors and their feedback mechanisms. Without question, these numerous feedback mechanisms are dominant and controlling in the regulation of breathing. The Rescue Breathing Pattern is **not** a competing theory of breathing control, but rather an **adjunct modulator response** to basic controlling mechanisms.

The **Rescue Breathing Pattern** is characterized by:

- Increased Respiratory Rate
- Increased Tidal Volume (Note: This is Rate limited)
- Forced Breathing ((Both inspiration and expiration)
- Increased air flow
- A focus on inspiration
- Shortened expiration time

I submit that almost everyone is subconsciously aware of the RBP. This can be demonstrated by asking yourself, or most medically untrained people selected randomly, the following questions regarding the RBP:
"What do you do if you are short of breath?" followed by:
"And what else do you do?" (In order not to provide inappropriately leading questions).

Almost everybody will easily reply to these questions that they breathe faster, with larger breaths, and attempt to breathe forcefully. Only a few will recognize increased air flow (though this is inherent in the reflex). Many will recognize their efforts are mainly directed to breathing in, though this may require some prompting such as "Do you find that you are working harder to breathe in or out." Only a re lative few subjects will recognize a shortened expiratory phase, unless given quite leading prompting.

The author finds it very curious that this easily observable Rescue Breathing Pattern appears to be unrecognized in the pulmonary and psychophysiology literature.

Note the Rescue Breathing Pattern when contrasted to the mechanical breathing **requirements** of patients with COPD, Emphysema, and moderately severe/severe Asthma:

NORMAL (Rescue Breathing Pattern)	**REQUIREMENTS** for COPD / Emphysema / severe Asthma
Rapid Rate	Slow Rate

Large Tidal Volume (Rate limited)	Large Tidal Volume (Not Rate limited)
Forced Breathing	Gentle Breathing (N.B. Expiration)
Increased Air Flow	Decreased Air Flow
Focus on Inspiration	Focus on Exp iration
Short Expiratory Time	Long Expiratory Time

Note that the RBP is essentially 180 degrees out of phase with the physiologic needs of the patient with COPD and severe Asthma, and particularly so in patients with Emphysema.
The Rescue Breathing Pattern is therefore a corrupt reflex in these COPD patients.

It is important to note however, the RBP is a physiologically appropriate response for patients with **Restrictive Lung Disease**.

Restrictive disease by definition is one that has limited the lung volume. The classic example is Pulmonary Fibrosis, and probably the most common example is cardiac disease with congestive heart failure. Here the limited pulmonary volume is being used to near capacity, i.e. working at the limit of the available elastic reservoir. This is why these patients typically have a rapid and shallow breathing pattern with a short expiratory phase, because this is the most mechanically efficient way to breathe under these constraints. Stretching this limited elastic reservoir further with large breaths will cause the Work of Breathing to increase markedly, and therefore cause further respiratory distress.

The RBP is in synchrony with the mechanical needs of these patients, and therefore is appropriate and should not be altered by breathing re-training.

In treating patients with Restrictive Disease **do not attempt to alter the native rapid and shal low breathing pattern.** The slow and deep breathing patterns taught by many breathing advocates are contrary to physiologic requirements. Calming and relaxation techniques, and coping strategies are appropriate, but altering native breathing patterns is detrimental to these particular patients.

It is instructive to read the definition of "**GASP**," as in "Gasping for breath" in non-medical dictionaries. Most definitions of gasp are related to theatrical descriptions, as in "...she gasped when she saw her husband in the arms of another woman..." and so on. However, there are a number of more physiological descriptions of interest.

The Random House Dictionary describes:

- "To struggle for breath with mouth open."
- "The long-distance runner gasped for air: struggle with open mouth (for breath), inhale frantically, labor for breath; suck in (air), breathe convulsively; have trouble breathing, respire labor iously; gulp, pant, wheeze, puff, catch the breath."

Webster's Dictionary describes:

- "To open the mouth wide in catching the breath; or in laborious respiration; to labor for breath; to respire convulsively; to pant violently."

It seems clear from these various descriptors that the lay public has considerable insight into, and recognition of, the Rescue Breathing Pattern.

Stedman's Medical Dictionary does not define the word "gasp," which appears to be a curious omission. Could this perhaps reflect that patients have been smarter than their physicians in recognizing the Rescue Breathing Pattern?

It is now necessary to consider some basic pulmonary physiology and mechanics of breathing, related to the conditions of "air trapping" and functional lung "overinflation," and this in turn related to the question of so-called "breathing exercises."

The 1955 seminal and classic textbook of pulmonary physiology *THE LUNG* by Dr. Julius Comroe (THE LUNG: Clinical Physiology as Pulmonary Function Tests - Julius H. Comroe et al, 1st. Edition, 1955) makes reference on page 127 in the section on Mechanics of Breathing to the phenomenon of "air trapping" and "...overdistention of the lung..." which "...may be noted to an even greater degree in emphysema..." The authors then went on to state "...If patients with air trapping are taught to breath out slowly, then can often breathe out more completely. This is one of the rational objectives of breathing exercises..."

It seems clear that the concept of air trapping and its detrimental consequences, and particularly so in Emphysema, has been well established for a long time.

