

Hyperventilation Syndrome: A Diagnosis Begging for Recognition

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Topics in Primary Care Medicine

Diagram Omitted. Please refer to source for complete article.

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Beginning with the American Civil War, military physicians seeing soldiers under the stress of combat have described a syndrome characterized by breathlessness, lightheadedness or dizziness, pronounced fatigue and exercise intolerance, numbness and paresthesias and chest pain. Rarely have organic diseases been found to account for the symptoms in such cases, yet despite reassurance, symptoms commonly persist for prolonged periods despite removal from the apparent stress setting. This syndrome has been given many names including irritable heart, soldier's heart, Da Costa's syndrome, effort syndrome, neurocirculatory asthenia and, more recently, hyperventilation syndrome.

Since the original descriptions in soldiers, it is now recognized that hyperventilation occurs in many persons under stresses of daily living. It is manifest not only in those overtly stressed, anxious and depressed but also in those who appear outwardly calm as they "bottle up" their feelings, often because of undeveloped or lack of acceptable emotional outlets. Physicians and lay persons alike readily recognize acute hyperventilatory attacks occurring under acute stress. However, chronic or recurrent hyperventilation problems often are unrecognized probably for a variety of reasons, including the frequent lack of obvious overbreathing, a tendency to focus on one or two complaints that alone are not particularly suggestive of hyperventilation, minimal discussion of the topic in medical school and cursory coverage in medical textbooks.

Physiology of Hyperventilation

Although precise delineation of the relationship between physiologic responses and symptoms of hyperventilation is lacking, an understanding of known physiologic mechanisms does provide

insight (Table 1). Hypocapnea and respiratory alkalosis develop rapidly upon onset of hyperventilation and can easily be maintained indefinitely, by nearly imperceptible hyperventilation, such as by taking an occasional deep breath while maintaining a normal respiratory rate. Without knowing this, physicians may directly observe the subtle, chronic form of hyperventilation without recognizing it or, upon considering the diagnosis, inappropriately reject it because the anticipated hyperventilatory respiratory pattern is not present.

TABLE 1.--Physiologic Responses Associated With Hyperventilation

Hypocapnic, respiratory alkalosis
Hyperadrenergic state
Increased oxygen binding to hemoglobin (Bohr effect)
Hypophosphatemia
Initial vasodilatory, later vasoconstrictive cardiovascular responses
Reduced cerebral perfusion Possible coronary vasospasm

Stress is often associated with a hyperadrenergic state that is known to provoke hyperventilatory responses in humans. Beta-blocking drugs may reduce not only stress levels but also ventilatory responses to catecholamine stimulation and have recently been shown to improve performance levels in stressful situations.

Respiratory alkalosis increases the avidity of oxygen binding to hemoglobin such that oxygen becomes less readily released to tissues (the Bohr effect). Hypophosphatemia develops rapidly and persists for the duration of respiratory alkalosis, probably related to intracellular shifts of phosphorus. With persistent hyperventilation, hypophosphatemia would impair generation of 2,3-diphosphoglycerate (2,3- DPG), further reducing oxygen availability for tissue utilization.

It is estimated that a 2 percent reduction in cerebral blood flow occurs for every decline of 1 mm of mercury in arterial carbon dioxide tension. This, along with the Bohr effect, leads to reduced cerebral oxygenation. Cerebral hypoxia, however, produces a vasodilatory response that may compensate for the initial reduction in cerebral perfusion.

Cardiovascular responses are variable and seem to be in large part related to the duration of hyperventilation. The initial response is a reduction in systemic vascular resistance and blood pressure with an increase in heart rate and cardiac output. Within four to seven minutes of sustained hyperventilation, however, this response diminishes or disappears.

Finally, several investigators have shown coronary vasoconstriction induced by hyperventilation in some patients with Prinzmetal's angina and others with fixed coronary occlusive disease.

Pathogenesis

How does the hyperventilation syndrome develop? Although hyperventilation may have organic or physiologic causes, the syndrome of hyperventilation is usually associated with emotional triggers and thoracic breathing tendency. Indeed, many persons who are anxiety-laden, stressed

or depressed have hyperventilatory breathing patterns and complain of their inability to obtain satisfying deep breaths. Anxiety, anger and other emotions produce increases in both rate and depth of respirations probably mediated by a hyperadrenergic state. Once hyperventilation is initiated, persisting stresses of everyday living or the stresses of new bothersome symptoms from hyperventilation create the potential for a self-perpetuating cycle of chronic hyperventilation (Figure 1).

