

CapnoLearning: Respiratory Fitness and Acid-Base Regulation

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CapnoLearning® is about learning breathing behaviors that facilitate optimal respiration and its associated regulation of acid-base physiology. CapnoLearning involves the use of a capnometer (or capnograph) which provides real-time information about carbon dioxide (CO₂) retention in the alveoli of the lungs for evaluating learned breathing behaviors that serve respiratory chemistry. Clients discover how they have learned to breathe, how their breathing affects them, and how to effectively self-regulate breathing behavior based on learning rather than prescriptive exercise. CapnoLearning includes the application of principles of phenomenological exploration, behavioral analysis, behavior modification, biofeedback, awareness training, and cognitive learning.

Few people, lay or professional, know that (1) breathing directly regulates body chemistry, including pH, electrolyte balance, blood flow, hemoglobin chemistry, and kidney function, and that (2) breathing is a behavior subject to the same principles of learning as any other behavior, including the role of motivation, reinforcement, emotion, attention, perception, and memory. Bringing together these two simple facts means integrating the biological and behavioral sciences in profoundly practical ways relevant to the lives of millions who have unwittingly learned breathing behaviors that compromise respiration and acid-base balance.

Breathing is behavior, and as a behavior it serves multiple objectives. Although, respiration is obviously and unquestionably the fundamental objective, breathing is required for talking, for singing, and for coughing. It is indicated in relaxation, yoga, and meditation. It is utilized as defensive behavior for triggering emotions (e.g., anger), for dissociating from trauma, for reducing fear (avoidance learning), and for achieving secondary gain (operant learning). None of these considerations, however, may necessarily be associated with healthy respiratory chemistry, and unfortunately, quite often to the contrary. CapnoLearning is about learning breathing behavior that serves respiratory physiology and its associated acid-base regulation, that is, respiratory fitness.

Respiration

Respiration can be broken up conceptually into three phases: external, internal, and cellular respiration. External respiration is about the mechanics of breathing, moving gases (air) in and out of the lungs. Internal respiration is about ensuring the transport of oxygen in the blood from the lungs to tissue cells, and then the transport of metabolic CO₂ from tissue cells to the lungs for both its excretion and its reallocation to systemic circulation for acid-base regulation. Cellular respiration is the utilization of oxygen in mitochondria for the synthesis of adenosine triphosphate (ATP), molecules that cells ultimately break down for their energy.

Basic to external respiration is the subject of gas exchange. Gases are measured by virtue of the pressures that they exert. When gases are mixed, e.g., air, they each contribute to a total pressure. Each gas contributes a partial pressure (P). Total atmospheric air pressure at sea level, at 15 degrees C and zero humidity, is 760 mmHg (millimeters of mercury). At sea level partial pressure oxygen, written PO₂, is 159 mmHg (20.93%), and partial pressure carbon dioxide, written PCO₂, is 0.3 mmHg (less than 0.04%). Most of the gas exchange, O₂ and CO₂, takes place in the fundamental alveolar-capillary unit, the alveolus. There are about 300 million alveoli in the lungs, surrounded by about 280 billion pulmonary capillaries. Capnography is about measurement of average alveolar PCO₂, which is observed/measured in the final portion of exhalation when gases are presumably 100 percent alveolar (not mixed with anatomical dead space gases); this measurement is known as End Tidal CO₂, or ETCO₂, or PCO₂ at the “end of the tides of air,” that is, when the “tide is out.”

The ***Henderson-Hasselbalch (H-H) equation*** is central to understanding internal respiration, which describes pH regulation in extracellular fluids: $pH = [HCO_3^-] \div PCO_2$ (in its simplified conceptual format), wherein PCO₂ is regulated by breathing, and bicarbonate concentration [HCO₃⁻] is regulated by the kidneys. These fluids include blood plasma, interstitial (fluids that surround tissue cells), lymph, and cerebrospinal fluids. Changes in the numerator of the equation, bicarbonate concentration, are generally slow (8 hours to 5 days), whereas changes in the denominator, partial pressure carbon dioxide (PCO₂), are immediate. This places breathing center stage in moment-to-moment acid-base regulation. Arterial levels of PCO₂, known as PaCO₂, remain between 35 and 45 mmHg to keep plasma pH within its normal range (7.35 to 7.45). In normal healthy lungs, when perfusion (blood) and ventilation (air) are matched, alveolar PCO₂ (and hence, ETCO₂) is approximately equivalent to PaCO₂.

