VOL. XIII.—NO. 10. OCTOBER, 1898.

THE

JOURNAL OF LARYNGOLOGY, RHINOLOGY, AND OTOLOGY.

Original Articles are accepted by the Editors of this Journal on the condition that they have not previously been published elsewhere.

Twenty-five reprints are allowed each author. If more are required it is requested that this be stated when the article is first forwarded to this Journal. Such extra reprints will be charged to the author.

The Editors are not responsible for opinions expressed in original Articles or Abstracts in this Journal.

Editorial Communications are to be addressed to "Editors of JOURNAL OF LARYNGOLOGY, *care of Rebman Publishing Company, Limited,* 129, *Shaftesbury Avenue, Cambridge Circus, London, W.C."*

SOCIETIES' MEETINGS.

BRITISH MEDICAL ASSOCIATION.

Meeting, July 8th, 1898.

PAPERS AND ABSTRACTS.

Dr. DAVID NEWMAN. *Early Symptoms of Pressure upon the Vagus and Recurrent Laryngeal Nerves.*

In a lecture published eleven years ago,[1] I endeavoured to show by reference to cases of aortic and innominate aneurysms—(1) That aneurysm of the aorta and of the innominate artery may exist and give rise to laryngeal symptoms only, but that in most instances, on critical physical examination, certain collateral signs may be made out sufficient to warrant one in forming a positive diagnosis, or to give rise to a very strong suspicion of an intrathoracic tumour; (2) that in the early stage pressure may cause paroxysms of most urgent dyspnœa, accompanied by laryngeal stridor and paroxysmal cough; (3) that at a later stage paralysis occurs, usually, but not always, limited to one side, characterized by phonative waste of breath and imperfect cough, but without dyspnœa, except when reflex spasm is indicated on the opposite side, or when pressure-stenosis is caused by the aneurysm.

Since the lecture referred to was published, other cases have come under my observation in which the respiratory symptoms were the first warnings of serious disease, aneurysmal or malignant, within the thorax.

It is because of some particular disturbance of function that the patient seeks advice, and in the cases referred to not infrequently the

[1] "British Medical Journal," July 2nd, 1887.

https://doi.org/10.1017/S1755146300167149 Published online by Cambridge University Press

respiratory organs suffered first, so that the condition was looked upon by the patient as a throat affection and nothing else; the symptoms, however, proved to be the danger signals of serious disease within the chest, causing, by pressure upon the nerve trunks, impairment of the functions of the laryngeal muscles. I do not propose in this short communication to refer in detail to individual cases, but prefer to give the general results of what I have observed.

1. In some cases one of the earliest symptoms of interference with the innervation of the larynx, by the pressure of an aneurysm or of a mediastinal tumour within the thorax, is *sudden and paroxysmal dyspnœa accompanied by laryngeal stridor*. While the stridor is, as a rule, most marked during inspiration, expiration being free, still in rare instances the exit of air also may be impeded. That death occurs in cases of aortic aneurysm as the result of laryngeal suffocation has been attested by numerous published cases, and is not now subject to doubt.

It is more particularly to the value of paroxysmal dyspnœa, as an early symptom of pressure upon the laryngeal or vagus nerves, that I now desire to direct attention, and this must be estimated by a careful consideration of the precise laryngeal conditions present, as well as by the other concomitant symptoms of the disease. In cases, however, where stridor is the first and only symptom, not infrequently the dyspnœa is so severe that during an attack a detailed examination with the laryngoscope is impossible, and when the attack passes off the appearances may be found to differ very little from the normal. During the intervals the voice may be comparatively little altered, there may be a total abeyance of symptoms, and the laryngoscope may fail to reveal any abnormality, but this negative fact I have found to be of the highest value. If there is, in a patient past middle life, a total absence of any lesion of the mucous membrane, and no condition can be discovered which may physically impede the movements of the vocal cord, and if there is no evidence of any central nervous lesion or of pulmonary disease, the presumption is strongly in favour of aneurysm or mediastinal tumour. Cases are seldom seen at this very early stage. The laryngoscope generally shows that there is some interference with the action of the muscles of the larynx.

Certain points of interest may now be considered. How does pressure upon the recurrent laryngeal and vagus nerves appear to the clinical observer to affect the muscles of the larynx? Is the dyspnœa due to paralysis of the abductors or to spasm of the adductor muscles?

I cannot here enter upon a critical enquiry of the various views advanced as to the anatomical origin of the laryngeal fibres of the vagus. It has been clearly demonstrated by experiments, supported by clinical observation, that on each side of the brain, at the lower extremity of the ascending frontal convolution, there is a bilateral cortical centre which controls the adductor muscles of the vocal cords, and that stimulation of one of these centres produces adduction of both vocal cords, while destruction of one, as in aphasia, does not involve paralysis of the laryngeal muscles on the corresponding side. Just below and in front of the adductor centre, Risian Russell describes a bilateral abductor centre, and below

https://doi.org/10.1017/S1755146300167149 Published online by Cambridge University Press

it an area stimulation of which results in a clonic abduction of the cords.

