CapnoBreath Training

Peter M. Litchfield, Ph.D. in *California Biofeedback*. Vol. 21, No. 3 (Fall 2005)

Good respiration requires neither relaxation nor a specific mechanical prescription, save one:
"The varied melodies of breathing mechanics must ultimately play the music of balanced chemistry."

Although nearly everyone agrees that good respiration is basic to healthy physiology and psychology, only a very few people who do breathing training know much about respiration and how its chemistry regulates fundamental physiology critical to good health and optimal performance. Deregulated respiratory chemistry is commonplace, and may have profound immediate and long-term effects that trigger, exacerbate, and/or cause a wide variety of serious emotional, perceptual, cognitive, attention, behavioral, and physical deficits in health and performance. Breathing evaluation and training, without regard to chemistry, leave out perhaps the most fundamental, practical, and profound factors that account for (1) the far-reaching effects of deregulated breathing, as well as for (2) the surprising benefits of proper breathing re-education.

Breathing is a behavior, and as a behavior it meets multiple objectives, including, among others:

respiration
acid-base balance
prophylactic intervention
communication
relaxation
performance enhancement
psychological access
flight-fight preparation
consciousness exploration
meditation.

The fundamental of these, however, are acid-base balance and respiration (also itself, regulated by shifts in acid-base balance). CapnoBreath Training is about serving these two inextricably associated objectives, while setting the stage for the others.

THE HENDERSON AND HENDERSON-HASSELBACH EQUATIONS

Breathing chemistry is about carbon dioxide (CO_2) regulation. *Carbon dioxide plays a critical role in acid-base physiology, where contained therein are the principles of oxygen transport and distribution.* The Henderson equation, known to virtually everyone who has studied acid-base, renal, and pulmonary physiology, tells us that hydrogen ion concentration, $[H^+]$, in extracellular fluids (plasma, cerebrospinal, lymph, and interstitial) is regulated by the relationship between partial pressure arterial carbon dioxide, $PaCO_2$, regulated by breath, and bicarbonate concentration, $[HCO_3]$, regulated by the kidneys: $[H^+] = K \times PaCO_2 \div [HCO_3]$, where K is the dissociative constant of carbonic acid (H_2CO_3). In other words, acid-base physiology is regulated by changes in the relative contributions of pulmonary and renal physiologies: $[H^+]$ = lungs ÷ kidneys. These contributions shift so as to maintain healthy fluid pH levels, e.g., 7.4 in plasma.

Changing the numerator of the Henderson equation may result in respiratory acidosis/alkalosis, whereas changing the denominator may result in metabolic acidosis/alkalosis. When either the numerator or denominator changes, there is a corresponding compensatory change in the other, although this compensation is always incomplete, and can result in serious side effects unrecognized by practitioners everywhere. The Henderson-Hasselbach equation, which was developed later, states the same relationship in terms of pH, which is the negative logarithm of $[H^+]$,: $pH = pK + \log [HCO_3]$, where pK is the negative logarithm of the dissociation constant (for H_2CO_3), or in essence pH = kidneys ÷ lungs

The clinical physiology literature describes how disease, dysfunction, and deficit impact the Henderson equation, along with the consequences of disturbed acid-base balance and the price for its partial compensation. The origins of acid-base disturbances in this literature are addressed exclusively from a medical perspective, where only minimal lip service, at best, is paid to the behavioral contributions which continuously, immediately, and significantly regulate the numerator of the Henderson acid-base balance equation.

Breathing is behavior, and like any other behavior, it is regulated in varying degrees by learning, and thus by motivation, emotion, cognition, perception, and memory. Integrating learning with this physiology means that the Henderson equation can be expressed in yet another way: $[H^+]$ = breathing behavior ÷ kidney physiology, OR interestingly perhaps, *acid-base regulation = psychology ÷ physiology*. Changes in acid-base physiology regulate not only our physiology, but also our psychology, e.g., emotions, cognition, and even personality.