Persons who hyperventilate more commonly exhibit obsessional behavior, excessive body consciousness, phobias, feelings of inadequacy and maladjustments in many stages of life. Lum believes that an exaggerated tendency to breathe using thoracic musculature is an important factor allowing for the development and, once developed, the persistence of the hyperventilation syndrome.

Symptoms and Signs of Hyperventilation Syndrome

Among the most difficult and frustrating patients for physicians are those with multiple complaints involving many organ systems who, despite seeing numerous physicians, fail to obtain a satisfactory explanation or relief from their symptoms. They often have a "positive review of systems." After numerous physicians have been seen and multiple diagnostic tests have been done, which have excluded organic disorders, such patients are often dismissed as having nothing wrong with them or having a severe neurosis, anxiety, depression, hypochondriasis or hysteria, despite the persistence of symptoms that may be disabling in their work and other aspects of everyday living. Unfortunately, this scenario continues to be a common occurrence and is the frequent setting in which the hyperventilation syndrome is recognized, months or years after its onset. Previous studies have shown that 5 percent to 10 percent of patients seeking care from primary care physicians have at least some complaints related to hyperventilation.

TABLE 2.--Signs and Symptoms of Hyperventilation Syndrome

GENERAL Weakness, fatigue, sleep disturbances, blurred vision

PSYCHIATRIC Anxiety, depression, phobias, feeling far away, sensations of unreality

NEUROLOGIC Paresthesias in extremities or periorally, lightheadedness, dizziness, disorientation, impaired thinking, seizures, syncope, headaches

CARDIOLOGIC Palpitations, chest pain

RESPIRATORY Dyspnea often without provocation characterized as being unable to take a satisfying deep inspiration, exaggerated thoracic breathing, sighing, yawning

GASTROINTESTINAL Dry mouth, bloating, belching, flatulence

MUSCULAR Cramping, spasm, musculoskeletal chest wall pain (chest wall syndrome)

The hyperventilation syndrome may be associated with a myriad of symptoms (Table 2), affecting both men and women equally. The most frequent complaints for which medical attention is sought are lightheadedness or dizziness, dyspnea and chest pain. Substantial weakness, exercise intolerance, fatigue and peripheral or perioral numbness and tingling, occurring in isolation or in concert with other hyperventilatory symptoms, are almost always present. Many patients have multiple other complaints. When symptoms are taken in isolation, the syndrome is often not considered. However, when taken together, the entire symptom complex often makes the diagnosis rather obvious.

The dizziness of hyperventilation may be described as lightheadedness or an unsteady, giddy feeling, similar to drunkenness or vertigo. In one review of 104 patients who presented to a specialty clinic for the evaluation of dizziness, 23 percent had hyperventilation as the sole or prominent contributing factor. There may also be some degree of disorientation and mental impairment.

Breathlessness is a common complaint and is usually described as the inability to inhale a satisfyingly deep breath. It may be manifested by periodic, predominantly thoracic deep breaths, sighing and yawning. Sighing dyspnea is not a manifestation of cardiac failure. Although the hyperventilation syndrome rarely is associated with an obvious increase in respiratory rate, astute observers usually will note an increase in thoracic respiratory efforts. Paradoxically, whereas many people take deep breaths in an effort to relax, they may be provoking the very state they wish to avoid. The dyspnea of the syndrome may arise from fatigued respiratory muscles, overworked from chronic, excessive respiratory efforts. Since this type of dyspnea rarely occurs in the absence of other related symptoms, it is important that other manifestations of the hyperventilation syndrome be sought in all cases of otherwise unexplained dyspnea.

Gastrointestinal manifestations include dry mouth, bloating, belching and flatulence, related to aerophagia associated with overbreathing. Depression with attendant anorexia and weight loss may mimic systemic disease.

