# CHORDITIS FIBRINOSA.*

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THE object of this paper is to direct attention to an uncommon variety of acute laryngitis characterised by the deposition of fibrin and the occasional formation of erosions on the vocal cords. All of my cases occurred in soldiers, and neither before the war nor since two months after the Armistice have I met with a case of the kind. The rarity of the affection amongst civilians seems unquestionable from the absence in the principal English text-books of any description corresponding with the undernoted appearances.

**Material.**—During four years and eight months' service (August 1914 till May 1919) in the 4th Scottish General Hospital, Glasgow, 113 cases of laryngitis were treated by me, 40 of which presented exudation or erosions, or both, on the vocal cords. The first case was seen in November 1915, the last in February 1919.

**Season.**—The disease set in during the cold season between 1st October and 31st March, with the exception of 2 cases in May, June and September respectively; 5 of these six had been gassed.

**Symptoms.**—Nearly all of the patients suffered from hoarseness or aphonia; the latter symptom was present chiefly in men who had been gassed. A few coughed. None complained of pain in the throat.

**Causes.**—The causes assigned for the disability were: gas (23), cold (10), influenza (3), shouting commands (1), and coughing (1). In two cases no cause could be given.

**Appearances.**—In a recent and pronounced case of chorditis fibrinosa the vocal cords are of a pure white appearance excepting in their posterior third and over a small area quite anteriorly, both of which are red (Fig. 1). At first, one may mistake the white patch for the normal cord, but further examination and the subsequent course show that it is due to a deposit which gradually passes off, leaving exposed the red upper surface of the cord. When this process of resolution has advanced a little, the affection assumes its most characteristic aspect. The patch

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on each cord has then a somewhat semi-oval shape, and on adduction the oval is completed, its long axis coinciding with the glottis. The middle of this axis corresponds with the middle of the ligamentous part of the cord, or the junction of the anterior and middle thirds of the entire cord. The fibrinous deposit by degrees loses its lustre and becomes thinner and greyish. Its extent may remain about the same for a fortnight, and then in the course of three days it may entirely disappear. Oftener, however, it clears up from the periphery inwards, so that the red area at each end of the cord lengthens, and the two become connected by a gradually widening red tract to the outer side of the patch. Later, the exudation appears as a narrow line at the edge of the cord, and it is last seen as a small white dot at the middle of the ligamentous cord. The changes on the two cords generally proceed *pari passu*.

The original extent of the membrane may vary considerably in different patients. Instead of covering nearly all the ligamentous cord as already described, it may be limited to a small area around its middle point, as in the last stage of a pronounced case. Between these two extremes plaques of various sizes may be met with.

Rarely the cords are involved asymmetrically.

In two cases membrane was found on one cord only.

Apart from the involvement of the true cords, the larynx presents a normal aspect.

The average duration of the fibrinous exudation, assuming that it forms about the time when the voice becomes affected, is from three to four weeks. The voice, as a rule, is normal earlier, while the redness of the cords persists longer.

**Erosions.**—Erosive action is seen in about half of the cases of chorditis fibrinosa, usually in the later stages. One or several tiny notches form in the edge of one or both cords near the junction of the anterior and middle thirds; generally they disappear in three or four days (Fig. 2).

**Chorditis Fibrinosa contrasted with Exudative Laryngitis due to Mustard Gas.**—Although in a considerable proportion of the cases of chorditis fibrinosa the men had been exposed to gas and suffered from conjunctivitis and bronchitis as well as laryngitis, I do not regard the exudation on the cords in this affection as solely or directly due to gas. When mustard gas definitely affected the interior of the larynx—as observed in other six cases—an entirely different picture was produced.



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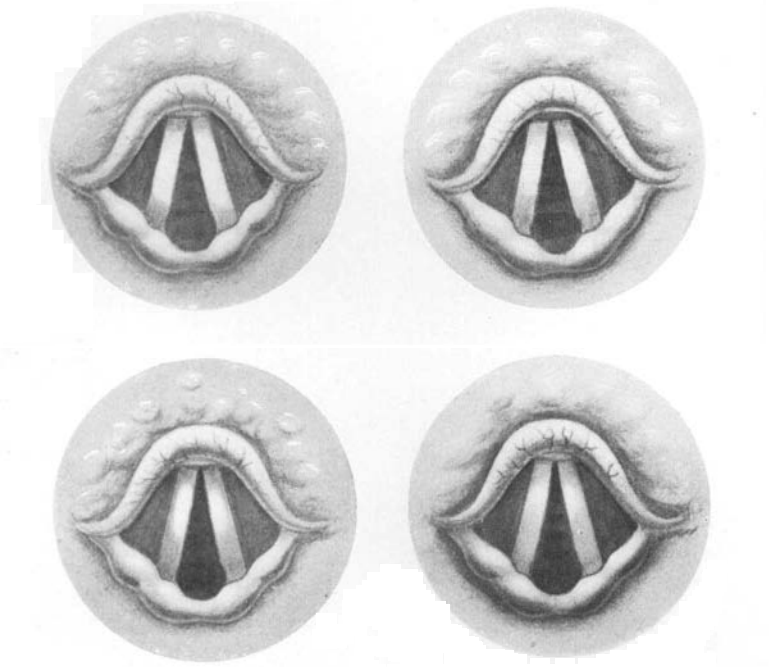


