to which I am very much indebted, it will be seen that he says of this joint: "Even minor degrees of mechanical impairment of its mobility might produce serious troubles in phonation, whilst the complete bilateral true or spurious ankylosis of the arytenoid cartilages in that position which they occupy in phonation might directly cause the death of the patient, by the complete occlusion of the glottis which results from juxtaposition of these cartilages." This shows that Dr. Semon fully recognised the possible occurrence of a stenosis of the glottis under conditions similar to those with which we are now dealing, though he does not connect this lesion with the tuberculous process.

On the other hand, Case I of the present series actually demonstrated the existence of a secondary adhesive perichondritis, and consequently supports Dr. Semon's contention, arrived at on other grounds, that perichondritis is not invariably suppurative, but may assume a plastic or adhesive character.

Koch² says that complete ankylosis of the crico-arytenoid articulations may show the same laryngoscopic appearance as bilateral abductor paralysis. M. Schmidt³ hints at the possibility of a similar occurrence in phthisis. But none of these authors give particulars of a case, and it remains uncertain whether they have actually observed this condition of the glottis in laryngeal tuberculosis themselves.

It appears, then, from the cases that form the subject of the present communication, as well as from the observation of Brissaud and Ruault above quoted, that fixation of the vocal cords in the median position, simulating bilateral abductor paralysis, may occur in tubercular disease of the larynx, as the result of three different causes:—

1. Plastic infiltration around the arytenoid cartilages leading to adhesive perichondritis and spurious ankylosis.
2. Ulceration, followed by morbid adhesion of the altered vocal cords.
3. Suppurative crico-arytenoid arthritis.

To these causes it may be necessary to add a fourth, if, as is not improbable, further investigations should demonstrate the existence of a non-suppurative adhesive arthritis.

In all cases of this description the performance of tracheotomy, or some other surgical measure, is indispensable, and should not be long delayed; consequently the early recognition of this condition is a matter of no small clinical importance.

CASE OF SUPPURATIVE LARYNGEAL PERICHONDritis, WITHOUT ANY ANTECEDENT DISEASE IN THE LARYNX.

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SO-CALLED primary laryngeal perichondritis, or, more correctly, perichondritis without any previous affection of the larynx, is so rare that I think it right to place on record the following case, which came under my notice a few months ago at the Glasgow Royal Infirmary.

Mrs. B., a Greek, aged 45, was admitted to the throat ward on October 19th, 1889, complaining of hoarseness, inspiratory dyspnoea, and cough. On inquiring into the history of the case, it was discovered that about three weeks previous to admission the patient had several rigors, and shortly afterwards she experienced slight pain in the throat. This illness she attributed to having slept in a damp bed while residing in the country. A week after the first rigor she noticed that her voice was not so clear as usual, and she suffered from difficulty in breathing. The hoarseness and dyspnoea gradually increased till she applied for admission to the hospital, and for two days previous thereto these symptoms were associated with a moderate cough and expectoration. In respect to personal history, nothing of any importance could be made out. She has always been strong and healthy, and never suffered from any throat or chest affection.

On admission, the patient was seen to be suffering from great difficulty in breathing, associated with noisy stridulous inspiration, but expiration was comparatively quiet. The respirations

were twenty per minute. Cough was not very troublesome, but there was considerable and profuse frothy mucous expectoration. At this time an examination of the chest was impracticable on account of the extremely noisy breathing. When I saw the patient next morning I found her sitting up in bed, her pulse weak, and temperature normal; the inspirations were very noisy, but to all appearance the patient was receiving into her lungs a sufficient supply of air, the voice was very hoarse and high pitched, and the cough somewhat stridulous. A laryngoscopic examination could not be made satisfactorily till October 21st, two days after admission, when the following condition was noted. "There is a marked oedematous swelling of the mucous membrane covering the arytenoid cartilages and the false cords, so that only a narrow chink is left between them. During inspiration this opening is apparently not, but must in reality have been, more than one-twelfth of an inch wide, and its edges are thrown into vibration by the ingoing current of air. Upon expiration the opening becomes wider, and permits of free exit of air. This change is most marked upon the left side, the false cord being drawn away from the middle line, so that a view is obtained of the left vocal cord. The left vocal cord is seen to be fixed in the middle line, but no view can be obtained of the cord on the right side."

On November 1st the respiration, although still noisy, was considerably improved, so much so indeed that the patient was very anxious to go home. An examination of the larynx showed the oedema of the mucous membrane to be greatly reduced, but still there was considerable swelling on both sides of the larynx. The patient was therefore strongly advised to remain in the ward; but, notwithstanding this advice, she left the hospital in the afternoon.

A physical examination of the chest made during her residence in the ward showed her lungs to be normal, but the heart was very weak, the area of cardiac dullness increased, but no murmur was present.