This subject has been very carefully discussed by so distinguished an expert as my friend Sir Felix Semon, who, along with Dr. Watson Williams, has contributed a most carefully considered article in Clifford Allbutt's "System of Medicine," Vol. IV., p. 841 *et seq.*, on "Laryngeal Neuroses."

After referring to the various experimental enquiries, the former author (Sir Felix Semon) calls attention to a law, namely, that in all progressive organic lesions of the centres or trunks of the motor laryngeal nerves, the abductors of the vocal cords succumb much earlier than the adductors, and immediately following he says, "although a large number of such cases of progressive organic disease acting upon the whole of the nerve trunk have been recorded, and publicly shown, in which the abductor muscles had undergone degeneration and atrophy, either alone or at any rate more advanced than in the adductors, not a single specimen has yet been demonstrated which, under similar conditions, exhibited the opposite order of events in the development of degenerative changes in the individual laryngeal muscles; all attacks made so far upon the law rest exclusively upon clinical observations, which are either incomplete, or are capable of an interpretation other than that adopted by their authors." At p. 851, he states that "paralysis of the abductors is almost invariably bilateral and due to functional disorders, probably cortical," while unilateral abductor paralysis is almost always the result of pressure upon the recurrent laryngeal nerve, and at p. 853 the following statement is found:—"In unilateral abductor paralysis the affected cord remains fixed in the median line, that is, in the position of phonation; and as the opposite cord is unaffected, respiration is not embarrassed unless the cause of the paralysis simultaneously produces direct compression of the lower air passages, as in not a few cases of aortic aneurysm. Under such circumstances, that is to say, in the initial stages of all the severe lesions mentioned as 'causes' which may implicate the laryngeal nerves—and indeed not rarely up to the patient's death—neither vocal nor respiratory symptoms need occur in adults: thus the laryngeal lesion, which may be of the greatest importance for the correct diagnosis of the whole case, will entirely escape notice unless it be a part of routine practice to examine all cases in which lesions of the laryngeal nerves could occur, whether there be symptoms pointing to the larynx or not."

The bearing of the above statement upon the question under consideration is most important, and while we must admit that in most cases of pressure upon the recurrent laryngeal nerve the vocal cord is in the position described, still the law is not absolute; otherwise, how are the paroxysms of sudden and paroxysmal dyspnœa to be explained? In complete bilateral abductor paralysis the vocal cords remain in or near the middle line, and the chink in the glottis presents much the same appearance as during phonation, so that during inspiration, especially when forced, a certain amount of stridor may be induced, but this form of paralysis is very rare, and when present is seldom complete. The

https://doi.org/10.1017/S1755146300167149 Published online by Cambridge University Press

dyspnœa may be explained in two ways—either from sucking in of the flaccid cords, or from the accumulation of mucus in the air-tubes on account of efficient coughing being impossible. Whether abduction or adduction follows upon electrical stimulation of the recurrent laryngeal nerve seems to depend upon the relative power of the antagonistic muscles of the larynx, for we find that while in one animal stimulation of the recurrent laryngeal leads to approximation of the vocal cords, in another it produces exactly the opposite effect ; and stimulation acting on the efferent fibres generally causes unilateral adduction or abduction of the vocal cord. Pressure on one recurrent laryngeal nerve is not likely to lead to serious dyspnœa, while, on the other hand, pressure on the vagus, by inducing spasm of the adductor muscles on both sides, or of paralysis on one side and spasm on the other, frequently leads to serious and sometimes to fatal laryngeal obstruction. When due to paralysis, dyspnœa is seldom severe, although persistently present in a greater or lesser degree on account of the partial closure of the glottis, while expiration is usually unimpeded. The dyspnœa observed at the early stage of pressure on the vagus is very different from this, the attacks are paroxysmal, sudden, and transitory, as in laryngismus stridulus. In spasm of the adductors, the closure of the glottis being complete, but transitory, gives rise to paroxysms of violent dyspnœa, which come on and pass off with equal suddenness. In the intervals between the paroxysms the voice is unaltered, and auscultation over the larynx fails to detect any impediment to respiration. The amount of pressure to which the nerve is submitted seems to my mind to determine whether or not the patient suffers from dyspnœa. If the pressure be slight, the irritation of the excito-motor nerve calls into action the muscles of the larynx, and the constrictors being more powerful, closure of the glottis results ; whereas in a later stage the function of the nerve is impaired or destroyed, and consequently diminished power is observed in the movements of the muscles on the affected side, while reflected spasm may be induced on the opposite side. The reason why dyspnœa should occur in the later as well as in the earlier stages is explained by the experiments of Dr. John Reid. I cannot understand how a paralysis of the abductor muscles could possibly cause sudden and transitory paroxysmal attacks, leaving the voice unimpaired during the intervals, and the larynx normal in appearance, as in a case (Case I.) referred to in the "British Medical Journal," 2nd July, 1887.