However, the subject of "Breathing Exercises" has been a controversial one, as articulated by the 1992 statement from the European Respiratory Society:
"...until more definitive evidence is available, breathing training cannot be recommended in COPD..."
(Pulmonary Rehabilitation in Chronic Obstructive Pulmonary Disease (COPD) with Recommendations for its use - Donner, CF, Howard, P; European Respiratory Journal, 5, 266-275, 1992)

This author would like to point out that there are many types of Breathing Exercises, as seen for example with:

- Chest mobilization techniques (several varieties).
- Respiratory muscle strengthening exercises A.) Strength training B.) Endurance train ing (several varieties of each).
- Diaphragm awareness and use (several varieties).
- Thoracic - Diaphragm coordination (several varieties).
- Hyperventilation Syndrome (several varieties).
- Cervical fracture - chest paralysis (several varieties).
- Yoga / Meditation / numerous non-medical techniques.

It seems clear that the term "Breathing Exercises" means many things to many people, and as generally used is an almost meaningless term. As such it is not surprising there is controversy surrounding this subject. Any rational discussion of Breathing Exercises requires a precise definition of what one means by Breathing Exercises, and precisely what one is doing in that domain of interest.

The authors definition of "Breathing Exercises" relates to the therapeutic Breathing Pattern associated with COPD / Emphysema / severe Asthma which is, breathing pattern training (in conjunction with chest physical therapy techniques) that promotes the minimal necessary level of Alveolar Ventilation in conjunction with minimal Work of Breathing.

While empirical knowledge strongly suggests this pattern is one of a slower respiratory Rate, a larger Tidal Volume, a prolonged expiratory phase, and non-forced respiratory effort, the precise definition of the optimal Breathing Pattern / Breathing Exercise has yet to be defined by advanced pulmonary mechanics studies. Indeed, the precise ventilatory pattern may be found to be somewhat variable between different patients (See: Hillsman 1995 ISARP presentation).

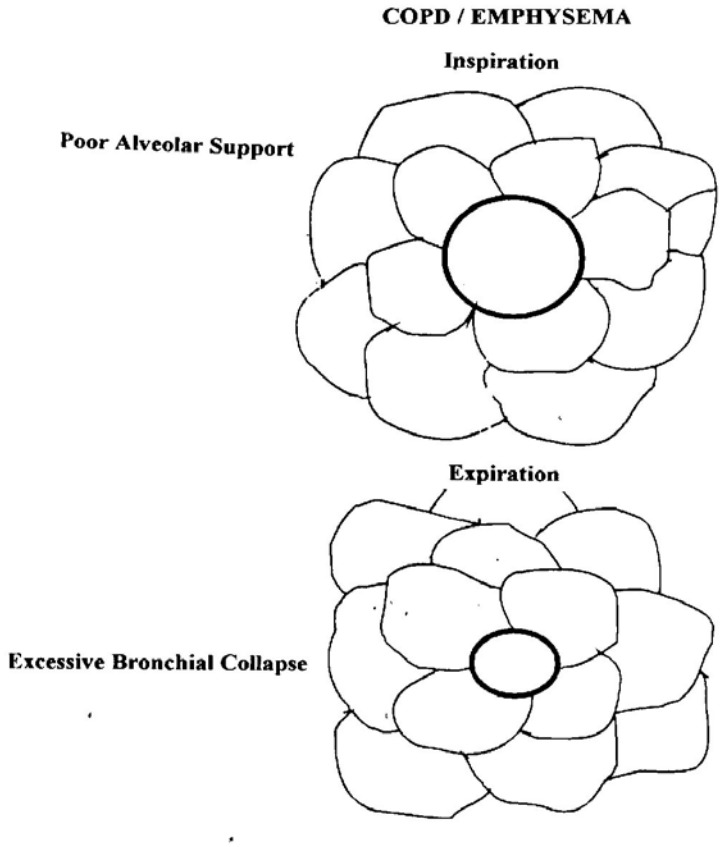
Undisputed is the need to prevent and/or correct Air Trapping and Functional Overinflation.

The optimal Breathing Pattern must be achieved within the constraints of appropriate Chest Physical Therapy techniques, a discussion of which is beyond this presentation. These Physical Therapy questions in turn relate to frequently distorted chest wall configurations and associated respiratory muscular dynamics, an emerging area of research. In addition, the author intuitively believes the prolonged expiratory phase is associated with critically important respiratory muscle rest, which remains an unanswered question dependant on yet to be developed methodology to measure respiratory muscular "rest."

PULMONARY PHYSIOLOGY and MECHANICS CONSIDERATIONS

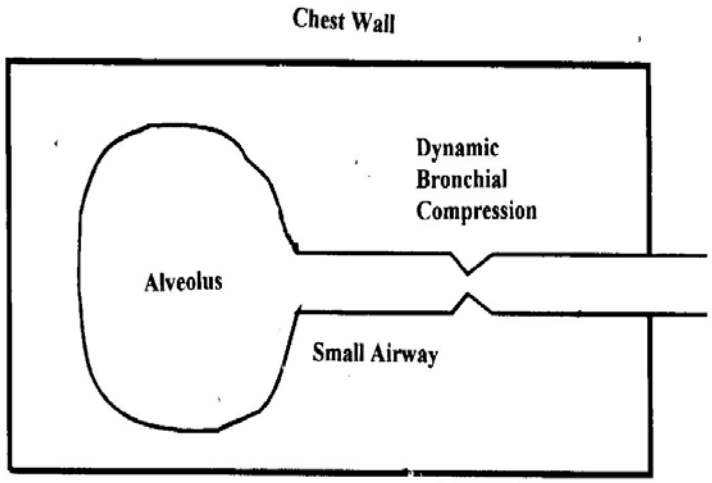
The normal bronchus is surrounded by and attached to a dense mesh of alveolar structures, which have elastic properties, largely due to embedded elastic fibers. These elastic structures constantly pull in all directions on the small airways, therefore helping to maintain open airways.