Breathing mediated neurophysiological pH regulation, for example, suggests that breathing may play an important role in the titration of subtle shifts in states of consciousness that mediate changes in cognitive, emotional, and behavioral patterns and hierarchies. It is surely an understatement to say that this profound relationship between behavior and physiology has been overlooked by both medical science and behavioral science practitioners. Even biofeedback and neurofeedback practitioners, who are trained in applied psychophysiology, are rarely familiar with this physiology and its far-reaching implications, e.g., how breathing mediates symptoms, deficits, and homeostatic deregulation resulting from stress.

HYPOCAPNIA AND ITS EFFECTS

Although CO_2 is, of course, excreted in the exhale, a significant portion of it is retained in the blood where it regulates pH levels vital to the distribution of oxygen and glucose to tissues such as the brain. In fact, while at rest, only about 12 to 14 percent of the CO_2 that travels in blood through the capillary bed of the lungs is actually excreted. In a healthy person, arterial CO_2 is precisely maintained (40 mmHg), even during exercise when CO_2 production may increase by tenfold.

Deregulated CO_2 chemistry results from either underbreathing or overbreathing. Underbreathing behavior, contrary to popular opinion, is rare in healthy people; it results in respiratory acidosis, which precipitates obvious, immediate, and uncomfortable sensations which in most cases are easily overcome by more rapid and/or deeper breathing. Overbreathing behavior, on the other hand, is common; it precipitates respiratory alkalosis (increased pH) brought about by a deficiency in extracellular fluid carbon dioxide (e.g., blood plasma), a physiological condition known as *hypocapnia* (CO_2 deficit). The effects can be insidious and dramatic.

Hypocapnia leads to physiological changes such as hypoxia (oxygen deficit), hemoglobin alterations (effecting release of oxygen and nitric oxide), cerebral vasoconstriction, coronary constriction, cerebral glucose deficit, ischemia (localized anemia), buffer depletion (bicarbonates and phosphates), bronchial constriction, gut constriction, neuronal excitability (sodium shifts), magnesium-calcium imbalance, hypokalemia (plasma potassium deficit), hyponatremia (plasma sodium deficiency), antioxidant depletion, platelet aggregation, and muscle fatigue, spasm (tetany), weakness, and pain.

These disturbances in physiology can trigger and exacerbate health-related complaints of all kinds, as well as deficits in physical performance (e.g., sports), including: phobias, migraine phenomena, hypertension, attention disorder, asthma attacks, angina attacks, heart attacks, cardiac arrhythmias, thrombosis (blood clotting) panic attacks, hypoglycemia, epileptic seizures, altitude sickness, sexual dysfunction, sleep disturbances (apnea), allergy, irritable bowel syndrome (IBS), repetitive strain injury (RSI), and chronic fatigue. The symptoms precipitated by overbreathing are dependent upon individual differences, including physiological propensities, physical compromise, health status, and psychological history. Overbreathing may also, of course, constitute a compensatory response to metabolic acidosis, e.g., ketoacidosis, by increasing pH.

The potentially debilitating combination of cerebral hypoxia and cerebral hypoglycemia, along with hemoglobin that is disinclined to give up its oxygen and the nitric oxide required for vasodilation, can result in profound psychological and behavioral changes: (1) deficits in ability to attend, focus, concentrate, imagine, rehearse the details of an action, engage in complex tasks, perform perceptual motor-skills (e.g., piloting vehicles), parallel-process information, problem solve, access relevant memory (e.g., test performance), think, and communicate effectively (e.g., public speaking); (2) emotional reactivity (e.g., marital conflict) that may trigger or exacerbate debilitating stressful states of consciousness, including, apprehension, anxiety, anger, frustration, fear, panic, vulnerability, and low self-esteem; and (3) personality shifts or dissociative states that result in social disconnectedness, emotional withdrawal, defensive posturing, emotional numbness, and inability to be present. How many neurofeedback practitioners consider the effects of hypocapnia?