In the lecture above referred to I made the following remarks :—"Paroxysmal or constant dyspnœa, attended by other laryngeal symptoms, but without structural disease of the larynx, cardiac or pulmonary lesions, to account for the symptom, should always lead one to suspect the presence of aneurysm, involving either the innominate artery or the posterior and inferior aspect of the arch. When the spasm or paralysis of the vocal cord is limited to one side, the probability is that the recurrent laryngeal on that side is alone compressed, and in neither of these instances is dyspnœa a dangerous symptom, although, in the former, it may be a prominent one. If spasm be present, the pressure is probably

https://doi.org/10.1017/S1755146300167149 Published online by Cambridge University Press

slight and of short duration; if paralysis, the pressure is either great or prolonged. When the paralysis or spasm is bilateral one of two conditions may exist, (1) pressure upon both recurrent laryngeal nerves, or (2) pressure upon the trunk of one vagus."

Further experience has tended to confirm the opinion above expressed, and I find that these observations are in accord with those of one of our greatest clinical teachers, Prof. Sir William T. Gairdner, who has kindly allowed me to read a proof of an article on aneurysm of the aorta which will shortly appear in Clifford Allbutt's "System of Medicine." In the article referred to Sir William says, "the late Sir George Johnson believed that he had obtained evidence in support of the 'theory that a long-continued irritation of the trunk of one vagus may, through its efferent fibres, so disturb the common centre of the two vagi as to cause either bilateral spasm or bilateral palsy of the laryngeal muscles.' As regards bilateral spasm, this position is quite unassailable, and in accordance with experimental evidence; but as regards palsy on the opposite side from the irritation it is different. 'Broadbent's law,' which is referred to by Sir G. Johnson as corroborating this theory, has, in fact, precisely the opposite effect. Broadbent's law is concerned with explaining (and it has very perfectly explained) the *exemption* from paralysis of muscles which normally act always together on the two sides when there is a centric unilateral lesion which otherwise might be expected to give rise to paralysis. To make this law cover the case supposed of a paralysis reflected from the seat of a peripheral unilateral lesion, so as to give rise to bilateral paralysis, is altogether at variance with the principles involved, and probably not supported by any precise experimental evidence."

2. The characteristic features of the *cough* in cases of pressure upon the nerve fibres supplying the larynx are in many instances easily recognized, and in some are so distinctive as to lead the trained observer at once to suspect the nature of the malady causing it. The cough is hoarse and imperfect; these are its two chief features, but in addition it may have other qualities, to which I shall presently refer. The cough is essentially a paralytic phenomenon. In place of the glottis being completely closed after the full inspiratory act, a chink is left so that the air within the trachea is not compressed sufficiently to produce the proper popgun cough explosion; this is explained by an observation easily made in the healthy larynx. When a person coughs, or rather prepares to cough, the laryngoscope shows that not only are the vocal cords approximated, but the margins of the ventricles of Morgagni are brought firmly together, and assist in forming a firm barrier to the exit of air until they are burst asunder by the forced upward draught from the trachea. The true cords form a valve, with the cusps facing upwards, while those of the false cords face downwards, and so when closed more efficiently oppose the upward passage of air. The true cords, if the abductors are paralysed, may form a valve against the entrance of air to the trachea. In cases of paralysis of the abductor muscles of the larynx, not only do the true cords fail to act, but in coughing the false

https://doi.org/10.1017/S1755146300167149 Published online by Cambridge University Press

cords are not approximated as just described. Hence the imperfect cough which is so distinctive of thoracic tumour, or of aneurysm pressing upon the recurrent laryngeal nerve.

Prof. Gairdner has long taught the importance of imperfect cough as a symptom of aneurysm ("Clinical Medicine," 1862). He was familiar with it long previous to the general employment of the laryngoscope, and he was able, by the careful use of the finger, to distingnish when the symptom was due to a primary laryngeal lesion, and when it was the result of paralysis of the laryngeal muscles. It is to his early observations, and to those of Dr. Wylie ("Edinburgh Medical Journal," September, 1866, and "Edinburgh Hospital Reports," Vol. I., p. 67), that we are indebted for the important position occupied by this symptom in the diagnosis of aortic aneurysm. Referring to cough and the associated group of laryngeal and tracheal symptoms, Sir Wm. T. Gairdner writes, in a lecture published in the "International Clinics," third series, 1895, Vol. IV., p. 45, "I have again and again made the diagnosis of aneurysm, provisionally at least, by means of this group of symptoms, without any other means of diagnosis, or especially of physical diagnosis, at all. Indeed, it is not necessary that you should even have said a word to the patient, or asked him a question. In some cases you may even make a very good guess at an aneurysm (when the facts are sufficiently characteristic) if the patient is within earshot, and you have not even seen him or known about his case otherwise, as I have occasionally pointed out in the wards, or long ago in dispensary practice, though I by no means advise you to be so satisfied with so guessing . . . and the symptoms . . . are not difficult to discriminate, and are of very great importance in prognosis as well as in diagnosis."

On several occasions I have been consulted by patients who complained of no symptom, laryngeal or other, beyond the cough, but its special characteristics were so distinctive that this symptom alone enabled me to form a provisional diagnosis of aortic aneurysm, which was supported by the physical signs found by the physician, and by the ultimate history of the case. The chief features of the cough are hoarseness and imperfection as to the explosion, but beyond these qualities there are other less significant peculiarities which require attention. It is generally a noisy, and not infrequently a harsh, "brassy" cough, or it may be what Dr. Wylie has described as "bovine cough"—like the cough of a cow. The cow "has no false cords or ventricles of Morgagni; its cough is, therefore, a long, loud grunt or wheeze, without any proper initiatory explosion." In some instances I have seen the qualities of cough just described, after being present for some months, disappear gradually, and remain absent for a time, to return again at a later date—this being probably due to a certain amount of compensation of the laryngeal muscles on the opposite side, and was in two cases coincident with improvements in the quality of the speaking voice, although to all appearances the laryngeal image remained the same as during the earlier course of the case.

