

FEATURE

Better Chemistry Through Breathing: The Story of Carbon Dioxide and How It Can Go Wrong

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Certain aspects of the chemistry underlying respiration should be considered when recommending breathing changes to clients. The interplay of breathing rate, breathing depth, and carbon dioxide (CO₂) processing is sometimes dysregulated when psychological factors dominate over physiological ones. Recommending “deep breathing” as a relaxation method can backfire if it initiates hyperventilation symptoms and discourages breath regulation. This article explains some details of breathing/CO₂/physiology interactions and offers suggestions regarding giving advice about breathing.

Introduction

In the biofeedback field we often give advice about breathing, so it helps to know about the underlying biochemistry in order to understand what can go wrong and something about how to fix it. Behaviorally, breathing is generally taken for granted; it goes on without our supervision, we breathe more during exertion, and we have a strong reflex to prevent suffocation. Because of our easy access to the breathing mechanism, however, and because we can think beyond the immediate present, we are at unique risk for disrupting the biochemical balance that normally allows for smooth physiological functioning.

Hyperventilation

Hyperventilation is defined as breathing in excess of current metabolic needs. It does not need to be florid and dramatic, or even fast; it can be unnoticeable to the casual observer but still cause trouble. For the majority of people, just 1 minute of determined overbreathing reduces cutaneous and peripheral blood circulation (colder hands, paler skin); interferes with peripheral nerve conduction (tingling in fingers and lips); lowers muscle contraction thresholds (more susceptible to twitching); and stimulates cerebral vasoconstriction (interference with vision, hearing, sensory perception, balance, orientation, judgment, and cognition). Episodes

of acute hyperventilation are usually brief and during emotional shock or a panic attack. However, symptoms such as the above can also develop to a lesser degree from chronic hyperventilation, which is more common.

How could this be, that breathing too much can cause such problems? Carbon dioxide (CO₂) is a waste gas destined for excretion, but it also has a crucial role in regulation of the body's pH and vascular tone: during its brief ride in the bloodstream, CO₂ is converted to carbonic acid and has a large responsibility for maintaining the body's pH. Normally there is a close match, breath by breath, between the amounts of CO₂ produced and exhaled. Therefore, when we breathe out more CO₂ than we produce, hyperventilation occurs; this creates a CO₂ deficit, which is called hypocapnia. Conversely, if breathing is not sufficient for the body's immediate needs, CO₂ starts to build up in the body; this is hypoventilation, producing a CO₂ excess. In this case lack of oxygen is a corollary problem, but in hyperventilation, it is not; the excess oxygen is simply excreted unused (see Figures 1 and 2). Details can be found in many sources, including the general references at the end.

Our need for oxygen is increased by muscle activity, and exercising generates large amounts of CO₂, which is dumped into the bloodstream. With more breathing, more CO₂ is excreted, but since more is also being generated, CO₂ in the bloodstream stays stable: about 5% in arterial blood, 38–40 mmHg, or “torr.” This maintains blood pH at around 7.35. Significant pH shift toward acidity leads to major metabolic problems and eventually to coma, whereas prolonged alkalinity creates a general hyperexcitability, and in the extreme this will lead to convulsions.

If the breath is stopped or inhibited, CO₂ rises and blood vessel diameter increases in order to maximize transfer of glucose and oxygen out of the bloodstream and into the tissues. But with hyperventilation, the opposite happens: blood vessels constrict, inhibiting transfer of nutrients from the bloodstream. This creates