FIG. 1.—STAGES OF RESOLUTION IN CHORDITIS FIBRINOSA.

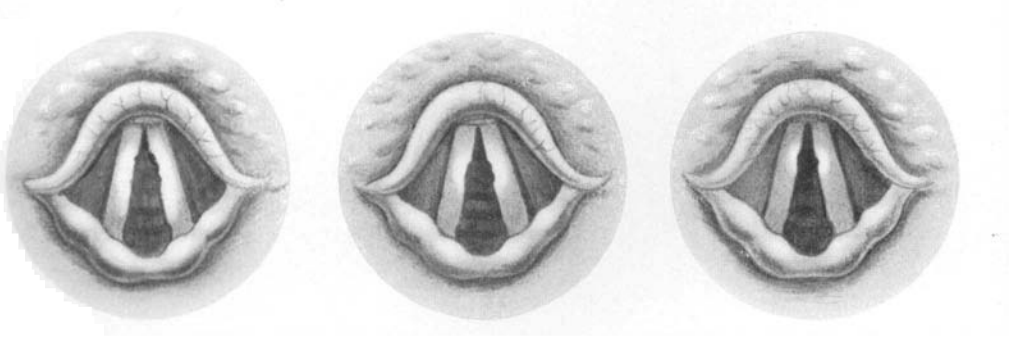


FIG. 2.—EROSIONS OF THE VOCAL CORDS IN CHORDITIS FIBRINOSA.

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The patient complained of painful deglutition, and on inspection irregular patches of white deposit were found oftenest on the posterior surface of the epiglottis, occasionally on the arytenoids and aryepiglottic folds, less often on the false cords, but never on the true cords. At the same time a patch usually covered more or less of the posterior pharyngeal wall, and smaller deposits might be seated on the palate and faucial pillars. The membrane in the larynx disappeared within a fortnight from the date of exposure to the gas; that in the pharynx, being thicker and more extensive, took a week longer to clear away. The duration in the larynx of membrane caused by mustard gas is thus much shorter than that of the exudation in chorditis fibrinosa.

**Treatment.**—The chief treatment consists in resting the voice. If this is neglected, or if, after the correction of a functional paresis of the cords, which is not uncommon in these cases, improper voice production is persisted in, the congestion of the cords and hoarseness may be maintained.

**Pathogenesis.**—In a paper published in 1890 on affections of the upper air-passages in influenza, B. Fränkel[1] described dirty white areas on the reddened upper surface of the vocal cords. He assumed that these patches were due to fibrinous infiltration, and regarded them as almost pathognomonic of influenza. Since then a few British writers, *e.g.* M'Bride, Watson-Williams and Tilley, have mentioned the occasional ulceration of the vocal cords in influenza. A lengthy review on fibrinous laryngitis by O. Seifert[2] appeared in 1916, in which it was pointed out that the term laryngitis fibrinosa covered a large number of affections of different aspect and etiology, including that under consideration. The writer therefore proposed the designation chorditis fibrinosa for the variety characterised by the formation of white plaques—presumably fibrinous—on the upper surface of the vocal cords, the rest of the larynx being apparently normal. This recommendation in nomenclature is likely to meet with general acceptance and has been adopted in this paper.

The description of chorditis fibrinosa given above agrees largely with Seifert's, but on some minor points our experiences have differed. Thus he does not appear to have met with erosions or ulcers at the site of the deposit as here reported, nor with a case in which one cord only was involved. Further, in all his recent cases he found a more or less pronounced degree of rhinitis sicca or rhino-pharyngitis sicca, to which he attaches



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etiological importance, whereas this condition existed in only one of my patients.

As to the pathogenesis of chorditis fibrinosa, our respective conclusions are entirely at variance.