In a gentleman examined by me four years ago, and who ultimately developed physical signs of aneurysm of the arch of the aorta, I observed

https://doi.org/10.1017/S1755146300167149 Published online by Cambridge University Press

a very peculiar form of laryngeal cough very early in the history of the case, before the physical signs were distinctive, and, therefore, led to considerable doubt as to the significance of the cough ; the movements of the left vocal cord were impaired, and the ventricular bands failed to reach the middle line during attempted phonation, but the voice was good, and at no time did the patient suffer from stridor. The cough was hard, brassy, and hoarse, but in place of one explosion, as in ordinary coughing, there was a kind of double explosion, one following immediately upon the other, the first being higher in pitch and harsher than the second. For this I could not find any reasonable explanation.

3. In cases of recurrent laryngeal paralysis the *speaking voice* may be little altered from the normal, because of the power of the muscles on the healthy side to compensate for the inefficient action of those of the paralyzed side. In other cases the voice may be impure, and during phonation, in order to throw the cords into tone-producing vibration, the air in the chest is subjected to abnormal pressure, and, as a consequence, if prolonged, the over-exertion of speaking speedily produces exhaustion of the patient's strength and inability to phonate, except in a very imperfect way. In cases of recurrent laryngeal paralysis the voice becomes altered in pitch, impure, and wanting in tone, and on straining to speak loudly the voice readily breaks into a falsetto, which may be of higher pitch than the ordinary voice. On the other hand, the voice may be lowered in register, hoarse, and muffled. The abnormal conditions of voice may be due simply to the imperfect movements of the vocal cords, or there may be other laryngeal or pharyngeal complications, such as acute or chronic catarrh, œdema of the mucous membrane, etc. I have seen more than one case where aphonia was the first and for some the only symptom of what at a later date proved to be aneurysmal pressure on the recurrent nerve. In a patient seen at the Royal Infirmary the voice began to fail three months previous to the time I saw him, and on inspection the left vocal cord was seen to remain stationary in the cadaveric position during attempted phonation or ordinary respiration. At this time he had not suffered from breathlessness, but within ten days he had a severe paroxysm of dyspnœa ; and in a private patient I have observed the same condition of voice for five months previous to other symptoms developing. In this case also the paralysis of the left vocal cord was apparently complete, and at no time was the breathing stridulous. While the alterations in voice are less characteristic, and hence less valuable than the other symptoms we have described as factors in diagnosis, still, when taken in conjunction with stridor and imperfect cough, they materially assist in forming an early diagnosis of aneurysm or of mediastinal tumour.

Dr. W. JOBSON HORNE. *The Pathogenesis and Earlier Clinical Evidence of Laryngeal Tuberculosis.*

In text-books on general medicine the isolation of diseases of the larynx and the singular crudity with which these diseases as a group, and laryngeal tuberculosis in particular, are expounded tend to create an early impression that the larynx stands by itself, that its interests are not

https://doi.org/10.1017/S1755146300167149 Published online by Cambridge University Press

intimately wrapped up with those of the body, that its study likewise stands by itself, and must be relegated to departments specially set apart for the purpose. Were the laryngoscope more frequently used in clinical medicine the important bearing the larynx may bring in diagnosis would be better appreciated.

To state that fifty per cent. of the cases of pulmonary tuberculosis under treatment are being overlooked may be a bold estimate, but it is one of which we are daily receiving confirmation. The dead-house teaches us that the stethoscope is deceptive, and that by the time the morbid signs are sufficiently established at one spot to permit of a diagnosis another lobe is usually already involved. Bacteriology is only too often negative at a time when a positive answer would be most useful.

It is unreasonable to suppose that in pulmonary tuberculosis the larynx would stand aloof, and give no evidence of the disease developing in the respiratory tract until the condition of "laryngeal phthisis" is established. True it is that mention is made of a pallor, or anæmia, of the larynx occurring at times in the earlier stages, that the voice may be lost for awhile from functional failure, but these changes are hardly regarded as coming within the range of a laryngeal tuberculosis, but rather as a part of a chronic laryngitis presenting nothing characteristic.

The idea that appears to be more widely held is that laryngeal tuberculosis, or laryngeal phthisis, as it is more commonly and unfortunately termed, may be summed up as infiltration and ulceration occurring in some twenty to thirty per cent. of advanced cases of phthisis, for which there is but little treatment which is palliative, and still less which is remedial. The term laryngeal phthisis is unfortunate; it helps to foster the idea that the disease is a separate entity, and it will presently be shown that laryngeal, apart from pulmonary, tuberculosis, can hardly exist. To restrict our views of laryngeal tuberculosis in this way is equivalent to taking up the study of consumption from the time when cavities have already formed.

