Medical

Hyperventilation and the body

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Hyperventilation has rapid and far-ranging physiological effects via its alteration of pH and depletion of CO_2 in the body, resulting in respiratory alkalosis, acute or chronic. The general effects on skeletal and smooth muscles, as well as neural tissue, are summarized. A wide variety of symptoms such as pain, tension, disturbances of consciousness, circulatory problems and cardiovascular effects can confound treatment efforts but may respond to alterations of breathing patterns. Suggestions are given for detecting this condition.

Introduction

Imagine the following set of symptoms reported to you: episodes of light-headedness accompanied by cool, moist hands, pale skin, perhaps some tunnel vision, chest pain, headaches, feelings of unreality, dread and agitation; heart palpitations, uncomfortable breathing and tender shoulder muscles. Suppose that medical consultation has found little of note, but at least there is no evidence of major disease.

Where would you start? A break for lunch, perhaps?

In working with such a diffuse set of complaints spread out over many systems, a practitioner will naturally tend to focus on the symptom that is most familiar and easiest to handle, and may postpone dealing with the rest of the picture in the hopes that perhaps everything will clear up in time, or that someone else will handle the other problems. You may receive a referral because you hold out promise of helping with the muscle pain or the headaches. But other things are probably happening as well.

The problem, in the example above, may actually be an intermittent biochemical derangement brought on by insufficient carbon dioxide (CO_2) in the blood, resulting in broad effects on every organ and nearly every bodily system. Even though there is no clear indication of overbreathing at that moment, it may be correct to consider hyperventilation.

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Background

The 'Fat folder' syndrome is one vivid description attached by Claude Lum to the so-called hyperventilation syndrome, indicating the tortuous, and tortured, doctor-to-doctor path followed by many individuals pursuing relief from their problems. Each specialist performs his tests, finds little that is specifically diagnostic, and dutifully relays the individual on to the next specialist because typically, with this disorder, symptoms come and go in many different systems.

Hyperventilation simply means breathing in excess of the body's needs for oxygen *at a particular moment*. The need fluctuates continuously, and the body gets what it needs by adjusting respiratory rate and depth. Strong, vigorous breathing, no matter how fast or deep, is not necessarily hyperventilatory unless it is in excess of the body's needs.

The problem in hyperventilation is not 'too much oxygen' – whatever is unabsorbed by the lungs is simply exhaled again. The problem comes with a deficit of CO_2. CO_2 is a waste gas, a byproduct of metabolism, primarily muscle activity; it is given off by tissues and carried in the bloodstream to the lungs, to be exhaled. Production of CO_2 is normally in equilibrium with O_2 intake.

This equilibrium is important because CO_2 has an essential physiological function, remarkable

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considering that it's on its way to being excreted: it is a prime regulator of vascular tone in the cerebral cortex, among other places. This is Nature's elegant solution: in a natural environment and assuming normal physiology, CO_2 builds up in the bloodstream only with breath-holding or while breathing oxygen-poor air. In either case, high blood CO_2 correlates with less oxygen available. Since oxygen deficit (hypoxia) is dangerous to the brain, several mechanisms combine in such a circumstance to maintain normal brain oxygenation, and vasodilation of blood vessels is one of them. This is probably due to alterations in calcium and/or phosphorus movement through the vessel walls. Arterial walls, when dilated, become more permeable to oxygen molecules.

The loophole

The opposite condition, however, is more relevant to this article. There seems to be a loophole, or flaw, in the system, possibly unique to humans: although high CO_2 stimulates vasodilatation and increased oxygenation, a drop in CO_2 stimulates vasoconstriction and in fact is said to be the 'most potent vasoconstrictive agent known' (Raichle & Plum 1972). This other half of a regulatory mechanism becomes rapidly dysfunctional as CO_2 concentration in the blood drops, and that is what happens when an individual breathes in excess of bodily needs for oxygen: more CO_2 is exhaled than is being replaced. The degree of circulatory loss in the cerebral cortex is around 2% for each 1 mmHg drop in CO_2 pressure (Gardner 1996) This loss is visible to an observer if the hyperventilating individual's cortex happens to be uncovered at the time; it has been described by neurosurgeon Wilder Penfield as a blanching of the surface of the brain.

