# THE HYPERVENTILATION SYNDROME

HADDON M. CARRYER

AN important medical problem, yet one which is often unrecognized, is the syndrome resulting from hyperventilation. Until recent years the mechanism of this disorder has been poorly understood. In 1933 and 1934 Maytum and Willius[11, 12] first called attention to sighing dyspnea, a common form of hyperventilation, in the production of symptoms frequently confused with those of pulmonary or heart disease. Other investigators have done much to clarify knowledge of the physiologic changes observed in the course of respiratory alkalosis caused by hyperventilation. Kerr and his associates at the University of California have been particularly interested in this problem. They found an early and interesting reference to this condition which bears quotation.

"Melancholoke folke are commonly giuen to sigh, because the minde being possessed with great varietie and store of foolish apparitions, doth not remember or suffer the partie to be at leisure to breathe according to the necessitie of nature, whereupon she is constrained at once to sup vp as much ayre, as otherwise would srue for two or three time; and this great draught of breath is called by name sighing, which as it were a reduplicating of the ordinary manner of breathing. In this order it falleth out with louers, and all those which are very busily occupied in some deep contemplation. Sillie fooles likewise which fall into wonder at the sight of any beautifull and goodly picture, are constrained to giue a great sigh, their will (which is the efficient cause of breathing) being altogether distracted, and wholly possessed with the sight of the image."[4]

## ETIOLOGY

The symptoms of the hyperventilation syndrome come about as the result of an increased loss from the body of carbon dioxide. This occurs from the lungs in the course of an excessive pulmonary ventilation. The usual concentration of carbon dioxide in the alveolar air is approximately 40 mm. partial pressure of mercury.[1] With unduly stimulated breathing this level of alveolar carbon dioxide may be reduced to approximately a half of that value. Thus a chain reaction is initiated which in turn affects the equilibrium existing between the carbon dioxide of the alveoli and of the blood, and subsequently that equilibrium existing between the carbon dioxide and the bicarbonate of the blood. An increased alkalinity is made temporarily present in

the blood as the so-called respiratory alkalosis is brought about. Numerous chemical changes occur in the body consequent to this respiratory alkalosis. Grant and Goldman recognized in 1920 that tetany could be induced by means of hyperventilation. This effect is mediated through the influence of respiratory alkalosis on the blood calcium. While no significant change in the total calcium of the blood takes place, the readily available, or ionized, portion is affected markedly. This ionized calcium is, in part, temporarily bound by the increased alkalinity present in the blood in respiratory alkalosis. The decrease in available calcium increases the excitability of the neuromuscular mechanism, thus inducing tetany.

It recently has been stressed that respiratory alkalosis will significantly alter the release of oxygen from hemoglobin within the tissues of the body.[3] With a lowered carbon dioxide pressure in the blood, hemoglobin clings more tenaciously to its bound oxygen. Less oxygen is relinquished in the tissues as the blood passes through. Another factor which interferes with tissue oxygenation is the decreased blood flow as a result of contracted blood vessels in tissues low in carbon dioxide pressure.[10]

Clinically the hyperventilation syndrome may occur in many forms and all forms have in common one feature—increased pulmonary ventilation. This respiratory abnormality may be observed in entirely stable subjects, particularly when they are exposed to emotional stress. Most commonly, however, this syndrome is observed in tense, excessively anxious, depressed or psychoneurotic patients. Such subjects often fall into the practice of hyperventilation when placed in a situation where embarrassment would be experienced should fainting occur, such as in crowds, in a front pew in church, or preoperatively in a hospital. At such times the respiration may be increased in depth, accelerated, or both, with the insidious onset of symptoms referable to respiratory alkalosis. Such patients will realize that something is amiss and will attempt to fight off their unpleasant symptoms through further hyperventilation by forced breathing.

Alkalosis may be induced by unrecognized stimulation of respiration as described. The frequent sighing observed in tense subjects may contribute to the production of an acapnia or lowered concentration of carbon dioxide in the blood. Experimentally forced breathing will reproduce the symptoms of respiratory alkalosis. It may well be that strenuous exercise by those unaccustomed to it will lead to acapnia; particularly is this true of swimming, an activity imposing a voluntary influence on an involuntary center for respiratory regulation. Physical activities at altitudes at which oxygen pressure is decreased may lead to a washing out of the carbon dioxide of the body more rapidly than this gas can accumulate through metabolic processes.

## SYMPTOMS

Following World War I White and Hahn studied large groups of subjects with and without the so-called effort syndrome. They observed sighing dyspnea in 80 per cent of such subjects. In a healthy control group this breathing irregularity was observed in but 19 per cent. They concluded that hyperventilation was responsible for the symptoms of this condition. In 1938 Soley and Shock likewise observed that hyperventilation was responsible for the symptoms of the effort syndrome. They substantiated their conclusions by carefully controlled physiologic studies of the changes occurring in the blood and alveolar air.

Gliebe and Auerback have emphasized the frequency with which hyperventilation may simulate organic disease. They have observed hyperventilation as a part of the fear reaction in emotionally unstable persons. Confronted by a situation in which he is inadequate, a patient may transfer his anxiety to any organ by a sequence of physiologic changes consequent to hyperventilation. Thus the precipitating psychic conflict may be ignored entirely as he pursues an explanation for his somatic symptoms.