On November 25th, a daughter of the patient came to the throat dispensary and informed me that her mother had been very ill for the last three days, and from her description the patient must have had several severe attacks of paroxysmal dyspnoea, probably due to laryngeal spasm. I therefore asked my assistant, Dr. D. McKellar Dewar, to see the patient at her own home for the purpose of ascertaining if the patient was in a fit condition to be removed to the hospital, which he kindly did on the same day. Dr. Dewar, on making a careful examination, found the patient too weak to be disturbed, and he was also satisfied that the obstruction to respiration was not greater than when she left the ward, but it was evident to him that the cardiac weakness had become more threatening.

The patient died suddenly on November 28th, and her friends said that she passed "quietly away" without manifesting any signs of dyspnoea. The friends objected to a *post-mortem* examination, and with considerable difficulty Dr. Dewar got permission to examine the larynx only. The following is his report:—"The appearance of the larynx before cutting open confirms the laryngoscopic view, but adds nothing to it. On splitting up the cricoid cartilage from behind and opening the larynx the posterior surface of the cricoid cartilage is seen to be distinctly eroded a little to the left of the posterior middle line. The right cord is nodular, and immediately below it there is an opening into an abscess cavity of small dimensions, which reaches down the trachea for about half an inch. In addition to the oedema, the mucous membrane of the whole larynx is injected."

REMARKS.—The case just described is peculiar (1) in the obscurity of the disease, the symptoms not being those usually met with in perichondritis, and (2) in so far that there was no apparent cause for what might, by some observers, be regarded as a spontaneous suppuration between the perichondrium and the cricoid cartilage.

(1). In perichondritis of the cricoid cartilage the presence of pain on deglutition is usually complained of by the patient, owing to the irritation produced by substances swallowed. The lymphatic glands in the neck generally enlarge early in the course of the case; there is swelling in front of or around the larynx; and, as a rule, external palpation of the cartilages with the hand produces crepitation, and causes the patient considerable pain; the expectoration contains pus, or purulent mucus may be seen occupying the larynx. In this case none of these symptoms were present. On the other hand, the inflammation of the cricoid cartilage evidently impaired the movement of

² *Annales des Mal. de l'Oreille et du Larynx*, No. 6, quoted by Semon, p. 14 of reprint.

³ *Deutsch. Arch. f. kl. Med.*, Bd. 26.

the abductor muscles of the left vocal cord, as ascertained by laryngoscopic examination, but whether the movements of the right posterior abductors were normal could not be determined during life. The alteration in the voice, the great oedema, and the characteristic cough certainly led one, in the absence of any disease in the mucous membrane, to suspect some serious deep-seated inflammation; but the absence of pain on deglutition or on palpation, the absence of swelling over the larynx and of enlargement of the lymphatic glands, and the circumstance that no abscess could be discerned either by the escape of pus or by the presence of an opening, tended towards the formation of a more favourable diagnosis.

According to Gerhardt,¹ when there is immobility of the vocal cords, clearly not due to paralysis, and associated with marked thickening of the wall of the larynx, particularly of the ventricular bands, then perichondritis should be suspected if a primary disease of the larynx exists which is likely to cause inflammation of the perichondrium. Dr. F. de Havilland Hall goes even further, and says: "Now I accept Gerhardt's conclusions, but would extend them to cases in which the causes of secondary perichondritis are absent."

The case I have just reported demonstrates that 'perichondritis' does occur independently of any antecedent disease in the larynx, and it appears to me that with the facts before us, at the time it was impossible to say definitely that the cartilages were diseased, although I think I was justified in assuming that there was serious deep-seated inflammation.

One of the difficulties we have to contend with is that so-called primary perichondritis is so rare that we seldom think of it in forming a diagnosis, and the symptoms of the disease are so mild, being less severe than those of secondary perichondritis, that death seldom occurs, and an opportunity is not afforded of verifying the diagnosis by a demonstration *post mortem*.

Two other cases of perichondritis have come under my notice, one in hospital, the other in private practice, in which considerable difficulty was experienced during the early stage of the disease in determining whether the lesion was primary laryngeal tuberculosis or inflammation of the perichondrium. Under treatment (on the assumption that the latter condition was the one I had to deal with) by rest, leeching, and the application of cold, followed by administration of iodide of potassium, both cases made a good recovery, and, with the exception of some occasional feebleness of the voice, they are practically well. These, I presume, were examples of perichondritis without suppuration.