Normal arterial carbon dioxide tension ($PaCO_2$) is around 40 mmHg. A level of 30 corresponds to pH of 7.5; this is the commonly-accepted point at which mild hypocapnic symptoms are likely to begin.Twenty mmHg is associated with a pH of 7.6, at which symptoms will appear in almost everyone.

Response to 'needs of the moment' may constitute one thing to the body but quite another thing to the mind, at least in human beings. For instance, an individual can be mentally not in the present moment but in some other moment more associated with frustration, anxiety, or anger. In such a case time and place are blurred as the memory or imagination stimulates the body to prepare for action: fight, flight, or tense-freeze. All these options require muscle contraction, which requires oxygen, so hyperventilation becomes a sensible precautionary preparation.

Apart from this reality-shifting effect, events actually occurring in the present moment may not really require any physical action: for instance, when one's hypercritical supervisor is coming to inspect your work and you are preparing for a confrontation that requires only words, not fists. Here there will be a discrepancy between the action-demands on the body and the mind's expectation of trouble. Apparently the possibility of threat of any kind, social or physical, reliably sets off physical preparation 'just in case'. Hyperventilation is part of this, more for some individuals than others. Also, there is evidence that the hyperventilation can easily be conditioned (Ley 1999) so that a stimulus associated with potential danger will trigger more breathing directly, just as Pavlov's dogs came to salivate to the sound of a bell.

Hyperventilation is functional as long as muscle use follows closely after; the resulting reserve of oxygen and loss of CO_2 permit a head start if danger should materialize. The regulatory mechanisms for breathing probably evolved without providing for the possibility that hyperventilation might occur at times other than preparing for action. When emotional arousal does not lead to physical action, it can leave the brain and body impaired by alkalosis because respiration is driven to a higher level than is required by the current situation. The muscles are not generating enough CO_2 to replace what is being exhaled; the organism is not active when the system expects it to be. Thus body and mind can live in two different worlds, and this odd mismatch can create a morass of symptoms and no end of distress (Table 1).

Importance of pH

The body takes pains to preserve a normal pH in the body, particularly in the bloodstream, extracellular fluids and cerebrospinal fluid. The body is optimized for a pH of 7.4, slightly above the neutral 7.0, toward the alkaline end. This

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Table 1 Notable acute effects of hyperventilation

Effects on smooth muscle
Generally, vasoconstriction and spasm
Gastrointestinal: tight throat, difficulty swallowing, intestinal cramps
Restricted circulation in cerebral cortex (leads to light-headedness, dizziness, visual disturbances)
Cold hands and feet
Impaired coronary artery blood flow
Effects on skeletal muscle
Weakness, fatigue, twitching
Heart muscle disturbance (palpitations, skipped beats, rapid pulse)
Vague shifting pains, especially around chest
Diaphragm weakness, impaired breathing
Tense or fatigued neck and shoulder muscles (from over use during thoracic breathing)
Effects on nervous system
Peripheral tingling, numbness
Hypersensitivity to lights and sounds
Faster reflexes
Enhanced sympathetic activity (more adrenaline)
Common psychological effects
Panic, fear of death
Restlessness, heightened vigilance
Time distortion, depersonalization, derealization
Misinterpretation of symptoms (catastrophic thinking, hypochondria)

supports endocrine, metabolic and other maintenance operations. Deviations in pH of a tenth of a point have noticeable consequences for the body, changing nerve conduction, the diameter of blood vessels and the contractility of smooth muscle.