The importance of hyperventilation as the so-called trigger mechanism in precipitation of manifestations of hysteria has also been emphasized. It is probable that hyperventilation contributes to the so-called acute anxiety attack.[2]

Because subjects who are otherwise quite stable will become subject to hyperventilation in times of anxiety or fear, this disturbance of respiration becomes of utmost importance in aviation. Faulty judgment and in-co-ordination in the course of overbreathing is one source of failure of pilots.[7, 8, 13]

The symptoms of respiratory alkalosis are insidious and minimal in their onset; however, if measures are not taken to rectify this abnormality of breathing, they may assume an alarming character. If the patient understands his condition poorly these symptoms may induce a state of panic as he realizes that something over which he has no control is happening. An early symptom is that of lightheadedness and unsteadiness. This is entirely subjective in its beginning. As the alkalosis progresses, a sense of a vacuum which is insatiable by the deep breathing it prompts is produced in the thorax. Patients may fan themselves and glance around for the nearest exit. Often they will go out of doors or open a window in an attempt to secure more adequate respiration. Explanations such as "The air won't go down far enough," "The air is doing no good" or "I can't get a satisfactory breath" may be offered. In the thorax a sense of dull pressure is often noted. This often leads to anxiety concerning the heart. For the examining physician such distress makes obvious the necessity for great care to exclude the pain arising from coronary artery disease.

With further hyperventilation a sense of numbness and tingling in the extremities and around the lips develops. Ultimately these pretetanic paresthesias are followed by spontaneous muscular twitching and then tetany of a carpopedal type. Throughout this sequence of physical changes mental astuteness is lost gradually and the loss is first apparent only in its effect on judgment and skill, but ultimately it progresses in some cases to stupor.

## DIAGNOSIS

On the basis of recognition of the manifestations of hyperventilation, the diagnosis can be made readily. Should the patient be examined by a physician at a time when evidences of respiratory alkalosis are not present, the characteristic history will be the guide to a correct appraisal of the patient's complaint. Under such circumstances, the symptom of which the patient complains frequently may be reproduced by a period of forced breathing sufficient to induce respiratory alkalosis. Three minutes of moderately accelerated breathing with care to exhale the supplemental air usually will suffice. Often only a part of the patient's symptoms may be reproduced, the deficit being attributable to the lack of a fear response associated with the release of epinephrine within the body.

## TREATMENT

Treatment of the hyperventilation syndrome depends largely on its diagnosis which enables the physician to offer an explanation of the condition to the patient. By avoiding the practice of forced breathing, by temporarily holding the breath, or by rebreathing air exhaled into a paper sack, the patient may bring about alleviation of his symptoms within a few seconds. Nasal, instead of oral, breathing likewise is of value in overcoming respiratory alkalosis. By means of these measures carbon dioxide is allowed to reaccumulate in the alveolar air. The most anxious patient appreciates the physician's demonstration that the terrifying sensations are not imaginary. He develops confidence that his condition is understood and becomes willing to discuss any underlying psychic factor. In many cases it is only necessary to demonstrate that the symptoms are the result of hyperventilation and to explain the nature of these symptoms to the patient. In other cases, the anxiety is more deeply motivated and prolonged psychiatric investigation and treatment may be indicated.

The recognition of the hyperventilation syndrome represents a means of explaining symptoms long known to be functional in origin but little understood. This knowledge has provided a way of adequately treating a previously unsatisfied group of deserving patients who "know something is wrong."

## REFERENCES


1. Best, C. H. and Taylor, N. B.: The physiologic basis of medical practice; a University of Toronto text in applied physiology. Ed. 4, Baltimore, The Williams & Wilkins Company, 1945, pp. 350 and 361.
2. Carryer, H. M.: The role of hyperventilation in functional disorders. Proc. Staff Meet., Mayo Clin. *21*:361–367 (Sept. 18) 1946.
3. Carryer, H. M.: The cause of dyspnea in the hyperventilation syndrome. Proc. Staff Meet., Mayo Clin. (In press.)
4. DuLaurens, Andre: Quoted by Kerr, W. J., Dalton, J. W. and Gliebe, P. A.
5. Grant, S. B. and Goldman, Alfred: A study of forced respiration: experimental production of tetany. Am. J. Physiol. *52*:209–232 (June 1) 1920.
6. Gliebe, P. A. and Auerback, A.: Sighing and other forms of hyperventilation simulating organic disease. J. Nerv. & Ment. Dis. *99*:600–615 (May) 1944.
7. Hinshaw, H. C. and Boothby, W. M.: The syndrome of hyperventilation: its importance in aviation. Proc. Staff Meet., Mayo Clin. *16*:211–213 (Apr. 2) 1941.
8. Hinshaw, H. C., Rushmer, R. F. and Boothby, W. M.: The hyperventilation syndrome and its importance in aviation. J. Aviation Med. *14*:100–104 (June) 1943.
9. Kerr, W. J., Dalton, J. W. and Gliebe, P. A.: Some physical phenomena associated with the anxiety states and their relation to hyperventilation. Ann. Int. Med. *11*:962–992 (Dec.) 1937.
10. McDowall, R. J. S.: The effect of carbon dioxide on the circulation. Part 1. J. Physiol. *70*:301–315 (Oct. 31) 1930.
11. Maytum, C. K.: Tetany caused by functional dyspnea with hyperventilation: report of a case. Proc. Staff Meet., Mayo Clin. *8*:282–284 (May 10) 1933.
12. Maytum, C. K. and Willius, F. A.: Abnormal respiration of functional origin. Proc. Staff Meet., Mayo Clin. *9*:308–311 (May 23) 1934.
13. Rushmer, R. F., Boothby, W. M. and Hinshaw, H. C.: Some effects of hyperventilation with special reference to aviation medicine. Proc. Staff Meet., Mayo Clin. *16*:801–808 (Dec. 17) 1941.
14. Soley, M. H. and Shock, N. W.: The etiology of effort syndrome. Am. J. M. Sc. *196*:840–851 (Dec.) 1938.
15. White, P. D. and Hahn, R. G.: The symptoms of sighing in cardiovascular diagnosis; with spirographic observations. Am. J. M. Sc. *177*:179–188 (Feb.) 1929.