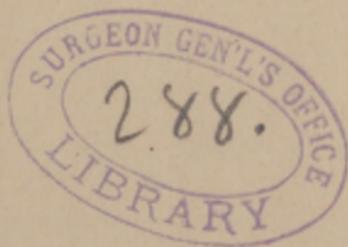


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of the Cricoid.

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A CASE OF
PERICHONDritis OF THE LARYNX;
NECROSIS OF THE CRICOID.*

By C. H. KNIGHT, M. D.

DESTRUCTION of the cartilages of the larynx, resulting from inflammation of their perichondrium, is by no means uncommon. Numerous instances have been recorded of its occurrence as a sequel of typhoid fever, as a complication of tubercular laryngitis, associated with malignant disease of the larynx, and in the course of syphilis. The opportunity of studying the condition in a pathological specimen may perhaps lend additional interest to the following history:

W. S. M., aged seventy-three, single, cabinet-maker, came to the Northern Dispensary in February, 1886, suffering from dyspnoea and dysphonia, which he said began about three months previous and had been gradually increasing. The obstruction was obviously laryngeal and affected equally expiration and inspiration. The voice was remarkably low-pitched and raucous. There was occasional hoarse cough, with expectoration of thin purulent sputa, which showed streaks of blood after an unusually violent paroxysm of cough. The breath was extremely foetid. There was no pain referable to the larynx,

* Read before the American Laryngological Association at its eighth annual congress.

except during phonation or deglutition and after coughing, but the patient complained of sensitiveness under external pressure, and when the larynx was moved from side to side. There was no apparent thickening, swelling, or œdema over the region of the larynx. The cervical glands were not enlarged. Examination with the laryngoscope showed a diffuse, symmetrical œdematous swelling of the posterior wall of the larynx, the natural contour of the arytenoids being entirely effaced. Only a small portion of the anterior extremities of the vocal cords was visible. They seemed to be normal. The epiglottis was congested and somewhat swollen. Oozing into the cavity of the larynx from its posterior wall appeared a small quantity of thin, white secretion. It was impossible to keep the laryngeal mirror in position more than a few seconds on account of the necessity of clearing the larynx. No ulceration could be seen, and the source of the secretion could not be precisely determined. Owing to the patient's condition, no attempt was made to probe the larynx or to carry on a more extended examination.

The general condition of the patient was extremely bad. He was very weak and much emaciated, having been able to take little or no solid food for three or four weeks. He had received no treatment whatever, except a series of cough mixtures, which, of course, gave him no relief. Stimulants and soft food were ordered, and the patient was directed to use every two hours a steam inhalation of compound tincture of benzoin. He was not seen again for four days, when he reported in a very much improved condition. Still the tumefaction over the arytenoids prevented a complete view of the cords. The amount of secretion in the larynx seemed to be less; its character was unchanged. Breathing was much easier, but the dysphagia was only little, if at all, diminished. Again the patient disappeared and was not seen for a week, when he returned in a very much worse condition than when he first applied for relief. His breathing was excessively labored and he was in a state of extreme exhaustion. He was at once ordered to the hospital, where it was my intention to perform tracheotomy, if necessary. Instead of following my instructions, he went to his home. The following morning he rose from his bed to light his

fire, fell on the floor, and died, before a friend, who occupied the room with him, could reach him. According to the best information obtainable, death seems to have occurred quietly, without struggle or sign of suffocation, apparently from as-thenia.

The autopsy was made, with the assistance of Dr. Henriques, fourteen hours after death. The larynx, pharynx, and œsophagus were first removed intact. On section of the trachea, a small quantity of bloody pus was found on the mucous surface as far down as the bifurcation. The lungs showed numerous emphysematous areas and old pleuritic adhesions, but otherwise were normal. No trace of tubercular deposit was found and no syphilitic lesion could be discovered. The heart appeared to be normal; the pericardial sac contained about two drachms of perfectly clear serum. The specimen itself, which has been most carefully examined by Dr. Ferguson, of New York, consists of the pharynx, larynx, and upper portions of the œsophagus and trachea. On the anterior wall of the laryngo-pharynx is an ulcer exposing the entire thickness of the partially ossified cricoid cartilage. It does not communicate with the larynx or trachea. Its edges are undermined to the extent of half an inch along the posterior surface of the cartilage. An ulcer involving the mucosa and the muscular tissue occupies the posterior wall of the pharynx at the level of the cricoid. The edges of both ulcers are elevated and very much thickened. There are superficial erosions of the vocal cords. There are numerous miliary masses on the mucous surface of the trachea composed of round and oval cells, granular detritus, bacteria, and micrococci. Numerous sections through the tissues surrounding the pharyngeal ulcers fail to discover tubercle bacilli or evidence of tumor development. Collections of small round cells are found, limiting small areas of degeneration, which give the appearance of tubercles. There are no giant cells. The fibrous tissue between the muscular bundles in the wall of the pharynx near the ulcers is increased and contains many groups of small round and spindle-form cells. Sections through the mucous membrane of the trachea show similar cells, and the stroma of the mucous glands is infiltrated with similar small round elements.