We can easily disrupt this balance with 2 or 3 deep breaths. pH will rise from 7.4 to 7.5 or 7.6, and CO_2 will fall from a normal 40 to 30 or 25 in less than 30 seconds (Figs 1 & 2). The combination of high pH and low CO_2 causes certain body changes. Acute, episodic hyperventilation is often associated with an emotional crisis, either current or relived. In the stereotype, hyperventilation is seen as a panic 'attack' – dramatic and obvious, but ameliorated by letting time pass or by breathing into a paper bag. This is actually much less common than subtle, chronic hyperventilation. In this case, symptoms are milder but more persistent;

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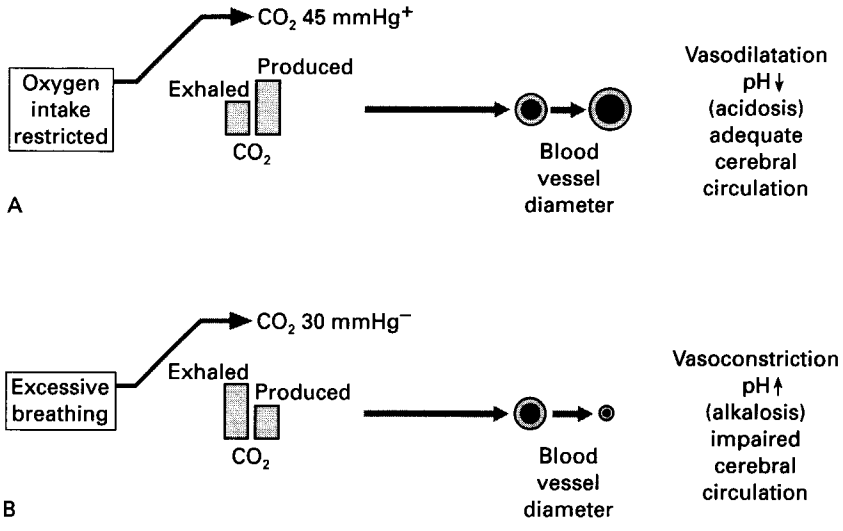

Fig. 1 CO_2 as a regulator of cerebrovascular tone. Breathing volume determines amount of CO_2 excreted per unit time; metabolic demand determines amount of CO_2 produced. With CO_2 production in equilibrium with CO_2 excretion, all is well; pH stays at 7.4 and CO_2 remains at 35–40 mmHg. However, (A) With restricted oxygen intake (breath-holding or high CO_2 air), more CO_2 is produced than is exhaled. CO_2 rises, stimulating blood-vessel dilatation to offset danger of suffocation. (B) With hyperventilation (breathing excessive for body's needs) more CO_2 is exhaled than is produced. CO_2 drops, stimulating blood-vessel constriction

breathing is less obviously 'off', and the body has started to compensate for the hyperventilation.

Effects on skeletal muscle

Respiratory alkalosis and low CO_2 affect muscle function in several ways. For one thing, the muscle potential threshold drops, making the individual more susceptible to twitches and spasms. Phosphorus tends to be lost during hyperventilation, and low serum phosphorus levels have been linked to skeletal muscle weakness and impaired nerve conduction in general. Contractility of the diaphragm may be disrupted, which would increase the difficulty in getting a deep breath and set off urgent attempts to get more air, resulting in a 'vicious circle'.

Claude Lum, a major figure in study of hyperventilation, describes a '... specific effect on facilitatory synapses involved in somatic motor reflex arcs – such as tendon reflexes – reflex time is shortened, and synaptic transmission is accelerated' (Lum 1981). Moderate hyperventilation causes motor excitability and hypertonicity, faster reflexes, and suppression of parasympathetic nervous system activity. Add to this the frequent occurrence of thoracic breathing at these times, associated with excessive use of the trapezius, sternomastoid and scalenes. Overworking these accessory breathing muscles can produce hypertonicity under normal