On expiration, with the chest decreasing in volume, all structures in the chest decrease in size. Alveolar volume decreases and air is normally evacuated, but simultaneously the airways also decrease in size. However, because of the normal elastic recoil properties of the surrounding lung, the relative degree of bronchial collapse is minimized.



In contrast the Emphysema patient has lost alveolar structure and elastic recoil properties. On inspiration there may be difficulties due to irregularities and distortion within the small airways, but the major problem is on expiration. Here the lack of elastic recoil results in excessive collapse of the smaller airways. Therefore the **Time Constant** for alveolar emptying must be prolonged.

The result is a basic need for a prolonged expiratory phase in COPD, and particularly so with Emphysema. Failure to achieve a longer Time Constant will result in **Air Trapping** and **Pulmonary Overinflation**.

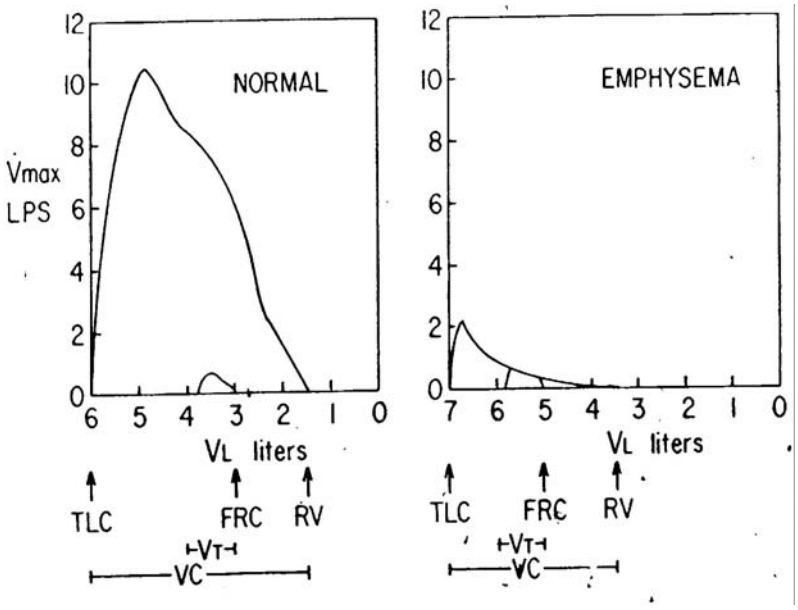


This diagram is a simplistic reduction of the lung to one alveolus and its smaller airways within the surrounding chest wall.

On expiration the chest wall volume decreases, therefore applying relative pressure on the Alveolus, and thus promoting air flow through the airways.

However, at the same time pressure is a lso being applied to the smaller airways, and at some time a **Critical Closing Pressure** will be achieved, and at some point in the system the smaller airways will collapse and expiratory air flow will be further impaired.

Forced expiratory efforts will exaggerate the problem of Dynamic Bronchial Compression and further impair expiration. The result is a basic need for non-forced, gentle expiratory efforts.



The mechanical consequences may be appreciated by examining the familiar **Flow Volume Loop**.

Here a normal resting ventilation pattern is superimposed on the forced expiratory Flow-Volume Loop.

Note the normal subject has a relatively low FRC and therefore the ability to take in a much larger breath. Also note the normal subject has a much larger reserve of forced expiration as there is no airway obstruction.

In contrast, the severe Emphysema patient has a diminished capability to take a deep breath as the FRC is elevated, and likewise a much

diminished capability of forced breathing due to airway obstructive disease. Therefore, the breathing reserve of the Emphysema patient is much diminished, and even with resting breathing these patients may be working near their available maximum reserve capabilities.

The mechanical consequences may be better appreciated by examining the **Static Pressure-Volume Curve**.

This curve is obtained by placing an esophageal balloon to measure the pleural pressure, and then having the subject inhale and exhale to several different lung volumes, with the glottis held open in a relaxed position. This measures the Transpulmonary Pressure, and is a reflection of the elastic recoil within the lung at different volumes.

Note in the normal subject because of the low FRC there is a large volume reserve to Total Lung Capacity. Resting breathing is accomplished with only a slight degree of effort, i.e. with only s slight degree of inspiratory pressure needed, and even a moderately large breath is achieved with only slight increased effort.

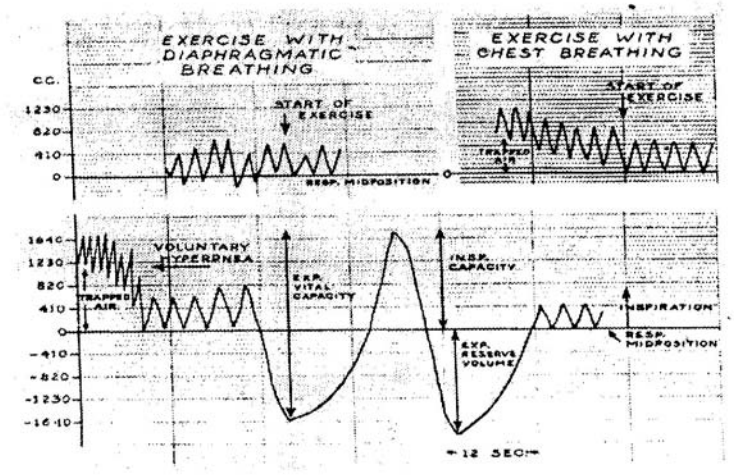
In contrast the Emphysema patient (for convenience shown on the same P-V Curve; the actual Emphysema P-V Curve is up and to the left and therefore with even more mechanical constraints) is working under great mechanical disadvantage. Even with resting breathing these patients are near their available Total Lung Capacity, and particularly if they are at or near the curvilinear position of the P-V Curve all breathing must require a much larger degree of effort.