Overbreathing is undoubtedly an insidious and debilitating response to everyday challenges, insidious because its presence goes unrecognized and its effects unidentified. In fact, surveys suggest that 10 percent or more of the US population suffers from chronic overbreathing and that 60 percent of all ambulance calls in major US cities are the result of overbreathing (Fried 1999)! For every person who shows up in emergency, how many more show up in physician's offices with unexplained symptoms? For every person who goes to see a physician, how many more simply go to work? And for everyone who reports a

"medical symptom" how many more suffer with performance deficits? Overbreathing is a behavior that precipitates changes in chemistry that can mediate these "unexplained symptoms," misunderstood performance deficits, and acute and chronic "effects of stress." The resulting effects of hypocapnia are profound and deserve full attention by virtually anyone doing breathing training.

THE IMPORTANCE OF GETTING THE PHYSIOLOGY "RIGHT"

Faulty assumptions and understandings about respiratory physiology are implicit in breathing training practices everywhere, which unfortunately, in many cases, may actually lead to counterproductive practice. Teaching good respiration through insistence on the mechanics associated with relaxation, for example, may create a problem rather than offer a solution; good respiration should not depend on being relaxed. And, teaching deep breathing for relaxation can, as a result of CO_2 deficit, trigger emotions, cognitive deficits, and misinterpreted physical effects. Breathing objectives, such as relaxation, must be ultimately subordinated to good respiration, and not the reverse as some would have it. *Evaluating, monitoring, and teaching good chemistry through breathing deserve serious attention by virtually anyone, layperson or professional, involved in learning and/or teaching breathing.* How many biofeedback practitioners are doing so?

Breathing training should not simply statistically favor good respiration, where the mechanisms responsible for positive outcomes are (1) only implicit in the training methodology, (2) unknown by both practitioner and client, and (3) often dismissed as not important in the name of "what we do works and that's what counts." Emphasis on slow breathing rather than on deregulated chemistry, for example, may *statistically favor* improvement of respiration, however it is easy to overbreathe while breathing slowly and does not by itself constitute better chemistry. It is important to know the underlying physiology that accounts for the positive outcomes of one's educational and therapeutic efforts, to make the implicit explicit, wherein relevant mechanisms are addressed directly rather than incidentally. And, in fact, as described earlier, some of these mechanisms are well documented in the fields of pulmonary and acid-base physiology, where focusing directly on chemistry and the basic mechanics that serve it, point the way to far greater efficacy, not to mention credibility.

Making the implicit explicit provides for direct focus on the variables that count, the ones that provide for the efficacy, including the kinds of clients that can be helped, the degree to which clients are helped, and the speed and cost of doing so. It also means (1) helping practitioners to evolve their interventions based on facts, rather than on tradition or professional rumors, (2) avoiding mixing effective factors with irrelevant ones, that take time, cost money, and side-track progress, (3) avoiding unwitting introduction of counterproductive elements of training, such as deep breathing, (4) avoiding faulty assumptions and misconceptions about what is required for healthy breathing, such as the suppositions by many that relaxation and slow breathing are necessarily prerequisite to good respiration, and (5) providing high impact patient education, where both the perceived efficacy and credibility of breathing self-regulation are enhanced.

GOOD CHEMISTRY TRAINING IS IMPLICITLY EMBEDDED

The relevance of breathing and acid-base physiology is illustrated below, as an example of how CO_2 and its regulation is *implicitly embedded* in breathing training traditions everywhere, in this case diaphragmatic breathing training for people who suffer with asthma.

(1) Fact: Increasing airway resistance, reducing lung compliance, and increasing bronchial constriction make it more difficult to breathe.

(2) Fact: Increasing airway resistance, reducing lung compliance, and increasing bronchial constriction increase the likelihood of asthma symptoms and attack.

(3) Fact: Lowering CO_2 levels *in airways* (local hypocapnia), through overbreathing, increases airway resistance, reduces lung compliance, and the likelihood of bronchial constriction.