Krause and Heryng have shown how even in the more severe cases much can be done to relieve the sufferings, if not to arrest the progress of the disease. But sufficient attention has hardly been given to the earlier recognition of a disease so insidious in its onset that it would seem to have no beginning, and yet so progressive in its character that when unheeded can have but one end.

To ascertain the earlier changes that occur in the larynx of a person smitten with pulmonary tuberculosis, and whether these changes are sufficiently constant and could be put upon a definite pathological basis and used in the diagnosis of the disease at a time when the lungs yielded no signs or only indefinite signs, were the main objects of a research commenced by the author some few years ago.

The present paper confines itself to the threshold of the disease, and can only be regarded as a brief sketch of a piece of work that has taken some years, and not as a complete summary of the results that have been arrived at. The first part is concerned with the pathogenesis, the portal of infection, the beginning and method of tuberculous destruction, the origin and formation of the giant cell. Although perhaps the larynx may

https://doi.org/10.1017/S1755146300167149 Published online by Cambridge University Press

undoubtedly be the most convenient site in the human body for studying pathological principles in either living or dead tissues, it is not suggested that the pathological changes noted are peculiar to the larynx. The second part of the paper deals with the clinical counterparts of these early pathological changes, and are held to be the earliest signs and symptoms not only of laryngeal but also of pulmonary tuberculosis.

The pathogenesis of the disease was worked at in larynges which *post-mortem* presented to the naked eye none of the usual signs of laryngeal tuberculosis, but which had been removed from bodies of persons who had died undoubtedly from tuberculosis. In the examination of these larynges, films were first prepared from the contents of the ventricles and stained for tubercle bacilli, the larynx was subsequently examined microscopically throughout.

The frequency with which the organisms were found within the ventricles of apparently healthy larynges would suggest that the importance of the ventricle as a harbour for tubercle bacilli is deserving of further attention.

Under the microscope the earliest changes noted were in the lymphatics, consisting of a proliferation of the parenchyma of the acini and ducts, with the formation of masses of small round cells, distending and choking the ducts and obliterating the glands, the adjacent and superficial structures at first remaining intact. These changes have been noted in the lymphatics situated in the submucous layer of the walls of the ventricles when a careful microscopic examination of the entire larynx had failed to reveal changes in the lymphatics in other parts. The nature of this cell proliferation was next considered. Is it to be regarded as nothing more than an outcome of a traumatism, or of some simple catarrhal process? or is it peculiar to the lymphatic structure of tuberculous patients? or is it to be attributed to one specific cause—namely, the presence of the tubercle bacillus, and to be regarded as tuberculous disease of the lymphatics? Dr. Horne said that, although he was not prepared altogether to accept or reject the theory that these lymphoid masses were due to a simple catarrhal process, he was now able to demonstrate the tubercle bacilli in the midst of some of them, and was of the opinion that, once having gained an entry into the lymphatic ducts, they acted as an irritant and caused a cell proliferation.

The *fons et origo* of the giant cell in tuberculosis next came under consideration, and by a series of lantern photographs of microscopic specimens its development from the wall of a lymph space was demonstrated. The series showed, first, the tubercle bacilli lying amongst the endothelial cells forming the wall of the lymph space; secondly, the fusion of the adjacent and divided cells; and, thirdly, the separation off of this plasmodial mass as a giant cell. Reference was made to the researches on lepra laryngis by Dr. Paul Bergengrün, of Riga, in which he had shown that the so-called "globi" were bacillary thrombi lying in dilated lymphatics, and that the lepra giant cell developed from the lymphatic endothelium. Although the sections Dr. Horne showed permitted the conclusions that had been drawn, he did not consider that they represented the whole story of the giant cell. If, then, as would appear

https://doi.org/10.1017/S1755146300167149 Published online by Cambridge University Press

to be the case from the foregoing, the tuberculous process commences in the lymphatics, it would be reasonable to suppose that those parts rich in lymphatics would be the more common sites for infiltration and ulceration, whilst such parts as are without glands would escape ulceration, except by continuity. This was clinically verified. The interarytenoid region, the posterior third of the cord, the ventricular band, and the epiglottis (especially the petiolus), are the parts more richly endowed with glands, and these are the parts more commonly ulcerated in laryngeal tuberculosis. The cord itself—that is to say, the functionally more important part of the cord, namely, that part lying between the two small cartilaginous bodies—being free from glands, escapes ulceration, excepting superficial erosion and by continuity. A proliferation of the subepithelial blood vessels was demonstrated as taking place side by side with the cell proliferation.

With regard to the muscles, changes were described as occurring at an early stage in the fibres, and traced to the glands lying in close proximity. These changes are brought into evidence by an early functional failure, which is mainly myopathic in origin.

The earlier clinical evidence of laryngeal tuberculosis, the clinical counterparts of these early pathological changes, were next described. Whilst clinically investigating these, it was necessary—inasmuch as only slight departures from the normal were being looked for—to retain a control mental image of a normal larynx. This was done by constantly examining a large number—some hundreds in all were examined—of larynges in healthy subjects, or, at least, presumably free from tuberculosis. And it was whilst in search of normal larynges that some important aids to the present research were stumbled upon. The larynx was examined in three hundred and fifty-nine consecutive cases of pulmonary tuberculosis, or cases in which there were reasons for suspecting pulmonary tuberculosis. The larynx was also examined in a large number of patients suffering from pulmonary diseases other than tuberculosis.

