

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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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 cavitory 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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The Mechanism of Bronchial Breathing

Though "bronchial breathing" is a well known physical sign and generations of medical students have been taught its recognition, no clear explanation as to the production of its characteristic features appears in medical texts.

Consideration of events taking place during the respiratory cycle provides an understanding of "bronchial" breath sounds. "Bronchial breathing" and its close associates "tracheal" and "tubular" breathing represent the normal sounds of air flowing back and forth through the bronchial tree. The sounds of bronchial breathing are heard almost unchanged since they are transmitted to the stethoscope by the nonaerated tissue of collapsed or consolidated lung. Because of the tubular structure of the bronchial tree, breath sounds originally contain a number of single frequencies giving them a somewhat musical quality.¹ Also, as might be expected, inspiration and expiration produce sounds of almost equal intensity, both reaching maxima at

periods of peak flow. It is these features which characterize "bronchial breathing."

So-called normal breath sounds, *ie* those heard over aerated lung, have necessarily a very different quality. Firstly, air passing through progressively smaller bronchi and bronchioles results in the production of sounds containing an increasing number of different frequencies, while the interposed alveoli provide a vast quantity of tiny sound absorption units,² so that sounds arriving at the chest wall have lost much of their musical character and volume. Of equal importance is that the auscultated sound has passed through the lung at varying degrees of aeration, the amount of which depends on the particular instant of time in the respiratory cycle. During maximal flow at the start of inspiration the lung is at its smallest volume and the minimum of aerated lung separates the bronchial tree from the chest wall. Inspiration is therefore comparatively loud. This is not so during expiration since peak flow takes place with the lung volume at its greatest, so that the maximal quantity of aerated lung lies between the bronchi and the chest wall. Expiratory sound is consequently much quieter than inspiratory.

It is therefore "bronchial breathing" which most faithfully represents sounds created in the bronchial tree. So-called normal or vesicular breath sounds are altered by the varying properties of the tissues through which they pass.

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