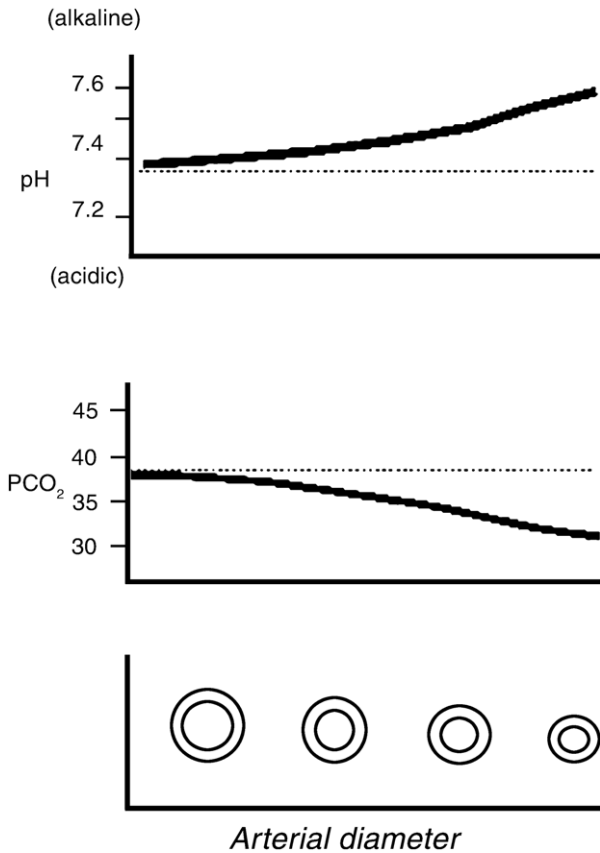


Figure 1. Schematic relationship between pCO₂, pH, and blood vessel diameter (hyperventilation depletes CO₂).

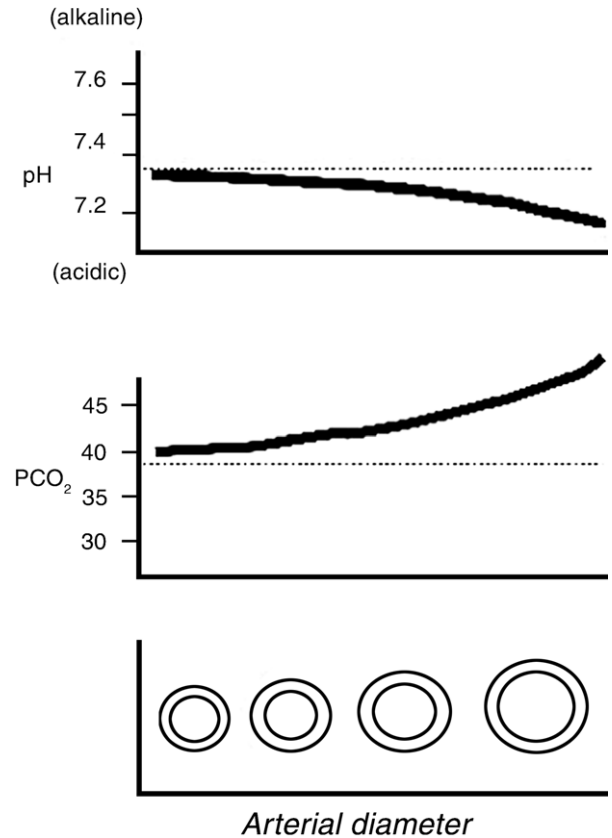


Figure 2. Schematic relationship between pCO₂, pH, and blood vessel diameter (breath-holding accumulates CO₂).

hypoxia in the cortex and actually reduces brain size. (With closed-head injuries, stroke, and hemorrhage, if the brain swells and is pressing against the skull, controlled hyperventilation is often a recommended short-term remedy to protect against damage.)

Chronic Hyperventilation

If overbreathing continues for several hours, the low CO₂ initiates physiological adjustments to compensate. Because hyperventilation is associated with alkalinity, the kidneys start retaining acid and more of the blood’s bicarbonate (alkaline) buffer is excreted. Although this does return the pH toward normal, it creates a false, fragile equilibrium that depends on continued hyperventilation. This new “set point” makes recovering a normal breathing pattern difficult. This person will be oversensitive to both exercise and relaxation and may suffer anxiety, chest tightness, fatigue, and muscle pain.

A “compensated hyperventilator” will often feel out of breath; because much of the bicarbonate buffer is gone, there is less of a defense against the rising acidity associated with retaining CO₂. Normal breathing will feel insufficient. Thus breath-holding time is typically shorter than average—often below 15 seconds.

To understand all of this better, try it yourself. (Caution: The hyperventilation provocation test carries a slight degree of risk because the resulting vasoconstriction affects the coronary arteries as well as other blood vessels. If there is a risk of heart attack or stroke, suggesting vigorous hyperventilation is not a good idea. Hyperventilation can provoke anginal pain, indicating cardiac insufficiency. Also, neurologists use hyperventilation to bring out abnormalities in the electroencephalogram (EEG), which may signal seizure susceptibility, so a known seizure risk makes hyperventilation unwise.) The following steps outline the hyperventilation provocation test:

Table. Relationships between blood pH and pCO₂, with symptom effects. Cerebral circulation drops 2% for every 1 mm drop in CO₂.

pH	PCO ₂ (in torr)	Effects
<i>Alkaline</i>		
7.8+	0	Death
7.7	10	Coma
7.6	20	Strong hypocapnia symptoms
7.5	30	Mild hypocapnia symptoms
7.4	40	Normal
7.3	50	Breathing reflex stimulated
7.2	60–70	Strong hypercapnic/hypoxic symptoms (drowsiness, confusion)
7.1	90	Coma, death
<i>Acidic</i>		