The points to which attention was mainly directed were the following: (1) disturbances of sensation, hypæsthesia, hyperæsthesia, paræsthesia; (2) colour changes, anæmia, hyperæmia; (3) functional disturbances; (4) impaired movements of the vocal cords, apart from paralysis; and (5) changes in the contour of the larynx due to slight œdema. Inasmuch as disturbances of sensation are subjective, too much significance could readily be attached to them, if taken alone, as a clue to pulmonary tuberculosis; but, in regarding them only as evidence of "hysteria," one is apt to miss important clues to pulmonary tuberculosis.

Hypæsthesia was not infrequently associated with anæmia of the soft palate; and it might be stated that, unless the subject is wilfully hostile to laryngoscopy, the more pallid the soft palate the greater the tolerance to laryngoscopy; and when in the course of the pulmonary condition the anæmia passes off, the tolerance often goes with it.

Anæmia of the laryngeal mucosa was noted as present in varying degrees in one hundred and fifty-seven out of three hundred and fifty-nine cases. At times it was universal. When patchy, it was confined to points where the mucosa was more easily exposed to tension, occasioned by a

https://doi.org/10.1017/S1755146300167149 Published online by Cambridge University Press

slight œdema in association with the proliferation of subepithelial vessels. If was as frequently met with in men as in women.

Hyperæmia was noted in one hundred and seventeen out of three hundred and fifty-nine cases, and rather more frequently in males. This hyperæmia must be regarded as a feature distinct from and not amounting to acute laryngitis. Acute laryngitis did not occur so frequently as one might previously have anticipated. The hyperæmia was often transient, within a few days giving way to pallor; but at times it was most persistent. These phenomena were also attributed to the proliferation of subepithelial vessels.

Disturbances in the vocal function were most frequently met with, often transient, not amounting to more than a weakness of voice or loss of tone. The production of voice called for a greater effort. There was a forgetfulness of office—a sluggishness of one or both cords to act; so that there was phonatory waste before voice was produced. And in connection with this required effort it may be mentioned that, in early tuberculosis, when the speaking voice is weak the singing voice may be quite clear. Then the singing voice may suddenly go. This causes the patient to seek advice; and the possibility of commencing tuberculosis should not be forgotten. Transient dysphonia was more commonly met with amongst women, and especially young married women, during pregnancy.

Not infrequently in cases of so-called "hysterical aphonia" thoracic signs of pulmonary tuberculosis had been subsequently made out, and the possibility of diagnosing "hysterical aphonia" in the presence of pulmonary tuberculosis should be thought of. Impaired movements of the vocal cords, apart from paralysis, were described, and considered to be myopathic in origin and due to the changes noted in the muscle fibres. With regard to transient œdema of the laryngeal mucosa, the contour in a large number of larynges in early pulmonary tuberculosis when compared with that existing in a normal larynx was found to be partially lost, more particularly in the interarytenoid and arytenoid regions along the ventricular bands, and at the base of the epiglottis. A fine crenating or fringing occurs upon the folds of mucous membrane in the interarytenoid space, not sufficient to be called an excrescence, but met with sufficiently often in pulmonary tuberculosis as to be deserving of attention. An early but transient loss of symmetry in the outline of the arytenoid eminences was noted. The ventricles become less patent, and this loss of patency was commonly associated with some œdema of the inner wall of the ventricular band, which was ascertained clinically in endeavouring to pass a curved platinum loop into the ventricle in search of tubercle bacilli. A slight œdema of this part of the ventricular band, together with an enfeebled action of the compressor sacculi laryngis, must effectually retain bacilli-laden sputum within the ventricle when once it had been lodged there in the act of coughing. Moreover, it was pointed out that the choking by a cell proliferation of the ducts of the muciparous glands opening into the ventricle, the closure of the ventricle by the œdema secondary to the proliferation of vessels, and the failure of the compressor sacculi laryngis through changes in the muscle fibres

https://doi.org/10.1017/S1755146300167149 Published online by Cambridge University Press

to discharge its contents, all combine to deprive the cords of the mucus necessary for their lubrication. The cords lose that semi-translucent mother-of-pearl sheen, and present an opaque pallor more approaching a dead ivory white. With insufficient lubrication superficial erosions occurred ; this did not amount to ulceration, as met with elsewhere in the larynx, inasmuch as the essential element was absent, namely, the lymphatics.

[A lantern demonstration was given of photographs of the microscopic sections on which the conclusions were based, and some of the more important sections were shown under microscopes.]

Dr. HAMILTON A. BALLANCE. *A Case of Septic Thrombosis of the Lateral Sinus, due to Streptococcus Infection. Secondary Abscesses. Operation. Recovery.*

L. B., aged twenty-four, pregnant five months with her first child, was admitted into the Norfolk and Norwich Hospital, November 30th, 1897.

History.—The present illness dates from three weeks previous to admission, when pain in the left ear, frontal and occipital headache, and sickness began. The pain has increased in severity up to the present time. The sickness has been frequent but not every day of the illness. She was repeatedly sick on Nov. 28th.