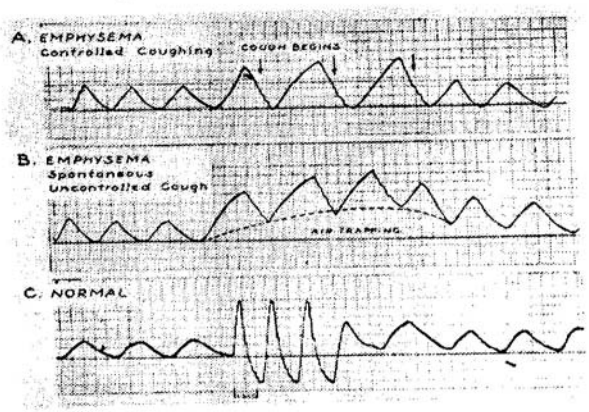
This is because the chest wall and the respiratory muscles are in positions of mechanical disadvantage and operating inefficiently. Any attempt to take a deeper breath can only be as a result of excessive mechanical effort.

Note that with Functional Overinflation the patient will shift up the Pressure-Volume Curve and their mechanical breathing problems will therefore be greatly compounded.

The clinical problems of overinflation have been known for a

PHYSICAL THERAPEUTIC MEASURES in the TREATMENT of CHRONIC BRONCHOPULMONARY DISORDERS:
Methods for Breathing Training
William F. Miller, M.D.
Am. J. Medicine - Vol. 24, January/June, 1958, p.929





long time, as elegantly shown with simple methodology in a 1958 paper by Dr. William Miller.

Air Trapping and Functional Overinflation is shown with exercise, and prevented by appropriate "Diaphragmatic Breathing" techniques.

Note particularly in the example of voluntary hyperventilation within only seven breaths a patient Air Trapping 1230 cc of air. Indeed, a striking example of how quickly significant overinflation, and therefore rapid decompensation, can develop.

Also shown is an example of Air Trapping with uncontrolled coughing, a very common problem causing respiratory distress in COPD.

More recent sophisticated studies by Michael Belman and colleagues (**Inhaled Bronchodilators Reduce Dynamic Hyperinflation during Exercise in Patients with Chronic Obstructive Pulmonary Disease** - Belman MJ, Botnick WC, Shin JW; American Journal of Respiratory and Critical Care Medicine 1996;153:967-75) as well as Denis O'Donnell and colleagues (**Breathlessness during acute bronchoconstriction in asthma** - Lougheed MD, Lam M, Webb KA, O'Donnell DE; American Review of Respiratory Diseases 1993; 148: 1452-1459 and **Exertional breathlessness in patients with chronic airflow limitation** - O'Donnell DE, Webb KA; American Review of Respiratory Diseases 1993 148:1351-1357) have given insight into the critical importance of dynamic Functional Overinflation in COPD / Emphysema / Asthma.

There can be no reasonable dispute as to the importance of Air Trapping and dynamic Functional Overinflation in the production of dyspnea in COPD / Emphysema / Asthma.

PSYCHOLOGICAL CONSEQUENCES OF DYSPNEA

CHRONIC DYSPNEA

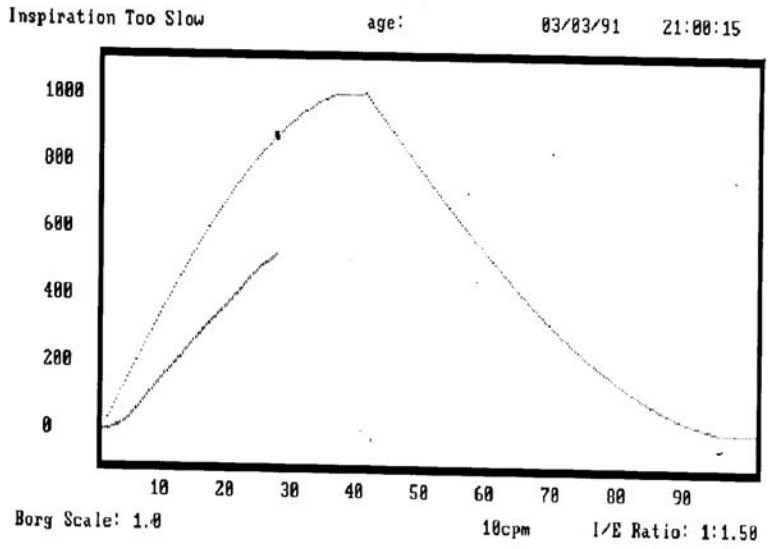
It is well established that chronic dyspnea may produce anxiety, depression, and a number of stress related psychological problems.

ACUTE DYSPNEA

Recurr ing acute dyspnea attacks are painful experiences, and it is understandable that patients frequently live in fear of these attacks. Fear of dyspnea attacks, and of the unknown, leads to a **Loss of Control**. The loss of control involves loss of the means to control the attack itself. Perhaps even more profound is the loss of control of "self," and the very basic qualities of what comprises the human experience.