Balancing the Henderson-Hasselbalch (H-H) equation is achieved through the presence of receptor sites in (1) the brainstem that are sensitive to interstitial pH and PCO₂, and (2) the arterial system (aorta and carotid arteries) that are sensitive to plasma pH and PCO₂. Changes in pH and PCO₂ in both locations together drive the respiratory centers in the brainstem, along with partial pressure oxygen (PO₂) changes detected also at arterial receptor sites. If pH is too low (< 7.35), or too high (>7.45), PaCO₂ is reduced or increased by altering breathing rate and depth (minute volume). Brainstem reflex-regulated breathing, under normal circumstances, maintains alveolar PCO₂ at 35 to 45 mmHg, wherein rapid diffusion from alveolus to pulmonary capillary provides for almost immediate equilibration, thus ensuring a PaCO₂ of about the same value.

Actual quantities of carbon dioxide generated by the body vary considerably based on metabolism, e.g., meditation vs. exercise, although the PaCO₂ values required for maintaining acid-base balance remain the same. At rest, for example, only about 15 percent of the CO₂ arriving in the lungs is actually excreted; the balance is reallocated to systemic circulation. Capnograph instrumentation does not indicate how much CO₂ is being exhaled, rather it indicates the alveolar PCO₂ being maintained, and thus the approximate PaCO₂.

Behavioral hypocapnia

The H-H equation from a medical perspective is about reflexes. When bicarbonate concentration [HCO₃⁻] drops as a result of metabolic acidosis, e.g., lactic acidosis during anaerobic exercise, breathing is considered to be a reflexive compensatory response that contributes to restoration of acid-base balance. When PCO₂ is too low, extracellular pH rises with resulting *respiratory alkalosis*, a condition identified as *hypocapnia*. When PCO₂ is too high, extracellular pH falls with resulting *respiratory acidosis*, a condition identified as *hypercapnia*. The medical perspective offers up organic explanations that may give rise to these conditions. Integrating behavioral psychology with the H-H equation, however, sets the stage for examining these conditions from a learning perspective where the denominator of the equation may be directly regulated by powerful reinforcement of operant breathing behaviors that compromise acid-base balance. Thus, the equation might be rewritten as follows: ***acid-base regulation (pH) = physiology [HCO₃⁻] ÷ behavior (breathing for PCO₂ changes)***. The implications are impressive.

Learned *overbreathing* behavior results in *behavioral hypocapnia*, where breathing rate and depth are mismatched. Its consequence is an increased level of pH, or respiratory alkalosis, which may have profound immediate and long-term effects that may trigger, exacerbate, and/or cause a wide variety of emotional (anxiety, anger), cognitive (attention, learning), behavioral (public speaking, test taking), and physical (pain, asthma) changes that may seriously impact health and performance (Fried, 1987; Laffey & Kavanagh, 2002). Practically speaking, behavioral hypocapnia is defined as ETCO₂ readings below 35 mmHg brought about by learned breathing patterns: 30-35 mmHg is mild to moderate, 25-30 mmHg is serious, and 20-25 mmHg is severe hypocapnia. Behavioral hypocapnia reduces respiratory fitness and disturbs acid-base chemistry as follows:

- Hypocapnia increases red blood cell alkalinity and reduces red cell CO₂ levels, thereby increasing hemoglobin's affinity for oxygen (Bohr Effect).¹ The consequence is "unfriendly" hemoglobin: oxygen saturation rises (HbO₂) but oxygen distribution to tissues is compromised. Note that the uninformed practitioner may mistakenly interpret higher saturation readings taken with an oximeter as a sign of improved respiration. The same red blood cell physiology also restricts the amount of nitric oxide (a potent vasodilator) released by hemoglobin, resulting in significant vasoconstriction, even ischemia. These two factors together may very significantly reduce reduction of oxygen and glucose to cells that require them.
- Hypocapnia increases plasma alkalinity, thereby triggering significant electrolyte changes. Calcium ions migrate into muscles in exchange for hydrogen ions, resulting in their immediate constriction, e.g., arteries, gut, and bronchioles. Vasoconstriction can lower cerebral and coronary blood flow/volume by up to 50 percent in a matter of seconds. Bronchiole constriction increases airway resistance and may trigger asthma symptoms or precipitate an attack. Gut constriction may result in nausea and cramping, as in the case of altitude sickness. Calcium-magnesium imbalance in skeletal muscles may increase the likelihood of spasm and fatigue. Sodium and potassium ions in interstitial fluids migrate into cells in exchange for hydrogen ions resulting in sodium and potassium deficiencies.
- Chronic hypocapnia orchestrates yet different physiological changes. The kidney requires CO₂ for the reabsorption of both bicarbonate and sodium ions, as well as for generating new bicarbonates lost in the urine as a result of buffering acids generated by protein breakdown (e.g., phosphoric acid). The resulting bicarbonate and sodium deficiencies may include some of the same effects as those identified with chronic stress, e.g., fatigue. Other effects include: elevated platelet level, aggregation, and "adhering" propensity; antioxidant depletion as a result of excitotoxin production (e.g., glutamate); and systemic inflammation.
- Hypocapnia may set the stage for intracellular lactic acidosis (e.g., in neurons) by significant reductions in oxygen supply and increased cellular metabolism resulting from the influx of sodium and potassium.