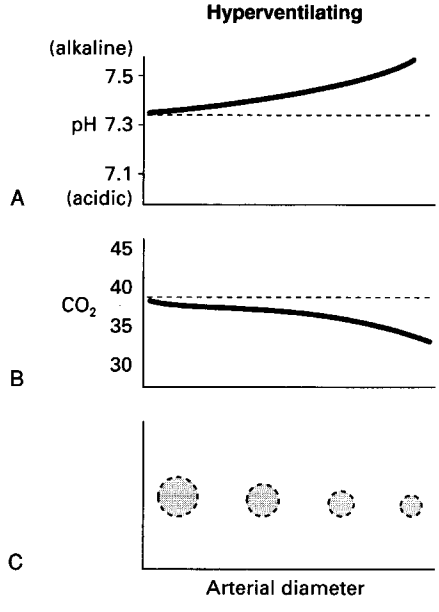

Fig. 2 'Hyperventilating:' as breathing increases beyond metabolic needs, CO_2 is depleted from the body and alkalinity rises. Smooth muscles, such as blood vessels, are more likely to constrict. (A) = pH; (B) = CO_2; (C) = arterial diameter

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conditions; if low CO_2 in addition is promoting weakness and hypersensitivity, it simply makes things worse. One study produced long-term reductions in non-cardiac chest pain mainly by training the patients to breathe more slowly and more abdominally, thereby taking the strain off of thoracic and neck muscles (DeGuire et al. 1992).

Thoracic breathing, by the way, seems to be the avenue of conscious influence, used when we wish to override the automatic regulation, whereas abdominal breathing is the default system. Most writers on hyperventilation have mentioned the prevalence of thoracic breathing, and Lum proposed that stress may be the provocative factor, while thoracic breathing constitutes the permissive factor (Lum 1976). It may be that voluntary use of the accessory breathing muscles begins as a response to suffocation fear, an attempt to improve breathing comfort, or a misguided preparation for exertion that never comes. This overcontrol can turn chronic, leading to dull, diffuse thoracic pain and intercostal tenderness.

Effects on the brain

Normal $PaCO_2$ concentration in the blood and in exhaled air is around 5%; this is associated with optimal blood flow in the cerebrum. The corresponding figure expressed in mmHg is around 40. Below 30 is a commonly accepted point below which symptoms are more likely, and there is usually serious interference with cerebral blood supply. Positron emission tomography (PET) and magnetic resonance imaging (MRI) scans have shown that cerebral blood flow correlates area by area with mental activity of the moment: for example, recalling a face requires more blood flow in the right hemisphere, while recalling a poem does the same in the left hemisphere. Hyperventilation will certainly affect this delicate allotment of resources and disrupt stability of consciousness in a global way as well.

With vasoconstriction (ischaemic hypoxia) attention and short-term memory are impaired; vision may be diminished and light-headedness may occur. The dominant electro-encephalogram (EEG) frequency actually drops, correlated with less efficient cortical processing. Hyperventilation destabilizes the brain and can easily precipitate seizures in susceptible people. Neurologists use hyperventilation routinely to 'push' the brain and bring out abnormalities which may indicate seizure potential.

Research with military pilots during the Second World War and later showed that hyperventilation impaired problem-solving and other performance tasks relevant to flying. Motor coordination and dexterity, balance, cognitive and perceptual tasks have all been shown to suffer with cerebral hypoxia induced by hyperventilation (Wyke 1963). Clinically, an individual in this condition will often complain of a 'spacey' feeling: impaired thinking, things or the self not feeling real, poor definition of body boundaries, etc. In fact, explanations and instructions about how to 'breathe better' may not sink in very well at that moment because the cerebral impairment will interfere with learning.

Apart from cerebral vasoconstriction, the general tendency of hyperventilation to promote smooth-muscle constriction can affect the gastrointestinal tract, peripheral and skin circulation ('cool hands, cold feet'), coronary arteries and the bronchioles. These transient, reversible effects can simulate more drastic ailments, and in some cases can magnify pre-existing problems such as heart disease, Raynaud's disease and spastic colitis.

Chronic hyperventilation

Acute hyperventilation provides a foundation for understanding, but I want to bring attention to the other, more common type: subtle and chronic, chemically more complex because the body is trying to adjust to it. This is an entity in itself and differs from acute hyperventilation in several ways, not the least being the difficulty detecting it.