2. In almost all cases of so-called perichondritis the disease can be traced to some definite cause. Thus cases have been recorded where it has resulted from injuries such as gunshot wounds, stabs, and incised wounds, or other traumatic lesions. The way in which such causes produce necrosis can easily be understood. On the other hand, in necrosis of the laryngeal cartilages, just as in other forms of necrosis, the disease may be due to deficient nutrition, which may arise from an incomplete or vitiated supply of blood, general marasmus, or hydræmia. Thus in old persons who have suffered from long-standing disease of the heart and arteries, necrosis of the extremities is liable to appear after slight injuries. In the same way in emaciated and bedridden patients necrosis of the cartilages of the larynx may occur. According to Dittrich, in such cases pressure upon the laryngeal cartilages, by reason of decubitus, the pharyngeal muscles being extremely relaxed (the posterior surface of the cricoid is pressed by reason of its own weight against the vertebral column), may cause trophic changes, which give rise to inflammation of the perichondrium, followed by what might be called senile or decubitus necrosis of the œsophagus and of the laryngeal cartilages. In the case under my care no such cause could be detected, nor could it be shown that the patient suffered from any of the diseases most commonly associated with secondary necrosis of the cartilages, such as rheumatism, gout, syphilis, tuberculosis, or any acute and chronic infectious, or infective disease, likely to give rise to the formation of metastatic abscess. There seem, therefore, to be only two possible explanations of the case: either we must believe that this is an example of spontaneous suppuration under the perichondrium, or we must seek for some cause of septic infection. Spontaneous suppuration properly so called, that is to say, the formation of an abscess without local injury or septic infection, is inconsistent with our present views of the pathology of inflammation. But that localised suppuration may occur without any very clear explanation of the mode of infection is willingly

admitted. In cases of perichondritis following upon syphilitic, tubercular, or carcinomatous ulceration, the morbid process is easily studied. Septic or specific micro-organisms penetrate the perichondrium from the mucous or ulcerated surface of the larynx, and there set up inflammatory changes, at first characterised by thickening of the fibrous tissue of the perichondrium, and followed by the formation of pus, which accumulates so as to dissect from the cartilage its source of nutrition, in the same way as in necrosis of bone the periosteum is separated, and leads to death of the part. The morbid process is generally confined to one cartilage—the cricoid—to begin with, but it may in severe cases extend to others. As the pus accumulates, the perichondrium softens and ruptures, and the inflammation spreads to the surrounding parts through which the pus burrows, and, according to the situation of the disease, an abscess may rupture into the œsophagus, pharynx, larynx, or trachea, or it may discharge externally, and produce a laryngeal fistula. The dead or necrosed cartilage becomes eroded, and is slowly separated from the living. When complete exfoliation has taken place, the abscess collapses, and, as a rule, leads to great deformity of the larynx.

In the case of Mrs. B., the disease is only in its early stage, the abscess having evidently just evacuated itself. We have here a disease very closely related to acute periostitis. The disease has probably been induced by some slight injury which escaped observation, and under ordinary circumstances would not have caused harm, but when associated with the accidental presence of septic microbes in the blood the injury otherwise trifling has been sufficient to reduce the vitality and resisting power of the part, and determine the colonisation of the micro-organisms in the perichondrium. When it is remembered that the cartilages of the body, except those of joints, are covered with perichondrium, it is a circumstance worthy of note that with the exception of the cartilage of the septum nasi the laryngeal cartilages are the only ones liable to perichondritis. The perichondrium of the nasal and laryngeal cartilages behaves more like periosteum, a structure which in certain situations is very liable to acute inflammation; why this is so is difficult to explain. In almost all instances when cartilage shows active inflammatory changes, these are due to secondary processes extending from other parts, not starting in the cartilage itself. For example, chondritis or perichondritis very rarely attacks the costal cartilages, or the cartilages of the ear, and inflammation of the articular cartilages, although very common, is invariably produced by an irritant being brought in contact with one of its surfaces, either by direct infection of the joint, or by disease of the underlying bone. One reason which may be advanced in explanation of the laryngeal cartilages being liable to inflammation is that in breathing, and swallowing heated food, they are more exposed to sudden changes in temperature, and during deglutition the bolus of food may injure them. This explanation is supported by the circumstance that the posterior aspect of the cricoid cartilage is more liable to attack than other parts of the larynx. Taking all the circumstances associated with the disease into account, I think it must be admitted that while perichondritis may be met with independently of any previous affection of the larynx, and may arise without any very clear explanation of the mode of infection, still the inflammation cannot be regarded as spontaneous. The pathology of perichondritis is probably similar to that of acute periostitis, the disease being due to septic influences exercised upon a structure whose resisting power has been impaired.

PASTEURISM IN CUBA.—At the Medical Congress recently held at Havana Dr. Acosta read a paper in which the results of M. Pasteur's preventive treatment in Cuba were set forth. The inoculations were made in the Bacteriological Laboratory of Havana, which was the first place in the tropics where the method was tried. From April 15th, 1887, to December 31st, 1889, 306 persons were treated by the "double intensive" plan; of these, only 2 died after going through the full course. The mortality was, therefore, only 1.63 per cent. The first death occurred in 1888 after 105 patients had been treated without any fatality. All the persons submitted to treatment were bitten by dogs proved experimentally and clinically to be rabid, or at any rate "suspect." Seven hundred persons in all had applied for treatment, but only the 306 referred to had been inoculated. Dr. Santos Fernandez, the founder of the laboratory, proposed that a telegram should be sent to M. Pasteur in the name of the Congress, expressing its endorsement of his teaching on the prevention of rabies. This was agreed to.

¹ *Berlin klin. Wochenschr.*, 1887, No. 24, p. 439.

² *JOURNAL*, 1889, vol. ii, p. 559.