Here are some of the symptoms and deficits triggered, exacerbated, caused, or perpetuated by hypocapnia:

RESPIRATION: shortness of breath, breathlessness, bronchial constriction and spasm, airway resistance, reduced lung compliance, asthma symptoms; CHEST: tightness, pressure, and pain; PERIPHERAL CHANGES: trembling, twitching, shivering, sweatiness, coldness, tingling, and numbness; HEART: palpitations, increased rate, angina symptoms, arrhythmias, nonspecific pain, ECG abnormalities; EMOTION: anxiety, anger, panic, apprehension, worry, crying, low mood, frustration, performance anxiety, phobia, generalized anxiety; STRESS: tenseness, acute fatigue, chronic fatigue, effort syndrome weakness, headache, burnout; SENSES: blurred vision, dry mouth, sound seems distant, reduced pain threshold; CONSCIOUSNESS: dizziness, loss of balance, fainting, black-out, confusion, disorientation, disconnectedness, hallucinations, traumatic memories, self-esteem, personality shifts; COGNITION: attention deficit, inability to think, poor memory, learning deficits; MUSCLES: tetany, hyperreflexia, spasm, weakness, fatigue, pain; ABDOMEN: nausea, cramping, and bloatedness; MOVEMENT: coordination, reaction time, balance; VASCULAR: hypertension, migraine, digital artery spasm, ischemia; BLOOD: red blood cell rigidity, thrombosis; SLEEP: apnea; PERFORMANCE: endurance, altitude sickness.

Breathing behavior

The above symptoms and deficits, mediated by learned breathing behaviors that disturb basic acid-base chemistry, typically go “unexplained” or are mistakenly attributed to other unrelated causes, e.g., stress. In this context of thinking, these effects become ***behavioral consequences***, rather than unexplained clinical symptoms and performance deficits. Most forms of breathing training, however, do not explicitly address the alignment of external with internal respiration, but rather focus on the mechanics of breathing, usually in the service of diminishing sympathetic arousal or changing states of consciousness, e.g., relaxation, meditation, yoga. Unfortunately, however, it is usually implicitly assumed that the specific breathing mechanics embedded in these practices (e.g., breathing more deeply and more slowly) necessarily pave the way to optimal respiration. This is a big mistake.

Respiratory fitness is vital to health and performance, and must be regulated despite the breathing acrobatics of talking, emotional encounters, and professional challenges. As a result of very specific learning, dictated by specific learning circumstances, breathing may “change on a dime” as a function of where (s)he is, who (s)he is with, and what (s)he may be doing, thinking, and feeling. Respiratory fitness needs to be in place regardless of whether or not one is relaxed or stressed, excited or bored, active or inactive, working or playing, focused or distracted. ***“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”*** (Litchfield & Tsuda, 2006). To insist on slow breathing and relaxation, for example, may be not only unrealistic, but may also be counterproductive.

CapnoLearning is about the application of traditional learning theory to breathing behavior. Applied behavioral analysis and behavior modification are thus central considerations. Behavioral detective work is essential, which means pinpointing the history of learned breathing behavior along with the factors that may be sustaining it. If overbreathing is a reinforced operant behavior, ***simply teaching clients the “right” mechanics may be both irrelevant and misleading***. Practicing “good” mechanics may mean nothing more than repetitive exercises that attest to one’s skills to consciously manipulate breathing behavior, which often may in itself be a problem. If learning history is overlooked, training will fail. The governing factors will continue to govern.