Recall the assertion that the body strives to keep the pH where it should be. In the short run, hyperventilation will raise the pH toward alkalosis, directly affecting circulation, muscle tension, smooth muscle, nerve conduction, sensory functions, cerebral function, etc. But there are safeguards to prevent this from going on too long. If hyperventilation persists for a few hours, the body begins to compensate, primarily by changing the proportion of bicarbonate ions in the blood. This is CO_2 in an alkaline, transportable form, and changing its concentration permits fine tuning of blood pH. Since hyperventilation is causing alkalosis

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(higher pH than normal) the kidneys begin to correct the imbalance by excreting more bicarbonate, thus retaining acid. The pH will return to normal, more or less, but the CO_2 remains low, leaving the individual in a precarious state (Table 2).

A parallel in principle to this bodily adjustment might be a postural defect which reflects a psychological attitude of depression, anxiety, or defensiveness; while not optimal structurally, the stance amounts to a compromise between emotional input and body regulatory mechanisms. Obesity (or skinniness) is also similar, in that the body is forced to accept and adapt to inadequate or excessive amounts of food consumed. We cannot assume that, because a process seems deeply automatic, it is strictly physiological and immune to tampering from the higher aspects of an individual. Emotions and habitual behaviour can intrude anywhere.

Living on the edge

Many individuals seem to live on the edge of symptoms of hypocapnia. A CO_2-deficient state can be maintained by an occasional sigh or deep breath, or by breathing only 10% above normal ventilation (Gardner 1996). In these chronic cases pH may be near normal because of the renal compensation, but the CO_2 remains low. Because of this, the chronic hyperventilator inhabits a narrow zone of comfort with a lower threshold for development of symptoms (Magarian 1982). A slight increase in breathing without accompanying muscle contraction may set off symptoms of hypocapnia. A slight increase in acidity from breath-holding or sudden exercise can set off 'air hunger' because the kidneys have dumped a proportion of the alkaline reserves (bicarbonate) in order to balance the excess alkalinity (Fig. 3). So poor exercise tolerance can be yet another symptom of this syndrome.

Table 2 Signs and symptoms of chronic hyperventilation

Respiration: primarily thoracic, over 18 per minute, irregular (sighing, holding, labile), mouth-breathing, dyspnoea
Cold hands, indigestion
Chest pain, tightness
Poor sleep, nocturnal panic attacks
Over-sensitive to both exercise and relaxation
Easily exhausted with effort
Anxiety, restlessness, fidgeting
Muscle pain and sensitivity
Lowered serum bicarbonate and $PaCO_2$, but blood pH normal
Chronic hyperventilation mechanisms
Long-term physiological adjustment (within hours) to respiratory alkalosis:
Bicarbonate buffer (alkaline) is excreted in order to reduce alkalosis and restore pH toward normal. Kidneys retain acid to compensate for loss of carbonic acid from the blood.
'False equilibrium': bicarbonate buffer depletion creates a fragile, somewhat stable biochemical state which depends on continued hyperventilation.
Symptoms are easily provoked by changes in breathing in either direction, because of a narrowed range between critical 'alarm' values for acidosis and alkalosis.

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Some researchers think that abrupt changes and drops in CO_2 level are more significant than the actual level itself: that is, erratic breathing, like an abrupt, jerky driver on the highway whom everyone avoids, may be harder on the body than slow, smooth changes (Lum 1987). The body may more easily adapt to steady hypocapnia than to an emotion-driven alternation between breath-holding and sighing.

Anxiety

The relation between anxiety and hyperventilation is bi-directional, and moderated by individual differences. The proposition that hyperventilation is the ultimate cause of panic attacks is not as tenable now as it once seemed, but the formulation goes as follows:

> Hyperventilation lowers CO_2, and the combination of alkalosis and hypocapnia cause a flurry of symptoms which alarm the individual, stimulating more hyperventilation when an emergency situation is perceived. CO_2 drops further; this worsens the symptoms, and so forth, in a classic vicious circle.

This does seem true in some cases of panic, but many others do not fit. An individual can report panicky feelings with normal CO_2, and CO_2 can be low with no panic at all. Some individuals can hyperventilate voluntarily to reach very low levels of $PaCO_2$ yet feel few symptoms, probably because their systems are better able to resist the effects or compensate.