(4) Fact: Making it more difficult to breathe, increases the effortfulness of breathing, and may introduce a sense of not being able to get one's breath, worry about breathing, and intentional efforts to get more air.

(5) Fact: Effortful breathing, "trying to get one's breath," increases the likelihood of overbreathing, which lowers CO_2 levels and results in increased airway resistance, reduced lung compliance, and increased bronchial constriction. These effects, as stated above, may then lead to greater difficulty in breathing and an increased likelihood of an asthma symptoms and attack.

(6) Fact: These factors described above, taken together, provide the ideal circumstances for vicious circle learning described in classical learning theory, involving both Pavlovian (emotional responses) and operant conditioning (breathing behavior) principles.

The vicious circle might go as follows:

(a) Anticipation of difficulty in breathing leads to fear of not getting enough air.
(b) Fear leads to reaching for more air.
(c) Reaching for air leads to overbreathing.
(d) Overbreathing leads to airway (local) hypocapnia.
(e) Airway hypocapnia increases the difficulty in breathing and likelihood of symptoms.
(f) Increased difficulty in breathing increases apprehension, worry, and fear.
(g) Cerebral hypocapnia exacerbates emotionality, and triggers fear, disorientation, and symptoms.
(h) Cerebral hypocapnia results in dissociation, e.g., emergence of defensive (asthma) personality.
(i) Emotionality and defensiveness result in "trying harder," failure, and sense of helplessness.
(j) Overbreathing sets the stage for the development of learned helplessness.
(k) Secondary gain for overbreathing sets the stage for learning dysfunctional breathing.
(l) Overbreathing generalizes as a coping style and becomes embedded in defensive personality.

(7) Fact: It has been clearly demonstrated that reducing breathing effortfulness through learning good diaphragmatic breathing helps people with asthma. This is the based on which "incentive spirometry" is implemented as a behavioral intervention worldwide to help reduce the likelihood of asthma attacks. Why can incentive spirometers be so helpful? What is the physiology that explains these results? It is hypothesized by many that the following considerations taken together make asthma symptoms and attacks less likely:

(1) Diaphragmatic breathing means much more air per breath.
(2) Diaphragmatic breathing means fewer breaths per minute for greater volumes of air.
(3) Diaphragmatic breathing by itself is less effortful than multi accessory muscle (chest) breathing.
(4) Greater use of the diaphragm eliminates the need for using accessory muscles.
(5) Effortless breathing reduces the physical "struggle" associated with "getting one's breath."
(6) Effortless breathing reduces fear, anxiety, and worry about breath.
(7) Diaphragmatic breathing results in slower breathing.
(8) Diaphragmatic breathing translates into relaxation, relief, a sense of confidence in breathing.

AND, teaching effortless diaphragmatic breathing may lead to:

(1) self-management of underbreathing,
(2) reduction of autonomic arousal, and
(3) a sense of self-empowerment.

Clearly, in the case of pulmonary pathology, good diaphragmatic breathing may very significantly improve tidal volume, and be absolutely essential to matching ventilation with perfusion, i.e., to ensuring adequate ventilation. This is the physiological basis for the benefits of learning through incentive spirometry. Although this is a significant self-management tool for increasing tidal volume, it says little, if anything, about factors that set the stage for bronchial constriction, increased airway resistance, reduced lung compliance, and the onset of asthma symptoms. In accounting for these physical changes, many practitioners point to the importance of emotional triggers and concomitant autonomic changes as the key factors, and hence to the importance of learning effortless breathing, relaxation, positive self talk, and physical confidence building.

Simply pointing to autonomic correlates, however, does not directly account for the physiological changes and symptoms associated with asthma. For example, how does autonomic arousal increase airway resistance, when, in fact, sympathetic activity actually decreases, not increases, airway resistance? And, how do slower breathing, decreased fear, and effortless breathing actually reduce the likelihood of asthma symptoms and attacks? Some of the physiology accounting for these considerations is likely to include: (1)

reduced airway (local) hypocapnia and its effects on airway resistance, lung compliance, and bronchial activity, and (2) reduced systemic hypocapnia (e.g., cerebral) and its effects on emotional reactivity and physical symptoms.