The ability to control acute dyspnea attacks restores patient confidence over the most distressing component of the COPD disease process, which in turn can restore the loss of control of "self." Restoring "self" can have a profoundly positive effect on the patient's general outlook and well being.

A SOLUTION TO THE OVERINFLATION PROBLEM THROUGH BREATHING PATTERN TRAINING

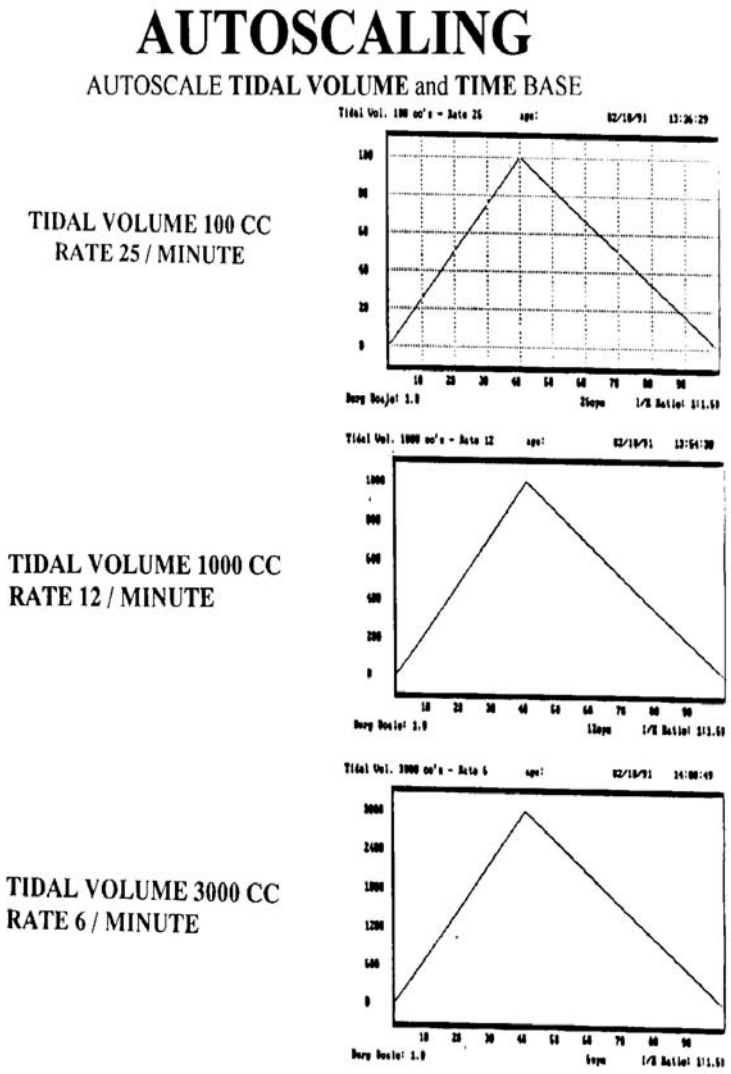


A visual biofeedback training system was presented at the ISARP meeting in Toronto in 1995 and therefore this will not be elaborated on at this presentation (See: Hillsman 1995 ISARP).

Briefly, by menu selection an infinite variety of inspiration and expiration breathing patterns may be displayed on a computer CRT. A cursor blinks along the programmed line, and the patient attempts to match their real time performance with the programmed line. Performance deficiencies, such as the slow inspiration rate shown in the diagram, are immediately apparent and the patient is therefore given a visual biofeedback signal to correct their training performance.

A version of the program done in the LabVIEW environment is available at http://www.sierrabiotech.com

It is emphasized that while the analog displays may appear simple, the information content of visual analogs is high. This display method is a paradigm shift in respiratory diagnostic and therapeutic technology, in that the clinician can literally **watch** the patient breathe.



One significant point about the visual training system realized since the 1995 ISARP presentation deserves particular mention, and that is the importance of the **AUTOSCALING** display.