At least three factors seem to maximize this potential hyperventilation/anxiety connection. First, a tendency to interpret the sensations as dangerous (heart attack, brain tumour, fainting, etc.) will magnify symptoms and amplify the 'resonance' between mind and body in a negative way. This cognitive view has led to psychologically-based treatment of panic (Salkovskis & Clark 1990). Second is the likelihood of breathing serving as a habitual expression of strong feelings, often conflict over anger, frustration, death-fears, or recalled grief (Conway 1994). Third is a physiological susceptibility to hyperventilation and its consequences. This may take the form of greater smooth-muscle contractility to a given amount of hypocapnia (Gibbs 1992) or a central sensitivity to CO_2. A 'suffocation alarm' has been proposed by Klein (1993) as an intervening variable affecting

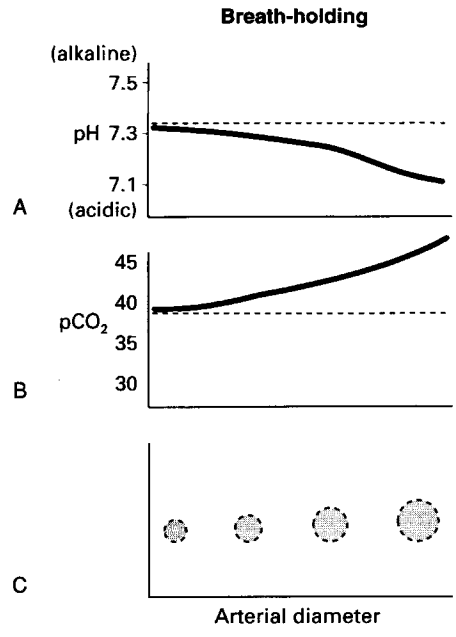

Fig. 3 'Breath-holding:' as breath is retained, CO_2 rises. Being acidic, it promotes a drop in pH, which is associated with blood vessel vasodilatation to facilitate oxygen diffusion. (A) = pH; (B) = CO_2; (C) = arterial diameter

CO_2 sensitivity. Also, blood glucose seems to protect against the effects of lowered CO_2. The symptomatic response to hyperventilation with glucose at 70–75 mg% is much greater than when it is around 100 mg, even though the former is within the normal range for fasting blood sugar (Engel Ferris & Logan 1947).

It seems that even the clearly physiological aspects of hypocapnia and alkalosis can be mediated by mental factors. Some individuals have a drastic drop in CO_2 during hyperventilation and have abundant symptoms, but they do not worry about it and may even bring the condition on for recreation. When hyperventilation is provoked during laboratory studies, panic patients often do not panic as expected because the context is different. Former panic patients who have gone through a successful course of therapy lose their fear of the symptoms. Typically they are trained in breathing regulation and also in reinterpreting the symptoms as transient, understandable and harmless, rather than catastrophic.

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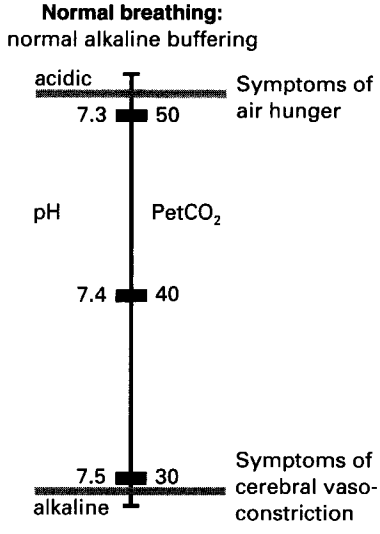

Fig. 4 When hyperventilation continues for several hours or more, there is a drive to restore normal pH by excreting more alkaline buffer. This compensation permits CO_2 at a level of 35 mmHg to co-exist with 7.4 pH. Such an individual becomes subject to reduced capacity to tolerate acidosis from any source, including breath-holding, and is also closer to the 'symptom-line' for reduced CO_2. Note the two horizontal lines closer together in the right-hand diagram

Diagnosis

The chemical imbalance described here has a number of possible causes, some so clearly organic that it is generally prudent to get medical clearance before attempting to alter an individual's breathing style in any detailed way.