So much has been written on the subject of laryngeal perichondritis that it is not worth while to detain you with an extended review. The exhaustive article by von Ziemssen in his "Cyclopædia" (vol. ii, 814), where numerous valuable references may be found, and contributions by Türk, Mackenzie, Schroetter, Retslag, and others thoroughly cover the ground. Perhaps the most important questions relate to diagnosis, and, secondarily, to prognosis. In many cases the laryngeal condition may escape the notice it deserves, since the constitutional disease provoking it or associated with it demands the larger share of attention. The prognosis must be considered grave, if not necessarily fatal, especially when the cricoid cartilage is involved. A case has been reported by Ruehle in which this cartilage became necrotic and was discharged entire. Cohen narrates an interesting case of chondritis of the cricoid in which he did tracheotomy, and opened several intra-laryngeal abscesses. There was apparent reproduction of the cartilage, "the original one remaining necrosed in the cricoidal portion of the interior of the larynx as a foreign body" ("Diseases of the Throat and Nasal Passages," p. 526; also, "Trans. of the Path. Soc. of Phila.," 1874, p. 148). The degree of deformity and consequent impairment of function depend upon what and how many cartilages may be involved. The difficulty of determining this point is often insurmountable. A thorough examination may be impracticable, owing to intolerance of the parts, or to the general condition of the patient; or, in case a laryngeal exploration is possible, the region may be so obscured and distorted by inflammatory thickening, ulceration, or œdema, that the usual landmarks can not be identified.

The question of ætiology is of scarcely less interest and importance. Necrosis of laryngeal cartilages following typhoid fever has been especially observed by Trousseau,

Rokitansky, and Sestier. Liebermeister maintains that in these cases the affection of the cartilage is not primary, but is due to extension of ulceration from the mucous membrane. On the other hand, Sestier asserts that it may occur without ulceration, and the latter view is corroborated by Greenfield. Tuberculosis, carcinoma, and syphilis have also been mentioned as prominent causes of laryngeal perichondritis. To these must be added variola and traumatism. Under the latter head should be included not only external violence, but also the injudicious use of œsophageal bougies, especially in the aged. In this connection I may refer briefly to a case not yet reported which occurred in the service of Dr. Asch, at the Manhattan Eye and Ear Hospital:

A woman, about forty years old, swallowed a plate of false teeth during an epileptic fit. The plate was dislodged from the pharynx with great difficulty. A few weeks later the woman came to the hospital with an abscess on the posterior wall of the larynx, which Dr. Asch opened. Subsequently the house surgeon did tracheotomy during an attack of acute œdema or spasm of the larynx. The patient recovered, with great deformity of the larynx and complete loss of voice.

Unfortunately, we have not had the opportunity to examine her recently, so that I am unable to describe her present condition. There is no doubt in my own mind that this was a case of traumatic perichondritis. Forceful and prolonged use of the voice, as in certain occupations, has been suggested as a cause by Flormann. Primary laryngeal chondritis, resulting from early ossification of the cartilage, may involve the perichondrium (Albers), and, finally, pressure of an ossified cricoid cartilage against the vertebral column may develop a traumatic perichondritis (Dittrich). In some cases it may be very difficult to discover the origin of the lesion, as in one reported by Hall ("Trans. of the Clin. Soc. of London," 1882, xv, 195; 1884, xvii, 151). His patient

died after the lapse of two years or more, and at the autopsy various lesions characteristic of syphilis were found. The conclusion was therefore adopted that syphilis had caused the necrotic changes in the larynx, which in the first instance had been attributed to a laryngitis accompanying an extensive bronchitis, no history and no signs of constitutional disease having been at that time discovered. In searching for the cause of the condition in the case which I have reported, it was learned that about one year ago the patient was violently seized by the throat in the course of an altercation and was nearly strangled. The immediate consequences of this incident were laryngeal pain and partial loss of voice, lasting nearly a week. No other trouble followed, except slight cough, which was supposed to be bronchial, until three months before the case came under my observation, when dyspnoea supervened, attended by marked change in the quality of the voice, and great pain and difficulty in swallowing. The patient had never had a serious illness since childhood, and positively denied syphilitic infection. He had never used liquor to excess; but it was found that his circumstances in life were such as to deprive him of proper nourishment, often for a very long period. It is quite evident that impairment of vitality due to this fact, combined with his extreme age and the condition of ossification of the cartilage, must have predisposed the patient to the development of the affection. It would seem, therefore, that traumatism may be regarded as the immediate cause of the laryngeal lesion. The unusual site and extent of the ulceration, and the absence of pulmonary tuberculosis and of tubercle bacilli in the sections examined microscopically, exclude the idea of tubercular laryngitis.



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