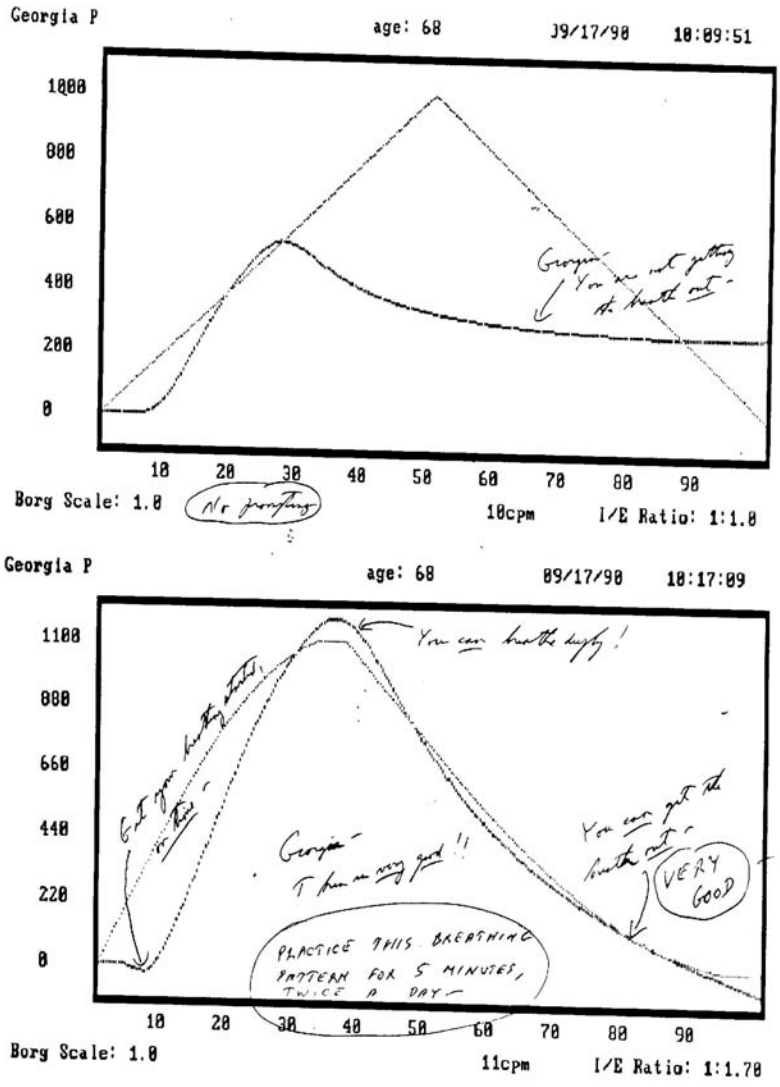
Note in the diagram the three displays are identical, due to autoscaling, but in fa ct while the breathing patterns are identical, they represent three very different breathing performances varying from 100 to 3000 cc Tidal Volume and 25 to 6 breaths per minute respiratory Rate.

In order to perform correctly, the autoscaling display forces the patient to focus on internal sensing of the biofeedback experience.

It is theorized this is why the autoscaled display appears to be a particularly effective respiratory biofeedback training tool.

It is speculated that autoscaling displays may be useful in other modalities of biofeedback training, such as Hyperventilation Syndrome.

THE BREATHING TRAINING METHOD



The diagram is an actual patient record from the original visual biofeedback device in an individual with COPD.

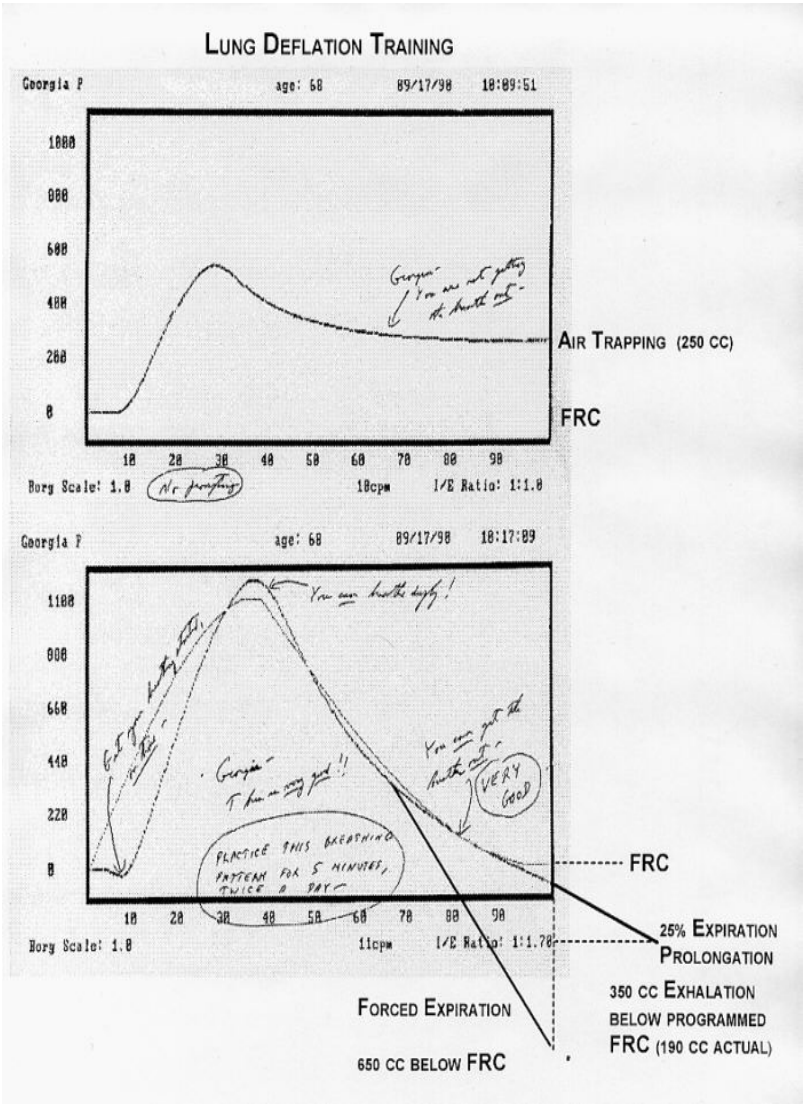
On the top screen the patient display is blanked, to record the native breathing pattern in a standard manner (Tidal Volume 1000 cc, Rate 10 bpm).Note the patient Tidal Volume of about 500 cc, and a long expiratory phase, but despite this expiration does not return to the FRC baseline. This is Air Trapping, and in this example about 250 cc.

The lower screen is a prompted display. This is the patient's first training session, eight minutes later. Note the patient breathing pattern almost perfectly matches the desired program, with only a slight delay in starting inspiration. Annotations are made e.g. "You can breathe deeply" (the patient now achieving a Tidal Volume of 1150 cc) and "You can get the breath out" (the patient now exhaling down to resting FRC). Note the home instructions to the patient.