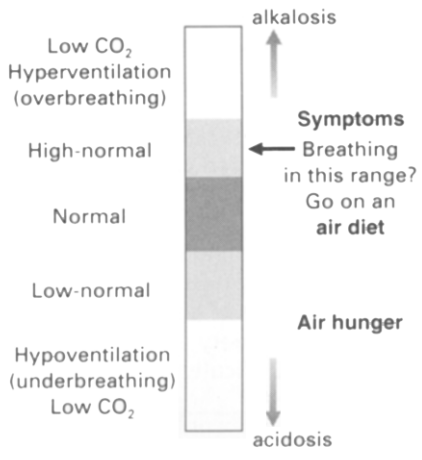

Fig. 5 Chronic hyperventilation can be compared to overeating; perhaps for fear of feeling breathless, people may overbreathe, gulping oxygen but depleting their systems of carbon dioxide and provoking symptoms associated with alkalosis

When hyperventilation does occur at times other than during muscular activity, it may be a necessary compensation for a condition that would worsen and be possibly life-threatening if hyperventilation were not occurring. It could be responding to diabetic acidosis, for example, or salicylate poisoning, which acidifies the blood; asthma and emphysema can induce hyperventilation; also pulmonary embolism or hypertension, liver failure and anaemia.

The possibility of seeing someone unaware of their relevant medical condition is small, but still present; people vary in their readiness to consult medical practitioners. Diagnosis of the so-called 'hyperventilation syndrome' should be a medical decision; possible organic causes must be considered. See Gardner (1996) and Fried (1993) for further discussion.

Having said that, here are some tips:

Tests

The clearest indication that an individual suffers from chronic hyperventilation is probably the combination of lowered CO_2 and near-normal blood pH, accompanied by habitual thoracic breathing. $PaCO_2$ and pH can be sampled from arterial blood, but this is invasive and represents a brief moment in time. If the patient happens to hold his breath during the puncture, the value may be misleadingly normal.

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Box 1 Indicators of hyperventilation representative items from the Nijmegen Questionnaire

Chest tightness
Blurred vision
Shortness of breath
Heart palpitations
Tingling in fingers
Dizziness
Loss of contact with surroundings
Anxiety
Difficulty taking a deep breath

End-tidal CO_2 can be measured easily from the exhaled air via a small sampling tube leading to a capnometer, which analyses each breath for CO_2 content. In the absence of lung disease, this correlates closely with arterial CO_2. Measurements can be obtained over long time intervals. However, capnometers are not standard equipment except in operating rooms or the office of a pulmonologist. They are quite useful for corrective breathing training and for feedback, however, and some respiratory therapists and physiotherapists use them routinely. Though their cost when new can be US $3000–$5000, used units are often available from hospitals or medical sources as new models are updated (Gilbert 1994).

Even if an individual reports symptoms suggestive of hyperventilation, it does not mean the condition is always present. Symptoms can come and go, and between times blood gas and end-tidal CO_2 values may be normal. Many people occupy a category midway between 'acute' and 'chronic'.

Several other tests have been found useful for diagnosis:

1. The Nijmegen Test (van Doorn et al. 1982) consists of 16 prime symptoms most commonly reported by people who by other evidence tend to hyperventilate (for example, 'Unable to breathe deeply' and 'Blurred, hazy vision'. It is quickly administered, allows for ratings of frequency, and can serve as a good basis for further investigation (see Box 1).
2. Recovery after hyperventilating (Hardonk & Beumer 1979): while being monitored via capnometry, the individual is asked to hyperventilate for 3 minutes to lower the CO_2, and then resume normal breathing. Patients with symptoms of hyperventilation are more likely to show a delayed return to normal CO_2 level. The best-discriminating ratio settled on by the authors was 1:5 to 1:CO_2 at baseline/CO_2 at 3 minutes after terminating hyperventilation. So if CO_2 originally at 40 does not recover to 27 after 3 minutes, that person would be predicted to have trouble with overbreathing. King (1988) showed that hyperventilators are less aware than others of when their CO_2 is low. After a hyperventilation test, they would signal that their breathing felt normal again when CO_2 was still in the subnormal range. This apparently altered set point may reflect habituation to a chronic imbalance or else 'loose standards' for some other reason.
3. Peter Nixon's 'Think test' (Nixon & Freeman 1987) involves exhorting the individual, while being monitored by capnometry, to dwell on a disturbing emotional event or situation. If the breathing becomes disordered and the CO_2 clearly drops below baseline, this is considered evidence of a general tendency to hyperventilate during emotional states. This loosely-defined test takes some skill to perform, but can be informative. Peter Nixon is a London cardiologist who has demonstrated the medical (especially cardiovascular) ramifications of chronic hyperventilation (Nixon 1989).
4. 'Effort syndrome': this is Nixon's delineation of how hyperventilation-related deficiency in alkaline buffering predisposes an individual to premature muscle fatigue, as shown by lowering of the anaerobic threshold (Nixon 1994). This is tested with exercising on an ergometer while CO_2 and work output are measured. An abrupt downturn of CO_2 indicates this threshold and the onset of hyperventilation associated with appearance of metabolic acidosis.
5. The 'hyperventilation provocation test' is widely done by physicians and others as a quick test for reproducing some of the symptoms. If the individual sees that the same symptoms that have driven him from office to office can be produced in less than 3 minutes of heavy breathing, he is likely to learn a valuable lesson. It is frequently reassuring to discover that the symptoms can be brought under voluntary control simply by terminating the hyperventilation.

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There is controversy about the safety of provoking hyperventilation for test purposes. The procedure does put strain on the heart and blood vessels, causing transient vasoconstriction, ischaemia and often disturbance of the heart rhythm. In the presence of heart disease or cerebrovascular abnormalities, the risk increases to the point that it would be wise to have electrocardiogram (ECG) monitoring and resuscitation equipment nearby if the test is done at all.

Other suggestive signs and symptoms include:

1. prominent thoracic breathing
2. frequent sighing or gasping
3. shortened breath-holding time
4. rapid or irregular breathing
5. mouth-breathing
6. reporting of some of the hyperventilation indicators such as:
   a. light-headedness
   b. tingling or numb extremities
   c. muscle weakness or twitching
   d. disturbance of consciousness or of vision
   e. breathlessness
   f. rapid heart rate
   g. cool, pale skin, etc.

None of these is truly specific to hyperventilation; they often accompany anxiety states. Together, however, the combination of visibly altered respiratory pattern with subjective reports as above should raise the question of possible alkalosis.

There is room for improvement in diagnosing a condition that varies so much in its expression. Yet some reading on the topic will give the reader a sense of how hyperventilation can sometimes disrupt the body's biochemical regulation in a major way, leading to quite florid symptoms. The distinction between 'acute' and 'chronic' is important; a panic attack with hyperventilation stands out clearly, but when the overbreathing becomes habitual for whatever reason, a murky, incomplete adjustment develops which is more difficult to reverse, and calls for much more than a paper bag or instructions to breathe more slowly.

Conclusion

To sum up, hyperventilation can affect many aspects of the muscular system, one's bodily perception, equilibrium, sensory functions and all smooth-muscle function, including circulation. If therapeutic bodywork is not proceeding well and some of the complaints mentioned are present, it may help to look deeper into the possibility of hyperventilation even though the breathing may not be noticeably different.

Also, it may be fruitful to investigate the circumstances under which the symptoms worsen. As you might inquire about the context of muscular or pain symptoms, ask as well about any associated emotional issues, social or psychological events preceding or following a flare-up, the habitual state of emotional tension, etc. Sometimes even talking about these things will exacerbate symptoms through a barely detectable increase in ventilation, which may still be adequate to drop CO_2 to a symptomatic level.

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