Four or five days from the first onset of the illness, she had a shivering attack lasting about ten minutes. She has had about six of these since, and the last one was on Nov. 26th.

On Nov. 23rd, that is, one week before admission, a discharge of offensive matter began from the left ear. The patient is positive that there had never been any previous discharge, and on carefully questioning the mother she is also quite certain that her daughter had no discharge from either ear during childhood. The right ear has never given any trouble, and the hearing with it is normal.

The patient suffered from measles in childhood, but not from scarlet fever. She has been in bed for a fortnight.

Condition on Admission.—The patient is rather a wasted woman of twenty-four, with sallow complexion, and looks very ill. Temp. 98·6°. Pulse 100. There is profuse local discharge from the left ear, with some swelling and œdema over the mastoid. On examining the meatus, the posterior wall is greatly swollen, reducing the canal to a narrow chink, so that the membrana tympani cannot be seen. In the centre of the posterior wall there is a discharging sinus lined by prominent granulations, and a little pus can be made to ooze from this by pressure over the upper part of the mastoid. This process is tender, but there is neither swelling nor tenderness below the mastoid in the neck.

There is marked double optic neuritis, more intense on the left side. Pupils, moderate size and react. There is no paralysis anywhere, nor loss of sensation. Power of flexion of the knees is good. Both knee jerks are much exaggerated, rectus jerk and ankle clonus being also obtainable on each side, more marked on the right.

The patient lies by preference on the right side.

Dec. 2nd. This afternoon the patient had a shiver, and the temperature

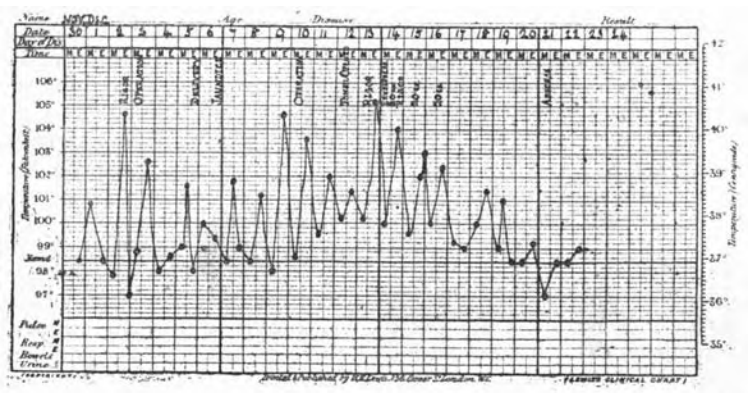
https://doi.org/10.1017/S1755146300167149 Published online by Cambridge University Press

rose to 104·6°; pulse, 134. I slit the sinus in the posterior wall of the external auditory meatus to give freer vent to the pus.

Dec. 3rd. As the temperature was again rising, although no further rigor had occurred, longer delay was contra-indicated, and the mastoid was operated upon.

About two drachms of pus were found on the surface of the bone, and as the membranous meatus was turned forwards out of the osseous meatus a hole was seen in the posterior wall of the latter exactly corresponding to the sinus previously mentioned as present in the external auditory canal. A seeker inserted into this hole passed directly into the antrum. The antrum, attic, and tympanum were then thrown into one cavity by the removal of their outer walls, and much pus and cholesteatomatous *débris* cleared away.

In removing the posterior part of the mastoid, two thin white sequestra, about one-quarter of an inch in diameter, were taken away. Immediately two or three drachms of pus in a sudden gush escaped from the



lateral sulcus, these sequestra having evidently formed part of the groove. The lateral sinus was then exposed for one and a half inches by the removal of bone with cutting forceps. Its outer wall had sloughed, and I pulled out from the interior of the sinus a cylindrical slough, one inch long, the remains of the infected thrombus.

After exposure of the internal jugular vein by an incision at the anterior border of the sterno-mastoid, the vessel was divided between two ligatures above its junction with the common facial. It was not thrombosed, but collapsed owing to the fact that there was no blood coursing down its upper part. The lower ligatured end was allowed to retract, but the upper one with the ligature upon it fixed in the upper angle of the neck wound, which was then entirely closed.

When all visible disease had been removed from the mastoid region and the wound cleaned it was stuffed with gauze, after a horizontal division of the membranous meatus posteriorly, to allow a large auditory canal to permanently form.

Dec. 5th. The patient has been much relieved since the operation, and, although the temperature reached 101·6° to-day, it was normal the

https://doi.org/10.1017/S1755146300167149 Published online by Cambridge University Press

whole of yesterday. This evening the patient was delivered of a still-born child without difficulty. It was not offensive, and had probably died during the septic illness of the mother.

Dec. 6th. Jaundice is commencing to-day.

Dec. 7th. Patient has still a temperature of 101-2° every day, but general condition is improved; pulse, 100-30. The ligature on the upper segment of the divided internal jugular was taken off this morning, and the vessel allowed to remain open at the upper end of the wound in the neck. No pus exuded from it on removal of the ligature. The mastoid wound is getting more healthy. The liver is not felt. The lungs are uninvolved, nor are any joints affected.