- Breathe deeply and quickly for 1–2 minutes. Chances are you will feel light-headed, unsteady, tingling in various places, tight-chested, and not overly bright, as well as other symptoms listed above. Imagine what is happening in your cerebral blood vessels according to Figure 1. During this experiment, if you try a task such as mental calculation, observe how your thinking gets fuzzy and slow.
- Restrict your breathing, or just hold it, while viewing Figure 2. You will pass through a window of feeling normal and then air hunger will start building up. Imagine what is happening in your brain as it tries to cope with the build-up of CO₂ and the waning of oxygen. Consider the Table and be relieved that you can adjust your position on the scale at will.

Why Does Hyperventilation Occur?

Whether episodic or habitual, hyperventilation might be explained by the concept of “action projection.” Getting advance warning of a challenge, threat, or physical exertion gives the animal (or person) more time to prepare physically. Besides stimulating sympathetic activity, overbreathing readies the body for running or fighting by storing up oxygen and reducing blood CO₂, which helps tolerate the surge of CO₂ resulting from muscle exertion.

In moderation, this works fine. Think of the deep breath you take before lifting a heavy box. That brief hyperventilation is quickly offset by extra muscle contraction, which keeps CO₂ stable. However, if you kept preparing to lift that heavy box but actually did

not, your CO₂ would drop and you would eventually feel symptoms. In general, our imagination can activate this preparatory state that, if not followed by action, leaves us chemically unbalanced and in a state of respiratory alkalosis. It is as if our car’s engine were to go in and out of tune depending on our thoughts and feelings.

In cases less easily explainable by action projection, perhaps incipient crying applies. Heavy, erratic breathing often occurs just before an individual bursts into tears. A strong emotional state may stimulate sighs and gasps; trauma is often associated with hyperventilation as well as crying. In fact, a recent study (Nixon & Bryant, 2005) studied this by having subjects hyperventilate (3 minutes of breathing at 30 breaths/minute). Subjects with Acute Stress Disorder reported increased incidence of trauma-related memories and flashbacks immediately after.

Measurement

Arterial CO₂ can be measured via blood sample, but a capnometer estimates the blood content well using only the exhaled breath sampled at the nostril. Without such a measurement it is more difficult to detect chronic hyperventilation because once a deficiency in CO₂ has been established, it can be maintained by only a small increase in respiration rate, volume, or both. Clues about the presence of chronic hyperventilation would include rapid or erratic breathing with frequent sighs; dyspnea; overuse of chest during breathing; frequent light-headedness and “spaciness”; breath-holding time less than 30 seconds; intermittent tingling in fingers, toes, or lips; and muscle weakness.

The Nijmegen Hyperventilation Questionnaire is a 16-item symptom list specific to hyperventilation syndrome that inquires about the frequency of symptoms such as shortness of breath, dyspnea, tingling around the mouth or fingers, and cold extremities. It is probably the best single questionnaire for the purpose because the unique combination of symptoms correlates well with clinical diagnostic criteria (van Dixhoorn & Duivenvoorden, 1985).

Individual Variation

Since arterial CO₂ has a fairly narrow normal range, reduced values from this range would seem to predict stronger symptoms. It does predict degree of alkalosis, but individuals vary in how much vasoconstriction results from a given degree of hypocapnia. A study of subjects with panic symptoms (Gibbs, 1992) used Doppler moni-

toring to assess basilar artery constriction for a given drop in CO₂ (induced by intentional hyperventilation). In some individuals, blood vessel diameter fell by 50%; in others, much less. Those with the strongest constriction generally reported more symptoms of panic.

Cortical Effects

Hyperventilation restricts circulation more in the cortex than in the lower brain levels and can seriously impair cortical functioning, disrupting judgment, perception, memory, orientation, and reaction time, and slowing EEG frequencies. Visual impairment may appear as spots or tunnel vision. General cognitive performance can suffer; Van Diest, Stegen, Van de Woestijne, Schippers, and Van den Bergh (2000) found clear deficits in subjects on a visual attention task immediately after 3 minutes of hyperventilation. Ley and Yelich (1998) studied junior high school students for test anxiety in relation to measured CO₂ and breathing rate; the students were monitored while taking achievement tests and showed correlations among lowered CO₂, respiratory rate, reported test anxiety, and the Nijmegen Hyperventilation Questionnaire scores.

