COMMUNICATIONS TO THE EDITOR

Primary Alveolar Hypoventilation in a Thin Young Woman

To the Editor:

Alveolar hypoventilation in association with obesity was described first by Burwell in 1956. In later years, many cases of this syndrome have been reported. A similar syndrome in nonobese subjects was recognized in 1965. By 1970, 30 cases, most of whom were men, had been reported.

We wish to report a case of idiopathic hypoventilation in a young, thin woman. We shall point out the clinical features that permit one to establish the diagnosis.

A 22-year-old woman developed malaise and weight loss over a three year period. Later, headache, fatigue and somnolence developed. There was no dyspnea. She did not smoke or use drugs and there had been no previous illnesses.

She weighed 100 pounds and her blood pressure was 110/84 mm Hg. Respiration was irregular at a rate of 12 per minute and with periods of apnea. Physical examination of the lungs was normal. There was good excursion of the chest during voluntary deep breathing. A right ventricular lift and an increase in the pulmonic component of the second heart sound were noted. Neurologic examination was normal.

The hematocrit was 57 percent. Spinal fluid examination was normal. Values of blood urea, sodium, potassium, glucose, uric acid, cholesterol, protein, bilirubin, alkaline phosphatase, lactic dehydrogenase and glutamic oxaloacetic transaminase were normal. Protein bound iodine, triiodothyronine and radioactive iodine uptake by the thyroid gland was normal. Hemoglobin electrophoresis was 100 percent A. Serum protein electrophoresis was normal. Carbon dioxide combining power was 33 mEq/liter. Electrocardiogram was normal. X-ray films of the chest and skull were normal. Arterial pH was 7.25, PaO₂ was 55 mm Hg, and PaCO₂ was 91 mm Hg while at rest breathing room air. PaO₂ rose to 76 mm Hg and PaCO₂ fell to 48 mm Hg during voluntary hyperventilation of room air for three minutes. Pulmonary artery pressure was 90 mm Hg during cardiac catheterization. No intracardiac shunts were detected with a hydrogen electrode. Pulmonary arteriograms were normal. Pulmonary function studies demonstrated minimal restrictive dysfunction which was attributed to somnolence and poor respiratory effort.

Carotid and vertebral arteriograms were normal and were performed without difficulty. However, coma developed during the cerebral arteriography, possibly related to further respiratory depression secondary to intramuscular administration of 10 mg of diazepam as a preanesthetic medication. Mechanical ventilation was initiated, but she died 36 hours later. No structural abnormalities were found in the heart, lungs or central nervous system at autopsy.

The absence of dyspnea in a patient with profound cyanosis and hypercapnia suggests inadequate ventilatory drive. The hypercapnia and the hypoxemia may lead to headache, fatigue, and somnolence. An irregular respiratory pattern with periods of apnea occurs, particularly during sleep. Polycythemia is a consistent finding, and there may be right ventricular hypertrophy, pulmonary hypertension and right sided heart failure.

The diagnosis of primary alveolar hypoventilation should be considered when there are no known causes of chronic alveolar underventilation. One should exclude chronic airways obstruction, diseases limiting movement of the thoracic cage, and diseases of the central nervous system. All of these conditions generally can be eliminated easily as causative factors.

The diagnosis can be confirmed by voluntary hyperventilation of room air. This should result in improvement in hypercapnia and hypoxemia. On the other hand, inhalation of increased concentrations of carbon dioxide up to seven per cent does not cause dyspnea or discomfort. Ordinarily, concentrations of carbon dioxide greater than 4 percent cause normal subjects to complain of shortness of breath.

The cause of respiratory center dysfunction in idiopathic alveolar hypoventilation is unknown. A consistent anatomic lesion has not been demonstrated in the central nervous system. The hypoxemia may produce pulmonary arteriolar spasm which could lead to increased pulmonary vascular resistance. Pulmonary hypertension, right ventricular hypertrophy and right heart failure may follow the increased vascular resistance. Fatigue, head-

Figure 1. Recumbent view of the chest illustrating clear lungs fields and normal cardiovascular system except for enlarged pulmonary outflow tract.
aches and somnolence probably are the result of the chronic hypercapnia.5

Therapy has not been effective and prognosis is poor. Heart failure and respiratory infection develop frequently, and mechanical support of ventilation is often necessary. Pharmacologic or electrical stimulation of respiration may offer hope for the future.

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REFERENCES


Thoreau, Pulmonary Tuberculosis and Dietary Deficiency

To the Editor:

I wonder if Henry David Thoreau was responsible for his death from pulmonary tuberculosis in his prime at the age of 45? He lived the simple life, did not drink or smoke, and spent much time in outdoor activities. As far as we know, he had no contact with a tuberculous individual. During the two years of Spartan life at Walden pond, from July 4, 1845 to September 6, 1847, he tried to be self-sufficient in all aspects of living and wanted to prove that one could maintain health on a cheap, plain diet. His expenses for food for eight months was $8.74, for such staples as rice, molasses, rye meal, Indian meal, salt pork, flour, sugar, lard, apples, dried apple, sweet potatoes, one pumpkin, one watermelon, and salt. In addition to these foods, he ate potatoes, green corn, and peas, which he raised. He wrote, “I sometimes caught a mess of fish for my dinner, and once went so far as to slaughter a woodchuck which ravaged my bean field. . . . It was fit that I should live on rice, mainly, who love so well the philosophy of India. . . . The reader will perceive that I am treating the subject rather from an economic than a dietetic point of view.”

Thus, Thoreau lived on a quantitatively adequate diet from the caloric standpoint, but on a qualitatively poor diet because of the very small amount of protein-rich foods. For two years, he ate enough to appease hunger and rarely catered to appetite. “Yet men have come to such a pass that they frequently starve not for want of necessities, but for want of luxuries,” he wrote. The essential and more expensive protein-rich foods such as, meat, poultry, fish, pork, eggs, cheese, milk and butter, presumably were the luxuries referred to.

My purpose in this communication is to suggest that Thoreau’s diet during the two years at Walden consisted mainly of foods with high carbohydrate content, chiefly starch, and was woefully lacking in high quality protein. Based on clinical research in the treatment of tuberculosis with a low carbohydrate high protein diet,1,2 I concluded that the most important factor in susceptibility to tuberculosis was poor nutrition, and specifically a diet deficient in high quality protein. Individuals who eat to excess to the point of obesity may develop tuberculosis because of deficient protein intake. Among my patients there were several who were obese and who had far advanced cavitary disease.

Faber,3 in an analysis of factors responsible for the increase in tuberculous mortality in Denmark, Sweden, and England during World War I, concluded that the reduced consumption of meat and fish was the most important nutritional factor. He found that “the total calories consumed was not diminished because there was sufficient bread and flour.”

Thus, it is possible that Thoreau developed a tuberculous lesion of minimal extent during the two years on his Walden diet. The lesion probably became inactive after he left Walden and returned to Concord to live with his family where his diet improved. The lesion could have remained inactive or quiescent until he caught a severe cold in December, 1860. The cold persisted and developed into chronic bronchitis with cough which lasted all through 1861. His condition gradually worsened and he died of “consumption” on May 6, 1862.

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