Cardiovascular symptoms of the syndrome are primarily palpitations and chest pain, which may mimic angina. Continuous ambulatory electrocardiographic monitoring of hyperventilators has shown frequent sinus tachycardia and supraventricular arrhythmias, even during sleep. Hyperventilatory symptoms without apparent provocation may occur during these times.

The chest pain of hyperventilation is variably described. It may be sharp and stabbing, thought to be related to pressure on the diaphragm from gastric distention or diaphragmatic hypertonicity related to a generalized hypertonic muscular contractile state. Other types of chest pain have features that may strongly suggest angina including location and radiation patterns. The pain may be described as dull, gnawing, burning or constricting and localized to the precordial or retrosternal area but is often rather diffuse and of greater duration than is typical of angina pectoris. It is not predictably associated with events that usually provoke angina, frequently occurring at rest or after exertion, and is not reliably relieved by nitroglycerin. Occasionally, "pseudoischemic" electrocardiographic patterns may be seen in patients with chest pain from hyperventilation. It currently remains uncertain whether hyperventilation-induced coronary

vasospasm and myocardial ischemia contribute to the chest pain associated with the hyperventilation syndrome. Unfortunately, a diagnosis of noncardiac chest pain, while initially gratifying, usually does not result in a significant reduction in outpatient clinic or emergency room visits as symptoms often persist. Therefore, in evaluating chest pain, the historical data base should include questions directed toward the possibility of hyperventilation lest the etiologic basis of the chest pain be dismissed as noncardiac, yet unrecognized as hyperventilatory.

Other symptoms of hyperventilation are usually present but rarely offered voluntarily. Apart from other disorders the patient may have, the physical examination is often normal. Patients often do not appear overtly anxious though they are frequently depressed. Obvious hyperventilation is usually lacking although occasional deep breaths, sighing or yawning and palpable chest wall tenderness may be noted. The diagnosis of chest wall syndrome requires exclusion of the hyperventilation syndrome which may be its basis.

It is critical to recognize that the presence of the syndrome does not exclude the presence of an organic disease. In fact, reaction to the symptoms of an organic disease may be a prime factor provoking hyperventilation.

Management of Hyperventilation Syndrome

As many patients with the syndrome have had symptoms for months or years and have seen other physicians without appreciating the cause of their symptoms, it is important that the patient be confronted with the cause-and-effect relationship between hyperventilation and their symptoms. A hyperventilatory trial is crucial for therapeutic success. This can be accomplished by having the patient breathe deeply at a rate of 30 to 40 times per minute. Most patients with the hyperventilation syndrome will recognize at least some of their symptoms within several minutes and often in seconds. This recognition and subsequent explanation of hyperventilation greatly enhances the potential for improvement. An explanation and reassurance without the patient actually experiencing the cause-and-effect relationship of overbreathing at the time is often without therapeutic benefit.

After provocation of symptoms .during a hyperventilatory trial, breathing into a lunch bag-sized brown paper bag will result in resolution of those symptoms that are directly related to hypocapnea. Dyspnea and chest pain, however, may persist in that they are not caused by hypocapnea, but more likely by the excessive use of thoracic musculature.

Because many patients have experienced substantial adverse effects on their employment and social interactions it is beneficial for a spouse or a friend to be present during a hyperventilation trial. Family and friends may be highly skeptical that something as simple as overbreathing can be having such devastating effects on the patient and indirectly upon them as well. Convincing both the patient and others provides support for the patient as he or she attempts to regain control.

Although some believe bag rebreathing is of little value, we have found it to be useful, allowing patients an escape from symptoms. Initially, we encourage patients to attempt bag rebreathing,

relax and get away from the situation that may have triggered the response. As a result, patients appreciate a newfound control. This greatly reduces the anxiety and stress that fuel the hyperventilation cycle.

Long-term control may be achieved by relaxation therapy and retraining patients to become diaphragmatic rather than thoracic breathers. Referral to behavior modification experts may be of value in particularly difficult patients with long-standing symptoms. In anxious and depressed persons with chronic hyperventilation we have rarely seen substantial benefit from the use of anxiolytic or antidepressant medications when the hyperventilatory component was unrecognized or being inadequately addressed. In conjunction with therapeutic measures directed toward the hyperventilatory tendency these drugs may be of additional benefit though we often find them unnecessary.

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