Diaphragmatic breathing is regulated by the brain stem medullary dorsal respiratory group (DRG) in accordance with the Henderson and Henderson-Hasselbach equations, which includes changes in pH or $[H^+]$, HCO_3 (bicarbonates), and $PaCO_2$ (and other fluid PCO_2). Using other breathing accessory muscles, such as during so-called "chest breathing," may quickly result in deregulation of this basic brain stem reflex, and increase the likelihood of deregulated chemistry; in fact, the onset of its effects, overlooked by most, may even be falsely attributed to both asthma and autonomic arousal. And, of course, breathing training which decreases the likelihood of hypocapnia, reduces the probability of deregulation. Here is a partial accounting of such variables:

Factors that trigger hypocapnia	Factors leading to hypocapnia prevention
Worry about breathing	Breathing self-confidence, trust
Using accessory muscles	Diaphragmatic control
Intentional breathing	Allowing breathing to happen
Deep breathing	Quiet effortless breathing
Rapid breathing	Allowing for exhale and its transition
Fear and anxiety	Relaxation
Defensiveness	Embracement
Negative self-talk	Self-affirmations
Misinformation about breathing	Education
Secondary responses to physical symptoms	Counterconditioning

Variables that decrease the likelihood of hypocapnia are *implicitly embedded* in effective breathing training protocols, e.g., incentive spirometry training. Unfortunately, however, behaviors which increase the likelihood of hypocapnia are also often encouraged by trainers, who unfortunately don't know about the basic biochemistry involved. Emphasis on deep diaphragmatic breathing during incentive spirometry training may result in the self-defeating effects of hypocapnia, whereas emphasis on quiet effortless diaphragmatic breathing is likely to normalize PCO_2 levels.

OVERBREATHING BEHAVIOR

Optimal respiration means regulating chemistry, through proper ventilation of CO2, relaxed or not, such as during the acrobatics of talking, emotional encounters, and professional challenges. Good breathing chemistry establishes a system-wide context conducive to optimizing physical and psychological competence, where chemistry needs to be balanced regardless of what we are doing, thinking, or feeling. Nevertheless, overbreathing behavior, like any other maladaptive behavior can be quickly and easily learned, and unfortunately, like so many habits, are often challenging to disengage, manage, modify, or eliminate; the learning principles, are the same.

Overbreathing can be learned as a defensive response to specific challenges (e.g., performing before an audience, or confronting a distressed partner), or it can mediate shifts in consciousness that set the stage for learning constellations of defensive behaviors that serve to protect against trauma, including people, things, and oneself. The desire or need for "control" is a metaphor frequently embedded in deregulated breathing behavioral patterns. These defensive behaviors, like many vicious circle behaviors, may come at a high cost, as described above: physical symptoms, emotional reactivity, cognitive deficits, and performance decrements with immediate, long-term, and profound effects. Herbert Fensterheim (Timmons & Lay, 1994), an internationally prominent psychotherapist, points to these considerations in addressing mental health professionals when he says:

> "Given the high frequency of incorrect breathing patterns in the adult population, attention to the symptoms of hyperventilation [overbreathing] should be a routine part of every psychological evaluation, regardless of the specific presenting complaints. Faulty breathing patterns affect patients differently. They may be the central problem, directly bringing on the pathological symptoms; they may magnify, exacerbate, or maintain symptoms brought on by other causes; or they may be involved in peripheral problems that must be ameliorated before psychotherapeutic access is gained to the core treatment targets. Their manifestations may be direct and obvious, as when overbreathing leads to a panic attack, or they may initiate or maintain subtle symptoms that perpetuate an entire personality disorder. Diagnosis of hyperventilatory [overbreathing] conditions is crucial."