Basic learning considerations include classical conditioning, operant conditioning, two-factor learning, avoidance learning, state-dependent learning, cognitive learning, and biofeedback. Operant conditioning includes concepts such as: operant response (e.g., breathing rapidly), positive reinforcement (e.g., feeling in control), negative reinforcement (e.g., fear reduction), and discriminative stimulus (e.g., sense of breathlessness). Discriminative stimuli (S^D) trigger operant behaviors based on reinforcement contingencies, e.g., “feeling challenged by an authority figure” may serve as an S^D for accessory muscle (chest) breathing, an operant response positively reinforced by “feeling in control.” Basic classical conditioning concepts, as applied to breathing behavior, include the (CS) conditioned stimulus (e.g., the experience of small breaths) and the (CR) conditioned response (e.g., fear), i.e., small breaths elicit fear. Both kinds of learning may be state-dependent which means that they may only be triggered in specific states, e.g., when hypocapnic. In fact, chronic hypocapnia may become the gateway into a different personality, a different sense of self, thus making it a form of “chemical” dependency.

Operant and classical conditioning invariably work together, and comprise what is known as two-factor learning. Classically conditioned responses provide both the motivation and reinforcement for the operant behaviors. For example, classically conditioned fear of the transition time between breaths (a conditioned stimulus) provides motivation for aborting the exhale (an operant response), which is then reinforced by fear reduction (a negative reinforcement). The transition time serves both as a conditioned stimulus AND as a discriminative stimulus. These principles, as applied to breathing behavior, are illustrated in the case described below.

Case history

For several years a physiotherapist, previously an Olympic athlete, was frequently (weekly) unable to go to work, finding herself “without enough oxygen” (breathlessness) usually immediately after having eaten breakfast. Her consultations with the medical profession had led nowhere, only to psychiatric medications. Based on her own interpretation of the symptoms, she was certain that underbreathing was her problem. Her solution was to implement relaxation and breathing techniques she had previously learned about from books and colleagues.

Her ETCO_2 levels were observed with an educational capnograph (the CapnoTrainer®, manufactured by Better Physiology Ltd) at rest as well as when challenged with specific tasks and emotions. There was no evidence of overbreathing, or hypocapnia. Nevertheless, based on her responses on the Breathing Interview Checklist (Litchfield) and the ensuing interview, it was decided to introduce guided intentional overbreathing where symptoms, emotions, and memories could be explored as her ETCO_2 values slowly diminished. After about a minute of increasing the depth of breathing while slowing the rate, her ETCO_2 dropped to about 28 mmHg, where upon she immediately exclaimed that “this is exactly what happens to me, and now I won’t be able to get out of it for the rest of the day!” When it was pointed out that the procedure was identical to that which she had described as her own solution to the symptoms, she was flabbergasted when she could also see for herself that her solution to the problem was its cause!

When she was asked to reinstate her previous pattern of eucapnic (normal PCO_2) breathing, she failed to be able to do so. She was then coached for recovery, which included the following elements: diaphragmatic breathing (to make breathing easy), passive exhale (to eliminate intention), allowing for more transition time between the exhale and the inhale (to extinguish fear), *systematically minimizing the size of the breath* (the desired operant), and thoughts about happy events (to change the state). Within about four minutes her PCO_2 levels normalized and she felt relaxed and relieved. As these instructions were not consistent with her belief system, she expressed her incredulity about the success of taking smaller breaths over and over again. Guided overbreathing was introduced a second time, and once again she was trapped in her vicious circle pattern and was unable to reinstate eucapnic breathing. After four or five occasions, however, with PCO_2 biofeedback she quickly learned (1) how to intentionally create a hypocapnic state by overbreathing (negative practice), and then (2) how to intentionally restore optimal respiration (35 -40 mmHg) while experiencing hypocapnia. She was no longer victim to her own learned vicious circle breathing behavior.

Eating and breathing behaviors had become decoupled as a result of being in a hurry to finish breakfast, with the result of disturbed respiration and the associated hypocapnic symptoms, e.g., breathlessness. Besides not being aware of the origin of the problem, nor of the behavior she had learned as a consequence, both her interpretation (e.g., not getting enough O_2) of her symptoms (e.g., breathlessness) and her solution (e.g., big breaths) to their amelioration were faulty. Thus, her first step to resolving her vicious circle breathing pattern was cognitive: to learn a new belief system about breathing based on the facts, to interpret her own breathing experience in a new way, and to replace her old hypocapnic self-talk, “I can’t get enough oxygen,” with new self-talk, “my body knows what to do.” These new cognitive behaviors are best learned through awareness training during the interval between exhalation and inhalation, where and when the brainstem reflexes can be identified and experienced.² Allowing for the reflex, and feeling it in action, builds a sense of confidence, an important new positive reinforcement for learning new breathing behaviors.