Patients are given a copy of their breathing record, and instructed to practice breathing twice daily, and for no more than five minutes (to avoid fatigue and boredom). They were told to sit in a comfortable chair, relax, and to concentrate on their breathing program and to "Put the picture in your mind." Depending on the situation various Physical Therapy techniques may be employed. Note carefully, this breathing pattern training will **prevent** Air Trapping. The usual patient demonstrates substantial retention of the breathing pattern prescription within two to three months. Note this training method is an example of **Positive Reinforcement Biofeedback** (i.e. the subject feels better with the biofeedback experience, and therefore the biofeedback is reinforced) which is inherently a more powerful biofeedback technique.

Typically a new patient is seen about three times in the first three months during regular office follow-up, with training sessions lasting about 10 to 12 minutes. While the learning rate is perhaps slower than other methods, the burden of training is almost entirely on the patient. As such, this method is exceptionally cost effective.

LUNG DEFLATION TRAINING



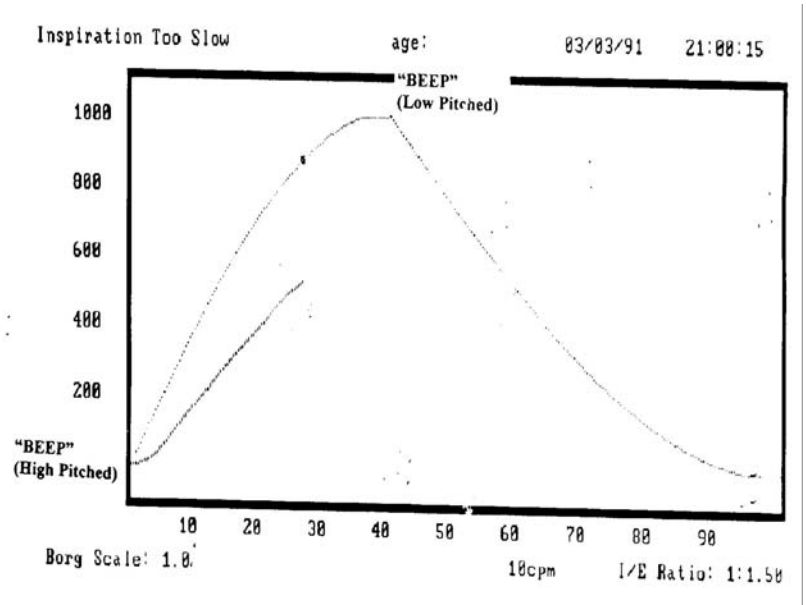
During regular training sessions the patient is shown on the visual display how to deflate their lungs, should they have an acute Air Trapping event.

Note carefully, these are Overinflation **corrective** breathing techniques, in contrast to the regular breathing pattern training which is a **preventive** Air Trapping technique.

The first technique is a prolongation of their regular expiratory breath, for a time approximately 25% longer that their programmed expiration time. In this example this would result in an approximate 350 cc of deflation below FRC.

The second technique is a gentle forced exhalation beginning approximately 1/2 to 2/3 through expiration, and lasting approximately the usual expiratory time. This technique is more effective for the patient in acute distress with severe Overinflation. Note that the expiration effort must be controlled and gentle, to avoid compounding the problem of dynamic bronchial compression and premature small airway collapse. Done properly, patients should feel substantial (but not complete) dyspnea relief within 5 to 10 breaths. At this juncture they can again switch to their regular preventive breathing pattern and completely releive their dyspnea within usually a few minutes.

This represents a **return of control** of acute dyspnea events, and with functional control comes the confidence that the psychological "loss of control" problems can also be resolved.



BREATHING PATTERN ACTIVATION

Several years ago optional sound

prompting was added to the visual biofeedback trainer display, as an aid for the visually impaired.

This consisted of a "beep" (high pitched) at the beginning of inspiration, and another "beep" (low pitched) at the beginning of expiration.

It was discovered that if patients trained with both the visual and audio prompts, and if the visual prompt was turned off, the audio prompt would cause the patient to reproduce the appropriate visual breathing pattern.

It is theorized that the visual biofeedback training teaches the patient complex breathing patterns, and the simple audio prompt triggers these complex patterns.

A simple portable prompting device based on these principals is anticipated in the near future. This has implications for not only COPD / Emphysema patients, but for other clinical purposes as well, e.g. Hyperventilation Syndrome.

RESEARCH AND CLINICAL QUESTIONS

ISARP literature has posed the following research priority questions regarding Breathing Patterns:

1.) Can Breathing Patterns be learned?
The answer is **YES**, at least in an elderly COPD population, and with the anticipation that a younger Hyperventilation Syndrome population would also be trainable. Furthermore, it is surprisingly easy to tea ch the elderly new Breathing Patterns.

2.) Can Breathing Patterns be retained?
The answer is **YES**, at least in an elderly COPD population, but with retention lost to a variable degree over time. Generally two or three re-training sessions a year restores the learned pattern, though the occasional patient will retain their learned patterns for a year or more. Furthermore, spouses often report that after approximately three or four months the patient appears to be breathing in the lear ned manner while sleeping. If this observation is confirmed by sophisticated sleep studies it would strongly suggest that indeed learned Breathing Patterns are being retained. And this would also suggest that perhaps learned Breathing Patterns may be chronically and subconsciously influencing the general regulatory control mechanisms of breathing.