Although breathing is subject to the same learning principles as any other behavior, it is a unique behavior in a number of significant ways, ways which makes its deregulation of very special concern to practitioners interested in teaching self-regulation for health and performance:

(1) It is a "perpetual" behavior. It does not emerge only at specific times and places. It takes place virtually all of the time, and when briefly it does not, its absence is still relevant.

(2) It is a behavior that is necessarily woven into virtually every mindful-physiology tapestry. It is an inevitable part of every behavioral topography. It transitions from one topography into the next, and may carry with it behaviors, emotions, memories, thoughts, symptoms, senses of self, personality styles, and physical reactions from the previous topography.

(3) It is serves as a gateway wherein it set stages, creates backdrops of meaning, establishes contexts, and changes states for management of the mindfulness of physiology.
(4) It is controlled centrally from diverse neurophysiological sites as well as locally by cells and tissues. Throughout the day it is voluntary and involuntary, conscious and unconscious.

(5) It is critical to basic human functions, including not only acid-base physiology and the delivery of oxygen and glucose, but is vital to social behaviors such as verbal communication.

(6) Its basic nature is reflexive. Intentional practice can be difficult because you can't do it for a while, take a break, and then continue again when you feel more confident. It will happen anyway. You can't avoid it. It's always there. You can't put it aside if you don't like it.

CAPNOBREATH TRAINING

CapnoBreath Training (where "capno" means CO_2) is about learning and teaching adaptive respiratory chemistry within a wide range of breathing mechanics. It means precision coordinating of breathing rate and depth through reflex control of the diaphragm, a brain stem coordinated reflex mechanism which can be easily deregulated, consciously or unconsciously. CapnoBreath training is about reinstating this reflex mechanism. It means integrating knowledge of respiratory chemistry with the mechanics of breathing, where emphasis is on the relationship dynamics of breathing mechanics for achieving good chemistry, rather than on specific "mechanics" prescriptions (e.g., a specific breathing rate), where the effects of breathing chemistry are neither accounted for during initial evaluation nor included as a part of self-regulation learning. Good respiration requires neither relaxation nor a specific mechanical prescription, save one: the varied melodies of breathing mechanics must ultimately play the music of balanced chemistry.

CapnoBreath training includes exploration, education, play, and training as follows:

(1) exploration: originating and sustaining factors and circumstances;
(2) identification: dysfunctional breathing patterns, when and where;
(3) phenomenology: feelings, memories, thoughts, and sense of self;
(4) knowledge-learning: understanding basic breathing concepts;
(5) sign-learning: recognizing physical, psychological, and behavioral symptoms;
(6) mechanics-learning: play for diaphragmatic, rate, & depth awareness;
(7) visceral-learning: developing an internalized sense of chemistry; and
(8) state-learning: developing a sense of chemistry for consciousness (e.g., emotions).

CapnoBreath training, in the larger context, is about learning "to embrace" (or to engage) a challenge rather than to "defend from" a challenge. Embracing means "being present," connecting, and learning, where defending (or bracing) means armoring, isolating, and disconnecting. Healthy breathing should not be state or context specific, e.g. during meditation, relaxation, or prophylactic intervention. CapnoBreath training is about learning to breathe with the whole body; every cell breathes, not just the lungs. Learning good respiration is learning about what breathing "feels like," and is ultimately not about what breathing "looks like." CapnoBreath training is about learning to breathe inside-out, rather than outside-in.

THE EFFICACY OF CAPNOBREATH TRAINING

Clinicians and researchers everywhere substantiate the "efficacy" of their techniques, protocols, interventions, training methods, and educational programs based on how they "stack up" based on whether or not, and to what degree they impact, for example, hypertension. But, what about the efficacy of reducing hypertension itself on heart attack or stroke? Does reducing hypertension through biofeedback, for example,

actually reduce the likelihood of a heart attack, or is this just an assumption based on statistics about hypertension? Few of us ask these questions. We simply assume that biofeedback regulated blood pressure is synonymous with reducing the likelihood of heart attack or stroke: the link is simply a leap of faith, not science.