Seifert appears to have taken for granted the influenzal nature of the affection. He maintains that it occurs exclusively in, and is typical of, influenza. He does not consider, however, that its development is dependent upon the severity of the infection, the amount of general disturbance or the degree of laryngitis. He states that one has often the opportunity of seeing recent cases in which the signs of inflammation are insignificant, while in others marked redness and swelling of the entire laryngeal mucous membrane are present. In his experience the formation of the fibrinous deposit takes place on the third or fourth day of the influenza and manifests itself by the sudden onset of hoarseness. By this time the general symptoms may have almost subsided, so that the patient is able to call upon the doctor. In other cases in which the general symptoms had been fairly severe, but convalescence had set in, he found the fibrinous exudation developing only on the eighth or tenth day, although the patient had been moderately hoarse from the beginning of the illness. He has seen chorditis fibrinosa very exceptionally in females—in neither sex under the age of 20—and chiefly in men between 25 and 50. This striking predilection of middle-aged men possibly points in his opinion to the existence of predisposing causes in other parts of the upper air-tract. In almost all the 38 soldiers with chorditis fibrinosa whom he examined during the first year of the war, dry catarrh of the nose and throat was found; the prevalence of this disease may have arisen from conditions in the barracks. Dry catarrhs of the upper respiratory passages, although not directly responsible for chorditis fibrinosa, are to be included, he thinks, amongst the factors favouring its development.

Chorditis fibrinosa may be due to influenza, but that it is always so and synonymous with influenzal laryngitis appears doubtful, even from some of Seifert's statements. In my experience a connection was exceptional, for in only 3 of the 40 cases was the aphonia or hoarseness said to have followed influenza. In some of those attributed to "a cold," influenza may possibly have been overlooked, but in the majority it could be excluded. While unwilling, therefore, to accord this disease the etiological importance hitherto claimed for it in chorditis fibrinosa, one



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cannot deny that the laryngologist ordinarily has few opportunities of inspecting the vocal cords in influenza or in acute laryngitis accompanied by constitutional disturbance; still this fact scarcely explains the rarity of chorditis fibrinosa in civilian patients. It is more likely that the comparative frequency of this affection in a military hospital is accounted for by the severe and exceptional conditions to which men are subjected while on active service.

Among the predisposing causes of laryngitis incident to the battlefield, are: shouting, panting respiration, violent exertion with subsequent exhaustion, and all that is involved in a death and life struggle. The inspired air may be cold, raw, and laden with irritating gases, exciting violent coughing—as already stated the affection was met with chiefly during the winter months. There may also be overuse of tobacco and alcohol. Some of these factors in a milder degree come into play on the training ground and at home, and account for the few cases that developed the disease in this country; the majority, however, were sent from the front as having been gassed.

A consideration of the local appearances, I think, throws light on the nature of the affection. The entire vocal cord is involved, but the white deposit is confined to the ligamentous part, *i.e.* the part that vibrates. The posterior third and a small area anteriorly at the attachment of the cord, both of which vibrate but little, if at all, are red and not covered by membrane. As the affection progresses towards recovery the white patch grows gradually less from its periphery inwards, so that before disappearing altogether it is reduced to a mere speck at the middle of the ligamentous cord close to its edge.

This point on the cord is that at which the maximum vibratory movement takes place. During phonation this point not only vibrates in a horizontal and vertical plane, but is pushed inwards so as to come into contact with the corresponding point on the other cord. It is, of course, at these points that singer's nodules develop.

Summarising these observations, it would therefore appear that in consequence of certain noxious agents, notably cold, gas, tobacco, and occasionally the influenzal virus, acute inflammation of the vocal cords is caused. Owing to exceptional factors, particularly shouting and rapid respiration, a damaging effect is produced especially upon the vibrating part of the cord, and necrosis of the superficial epithelium results. A fibrinous exuda-



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tion representing the process of repair takes place over the area so destroyed. The extent of both the necrosis and the resulting fibrinous deposit corresponds presumably to the severity of the process, and may vary from a small patch at the centre of the edge of the ligamentous cord to one covering this entire region. In all cases the former situation is evidently that at which the process is most intense or the aggravating conditions most active, for there the patch persists longest. Briefly, chorditis fibrinosa appears to be a variety of acute laryngitis in which the inflammation has been limited to the true cords, the ligamentous portions of which have suffered specially owing to excessive vibratory movement.

REFERENCES.—[1] Fränkel, B., *Verh. d. lar. Gesellsch. zu Berlin*, Bd. i. S. 30. [2] Seifert, O., *Arch. f. Lar.*, Bd. xxx. S. 83.



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