3.) Can Breathing Patterns be used clinically, outside the laboratory?
The answer is **YES**, at least in an elderly COPD population. The present proof of this is the time honored "*clinical trial*" or "*therapeutic trial*" as indeed the learned breathing patterns and techniques directed specifically at preventing and/or correcting pulmonary Air Trapping and Overinflation are clinical trials, and patients report success. Sophisticated ambulatory monitoring studies to confirm these patient reports would be highly desirable.

CONCLUSION: The Rescue Breathing Pattern is a common and important factor, and a corrupting cognitive response in COPD / Emphysema / and severe Asthma which produces exaggerated acute dyspnea events.

Dyspnea exacerbations are the commonest patient complaint in symptomatic COPD. The resultant adverse physiologic consequences of Air Trapping and Functional Overinflation may be prevented and corrected by appropriate Breathing Pattern training methods.

A system to train patients in appropriate Breathing Patterns to prevent and correct Functional Overinflation has been developed.

The Respiratory P300:
in support of the triarchic model of amplitude.

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Department of Psychiatry and Behavioral Sciences, Stanford University and
Department of Psychology, The University of Melbourne.

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LaTrobe University Musculo-skeletal Research Center.

Johnson has proposed a triarchic model of P300 amplitude that identified three underlying constructs: information transmission, stimulus probability and stimulus meaning. These constructs are thought to map onto underlying neural generators that are responsible for the different aspects of processing. The output of these generators then sum to produce the stereotypical P300 response with its characteristic scalp topography. A late positivity can be readily observed in the averaged evoked response to inspiratory loading or occlusion stimuli. Dat a will be presented from four studies, which test different aspects of Johnson's model to see if it also applies to the respiratory P300.

The data indicate that the respiratory P300 is affected by information transmission, as it is larger to unequivocal stimuli and shows a significant enhancement with attention. It is affected by stimulus probability, with a highly significant relationship between amplitude and the number of un-occluded breaths preceding the stimulus. Finally, in comparison with auditory evoked potentials from the same subjects, it shows a very large effect of stimulus meaning, with baseline amplitude values being very high to even high probability stimuli.

Data will also be presented showing the effects of early sleep onset on the P300. The results of all studies indicate that the topographic distribution of the respiratory P300 maps nicely onto that seen from other stimulus modalities. Respiratory and auditory P300 amplitudes are diminis hed in asthmatics.

The present study investigated the respiratory-related evoked potential (RREP) in 16 asthmatics and 16 matched controls. The goal was to evaluate the relationship between respiratory sensitivity and the P3 component.

It was hypothesised that asthmatics would be less sensitive to respiratory stimuli and have reduced P3 amplitudes.

Additionally, auditory evoked potentials (AEPs) were recorded from an oddball paradigm in both groups and compared with th e RREP results.

The perceptual sensitivity of subjects was assessed by means of a load magnitude estimation task. Four magnitudes of resistive loads and no-load were applied for a single breath 10 times each in random order.

Respiratory sensitivity was estimated using a modified Borg scale.

Results showed little difference in perceptual sensitivity between the groups and in both groups resistive load was linearly related to magnitude estimation (asthma, $R2=0.96$; control, $R2=0.99$).

For RREP assessment, inspiratory occlusions were presented for 500ms.

For AEP assessment, 60dB 1000 and 2000Hz pure tones were presented for 100ms, with the 2000Hz tone presented with a 20% probability (target).

Scalp EEG was measured from 29 channels.

Results showed that both the respiratory and auditory P3 components were markedly reduced in the asthmatic group compared to the control group (reduction of 30%). All other components were similar between the groups. These results suggest an intrinsic reduction in P3 amplitude for asthmatic patients. This may relate to differences in processing perceptual information. Importantly, whatever the mechanism of the P3 deficit, it is not specific to occlusion stimuli.

A Respiratory Biofeedback Solution for the Rumination Syndrome

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Weill Medical College of Cornell University, New York, NY, USA

Recurrent regurgitation, during and/or after meals is the major symptom of a perplexing condition known as rumination, but often diagnosed in such terms as gastroesophageal reflux disease, gastrointestinal motil ity disorders, psychogenic vomiting, etc. Unliike vomiting, rumination feels effortless and is not associated with with an organic etiology. Against the sufferer's volition, food is brought from the stomach into the mouth where it is spit out or re-swallowed.

In successfully treating 7 or 8 medically-screened ruminators, ages 16-72, with duration of symptoms from 1 to 30 years, we noted a consistent breathing pattern in these patients, of which they were unaware. They all tightened their abdominal m uscles while inhaling, as if they were in a constant fight-or-flight reaction.

While a few earlier studies noted that ruminators constract their stomach muscles while eating, we were the first to report that such contraction, along with a strong ribcage movement upwards, was part of a thoracic breathing pattern. By modifying the patient's respiratory style to a slower, effortless, more diaphragmatic mode of breathing , abdominal muscles were more relaxed.

Patients were taught ways of incorporati ng this breathing approach into their eating and swallowing behavior. In addition, they were taught a muscle relaxation exercise to practice before, during and after eating and drinking.

This practice led to complete or almost complete symptom cessation in 7 out of 8 consecutively referred patients. (The 8th person did not comply with the protocol.)

ERP to Airflow Obstruction: Age's PFTS and OADS

Andrew Harver, Ph.D.

********** ABSTRACT PENDING **********