Curiously, on the other hand, although the clinical research literature very clearly, without controversy, and with exceptionally well documented science, points to the profound effects of hypocapnia, no one talks about substantiating the efficacy of their interventions, e.g., biofeedback, based on changes in $PaCO_2$ which lie at the very heart of acid-base physiology and its effects on health and performance! Additionally, and important to point out, is the fact that breathing regulation is under our immediate and direct control (in healthy people), and unlike methods contributing to changes in blood pressure physiology, shifts in breathing are not indirect and do not require life style changes.

Although the importance, relevance, and efficacy of breathing training is indisputably acclaimed and widely practiced in professional and lay circles worldwide, it is curious, indeed, that the practical relevance and efficacy of breathing chemistry (CapnoBreath) training, which rests on the firm ground of a vast empirical science, is so frequently challenged and questioned by these very same practitioners. It is this chemistry which, in fact, offers up perhaps the most fundamental reason for the efficacy of breathing learning and training so widely embraced by all.

Making the case for (1) the clinical and educational relevance of the management of hypocapnia, and (2) the potential effectiveness of clinical and educational interventions for its amelioration, far exceeds making the case for blood pressure reduction, or most any other behavioral intervention that involves physiological self-regulation learning. Why is training for good chemistry not widely recognized and practiced by practitioners everywhere? The answer is simple: this fundamental, highly-documented, non-controversial practical science has not been adequately and effectively brought to their attention. **Now is the time.**

Note: When perfusion is greater than ventilation, some of the blood that passes through the pulmonary capillary network is not ventilated. This means that some of the CO_2, which would otherwise diffuse into the alveoli, is returned to the arterial system. This blood mixes with the blood that has been partially or fully ventilated, with the result that the arterial $PaCO_2$ is higher than it would be otherwise. This is known as "CO_2 retention," a phenomenon identified with people who suffer with asthma and other pulmonary disorders. There is, of course, immediate compensation for "CO_2 retention," as a result of increased ventilatory drive, which restores normal $PaCO_2$ (arterial) levels.

Although $PaCO_2$ levels may be normalized (eucapnia), $ETCO_2$ levels (end-tidal CO_2) will be lower, giving the uniformed observer the false impression of overbreathing and hypocapnia. The readings are lower because of (1) the mixture of alveolar gases containing different partial pressures of CO_2, and (2) the diffusion of proportionately greater amounts of CO_2 in alveoli which are fully ventilated. Thus, although alveolar and end-tidal CO_2 are low, $PaCO_2$ may be normal. This is often the explanation as to why people with asthma "overbreathe:" they have "CO2 retention." If this is true, of course, they aren't really overbreathing: there is no (systemic) hypocapnia. Unfortunately, this "organic variable," leads many to precluding further behavioral considerations, when in fact its very presence establishes the basis for learning dysfunctional breathing: the resulting local airway hypocapnia may produce asthma symptoms, leading to the vicious circle learning pattern described above, with the consequence of systemic hypocapnia (low $PaCO_2$) and its effects on emotionality and physical symptoms. Mismatch of ventilation and perfusion does not preclude overbreathing behavior in people with asthma, but rather serves to set the stage for its acquisition.

REFERENCES

Fried, R. (1999). *Breathe well, be well*. New York: John Wiley & Sons.

HealthCare Professional Guides (1998). *Anatomy and Physiology*. Springhouse: Springhouse Corporation.

Laffey, J.G., & Kavanagh, B.P. (2002). Hypocapnia. *New England Journal of Medicine*, 347(1) 43-53.

Levitzky, M. G., (2003). *Pulmonary Physiology*. New York: McGraw Hill.

Thomson, W. S. T., Adams, J. F., & Cowan, R. A. (1997) *Clinical Acid-Base Balance*.
New York: Oxford University Press.

Timmons, B. H., & Ley, R. (editors, 1994). *Behavioral and psychological approaches to breathing disorders*.
New York: Plenum Press.