The second step was to identify the (operantly) learned breathing behaviors (e.g., taking deep breaths) that steered her into hypocapnia, the specific discriminative stimuli (e.g., feeling in a hurry while eating) that triggered these behaviors, the classically conditioned stimuli (small breaths) that triggered the emotions (e.g., fear) that motivated the behaviors, and the reinforcements that maintained the behaviors (e.g., fear reduction). Her learning was based on PCO_2 biofeedback where she learned about the effects of breathing patterns on her physiology and psychology.

The third step was to extinguish the conditioned fear response (the CR) to small breaths (the CS), thereby removing the negative reinforcement for bigger breaths (fear reduction). The fourth step was to learn new breathing behaviors (e.g., taking small breaths) triggered by new discriminative stimuli (e.g., early-on symptoms of hypocapnia), and new conditioned stimuli (e.g., also early-on hypocapnic symptoms) that provided new sources of motivation and reinforcement (e.g., instant relief) for the new behavior. All of these learning considerations were specific to the client and became embedded in her own physiology, a part of who she is; they were not generic prescriptive exercises simply imposed on a learned faulty breathing pattern.

Efficacy

Does CapnoLearning work? In regard to efficacy, four separate questions emerge: (1) Have changes in respiratory chemistry been clearly demonstrated to regulate the appearance and disappearance of physical and mental symptoms and deficits? Yes. The answers abound in pulmonary and acid-base physiology textbooks everywhere (e.g., Levitsky, 2007). (2) Are the behavioral techniques utilized for assessing behavior, extinguishing behaviors, and learning new behaviors supported by the research literature? Yes. The answers abound in behavioral psychology textbooks everywhere (e.g., Miltenberger, 2008). (3) Are these same behavioral techniques successful when applied to breathing behavior? Yes (e.g., Ley, 2001). And, (4) does restoring good respiration in clients with compromised respiration ameliorate specific symptoms and deficits? The answer is always, “it depends.” It depends on how compromised respiration may be playing a role in a specific client’s presenting complaints. And thus, learning new breathing behaviors may not help, help a little, help some of the time, help a lot, or eliminate the problem altogether.

Millions of people, worldwide, teach and learn about breathing, but unfortunately, little of what is practiced is rooted in the textbook sciences of pulmonary physiology (e.g., Levitsky, 2007), acid-base physiology (e.g., Thomson, Adams, & Cowan, 1997), behavioral analysis (e.g., Leslie 2005), behavior modification (e.g., Kazdin, 2001), cognitive learning (Freeman, 2005), biofeedback (e.g., Schwartz & Andrasik, 2003), and the psychology of respiration (e.g., Fried, 1987, 1993). Unfortunately, misinformation, misconceptions, pseudoscience prescriptions, and ignorance about breathing and how it affects respiration have predominated with little attention, if any, paid to these immensely rich literatures and their relevance to breathing behavior. Failure to directly address breathing as learned behavior, and how it regulates fundamental body chemistry, means leaving out the most fundamental, practical, and profound factors that account for (1) the far-reaching effects of maladaptive breathing habits, as well as for (2) the surprising benefits of learning breathing behaviors that optimize respiration. CapnoLearning represents an effort to address this opportunity.

Footnotes:

1The Bohr Effect is descriptive of how (1) the presence of carbon dioxide in red blood cells and (2) the pH of blood cell cytosol, alter the affinity of hemoglobin for oxygen as depicted in the oxyhemoglobin (hemoglobin that is carrying oxygen) dissociation curve. This S-shaped curve specifies the relationship between PO₂ and hemoglobin oxygen saturation (SaO₂), wherein as PO₂ drops so too does SaO₂. Decreased levels of carbon dioxide and increased levels of pH (higher alkalinity), as a result of hypocapnia, increase hemoglobin’s affinity for oxygen, thus moving the dissociation curve to the left and requiring yet lower levels of PO₂ for the release of oxygen.

2The brainstem reflex referred to here is the dorsal respiratory groups (DRG), a respiratory medullary center located in the nucleus of the tractus solitarius (NTS), which consists mostly of inspiratory neurons. The center is the principal regulator of diaphragmatic activity. This reflex can be directly experienced, with practice, as “air hunger” during the interval immediately preceding inhalation.

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