Implications for Breathing Guidance

Most people believe that taking a deep breath creates a calming effect. Taking one or two deep breaths seems to re-establish the primacy of self-control. It can be overdone, however. “Deep breathing” has two potential meanings: large volume, and directing the inhalation down below the chest. People usually latch onto the first meaning and so they fill their lungs more completely, but if this is not balanced by slower breathing, then hyperventilation—alkalosis—is the inevitable result. A racing, anxious mind can easily contaminate the breathing process, speeding up breathing and turning an attempt at self-control into hyperventilation.

The instruction “take a deep breath” is better than “breathe deeply” because the second is not time-limited, and if done too quickly, can promote hyperventilation, so that the individual now associates relaxation with lightheadedness or ominous tingling. Monitoring the effects of a few deep breaths with a capnometer reveals how drastically end-tidal CO₂ drops; it can take several minutes for the blood chemistry to recover. Encouraging slow exhalation helps, because it buys time and allows CO₂ to build up before the next inhalation. “Breathe deep down, but slowly” is good advice, as is the simple instruction to breathe “low and slow” in the Bradley,

Gracewood, & Henley (2001) book *Self-Help for Hyperventilation Syndrome*.

People with panic disorder have a high incidence of hyperventilation, but often they do not know that their breathing is creating or worsening some of their symptoms. A hyperventilation provocation test can convince them that these disturbing sensations are lessened simply by breathing control. Excess sighing and deep breaths are markers for panic disorder (Wilhelm, Trabert, & Roth, 2001). Cause and effect are hard to distinguish, but breathing and thinking interact in a tangled psychosomatic way. For such a person, deep but urgent breathing may be an attempt to overcome a tight diaphragm or other muscular rigidity, so emphasizing muscle relaxation and gentler breathing might be more helpful than recommending deep breathing. Conversely, when an anxiety-prone individual senses somatic changes caused by hyperventilation, he or she may attribute these sensations to the current context (for example, being in a confined space). This anxiety may then aggravate the breathing imbalance and magnify the physical symptoms in a vicious mind-body circle.

With breathing, remember these key points:

- When breathing exceeds the body’s needs (hyperventilating), the problem is that the body’s CO₂ is depleted. Excess oxygen is exhaled unused, without problems.
- Although CO₂ is a waste gas, it regulates pH and blood vessel tone while in the bloodstream on the way out of the body.
- Muscle activity increases CO₂ production and helps to counterbalance hyperventilation.
- Insufficient breathing (hypoventilation) raises CO₂ (more is produced than exhaled).
- When CO₂ is below normal, blood vessels constrict and blood flow is restricted (especially relevant for coronary and cerebral arteries).
- When CO₂ production equals CO₂ excretion, blood vessels stay at their normal diameter and pH remains where it should.
- Changes in blood pH follow CO₂ level; more CO₂ = more acid, less CO₂ = more alkalinity.

References

Bradley, D., Gracewood, G., & Henley J. (2001). *Self-help for hyperventilation syndrome* (3rd ed.). San Francisco: Hunter House.

- Gibbs, D. M. (1992). Hyperventilation-induced cerebral ischemia in panic disorder and effects of nimodipine. *American Journal of Psychiatry*, *149*, 1589–1591.
- Ley, R., & Yelich, G. (1998). Fractional end-tidal CO₂ as an index of the effect of stress on math performance and verbal memory in test-anxious adolescents. *Biological Psychology*, *49*, 83–94.
- Nixon, R., & Bryant, R. (2005). Induced arousal and re-experiencing in acute stress disorder. *Journal of Anxiety Disorders*, *19*, 587–594.
- Van Diest, I., Stegen, K., Van de Woestijne, K. P., Schippers, N., & Van den Bergh, O. (2000). Hyperventilation and attention: Effects of hypocapnia on performance in a Stroop task. *Biological Psychology*, *53*, 233–252.
- Van Dixhoorn, J., & Duivenvoorden, H. J. (1985). Efficacy of Nijmegen questionnaire in recognition of the hyperventilation syndrome. *Journal of Psychosomatic Research*, *29*, 199–206.
- Wilhelm, F. H., Trabert, W., & Roth, W. T. (2001). Characteristics of sighing in panic disorder. *Biological Psychiatry*, *49*, 606–614.

Additional Reading

- Chaitow, L., Bradley, D., & Gilbert, C. (2002). *Multidisciplinary approaches to breathing pattern disorders*. Edinburgh: Churchill Livingstone.
- Fried, R. (1993). *The psychology and physiology of breathing*. New York: Plenum.
- Taylor, A., Rehder, K., Hyatt, R., & Parker, J. (1989). *Clinical respiratory physiology*. Philadelphia: W. B. Saunders.



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