Dec. 9th. There is a little swelling and tenderness in the upper part of the neck just below the mastoid, and about one drachm of sero-purulent fluid escaped from the open end of the internal jugular vein. There is slight double tonsillitis.

Dec. 10th. The temperature reached 104·6 yesterday evening, but without a rigor, and as it was thought that pus might be pent up in the depths of the upper wound, at the junction of sigmoid sinus and internal jugular vein, the patient was again put under chloroform and this region explored by the further removal of bone, but no collection of pus was found. The wound in the neck had, however, become infected by pus running down the jugular, so all the stitches were removed, and it was left widely open and stuffed with gauze.

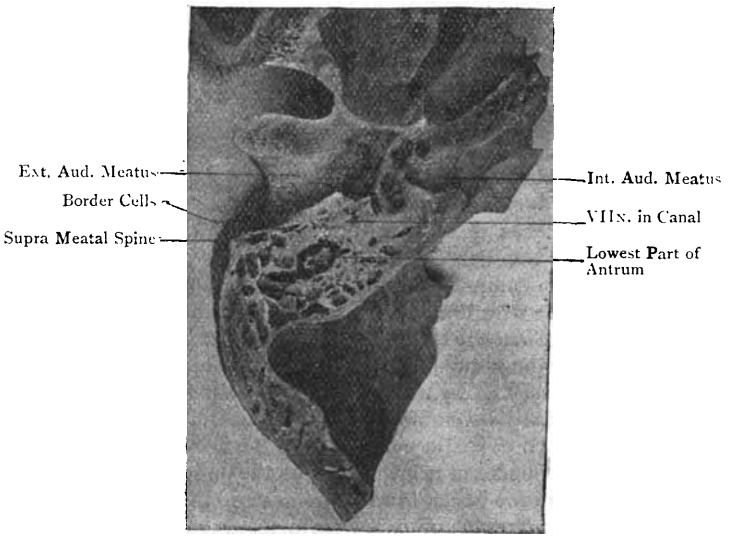
Dec. 12th. The last two days the patient has been greatly distressed owing to acute tonsillitis on the left side. She has had intense pain with great swelling of the tonsil and uvula, and has been able to swallow nothing at all. This morning I opened the tonsil and let out one to two drachms of creamy pus. The patient nearly lost her life about this time from cocaine poisoning, when a ten per cent. spray was used for the throat, preparatory to incising the tonsil. She became unconscious, with ghastly pallor, widely dilated pupils, imperceptible pulse, rapid respirations, and clonic convulsions, but she ultimately came round.

Dec. 14th. Since opening the tonsil the throat has improved, the patient can now swallow and the voice has returned. Last evening there was a rigor of five minutes' duration, and a temperature of 105·2°. This morning there is a butterfly patch of facial erysipelas over the bridge of the nose and inner part of each cheek, quite away therefore from the wound behind the ear. The patient was at once isolated and given twenty cubic centimètres of streptococcus antitoxin. Another rigor of ten minutes' duration, with a temperature of 104°, occurred this evening.

Dec. 16th. The patient has had two more doses of antitoxin yesterday and to-day, and the temperature is gradually falling. The rash has spread over the whole of both cheeks, but the colour is fading. During the course of the erysipelas the patient has been extremely prostrate; pulse, 130 to 140, and has had much bilious vomiting. The tongue is dry, and the urine shows a cloud of albumen. The jaundice continues about the same, but has never been intense. The patient has been so weak, the last day or two, that it was quite impossible to say whether or no she would last another twenty-four hours. The neck wound is less

https://doi.org/10.1017/S1755146300167149 Published online by Cambridge University Press

small sinus now existing in the calf; but to-day the temperature suddenly rose to 103·6°, with a sense of chilliness and some nausea. The patient complains of pain in the right calf, which is rather swollen.



Horizontal Section of Left Temporal Bone of Adult, through upper part of External Auditory Meatus: Membrana Tympani and Ossicles removed.

March 28th. As seen in the chart the patient has passed through a period of severe pyrexia, due to an attack of erysipelas in the right leg, starting from the unhealed sinus in the calf. Swelling and redness extended from the hip to the ankle, with much pain in the limb. Eighty cubic centimètres of antitoxin were used throughout the attack, but its immediate effect on the temperature was not evident. Now, however, there is no fever, and it seems unlikely to return; the patient is much better, and the swelling is gradually subsiding. The sinus has had to be slit up a little, to enable some pent-up pus to find its way out.

April 21st. The temperature remained down until the last few days, and as it seemed the leg did not discharge freely enough, I opened up the calf under ether to-day, by a four inch incision, down to the deep muscles, and kept the wound widely open by gauze packing.

May 29th. The leg is healing healthily. The left ear has been quite dry for some weeks, and the site of the operation behind it is all depressed and soundly scarred over, leaving no fistula. The neck has quite healed. The watch is only heard on the left side on contact. The optic neuritis has entirely disappeared, and the urine is normal, free from albumen and pus.

June 25th. Went to convalescent home. In very good health, except for some loss of flesh. Leg almost healed.

https://doi.org/10.1017/S1755146300167149 Published online by Cambridge University Press

Remarks.—This case seems to me to illustrate the fact that these patients do well even in the presence of secondary foci if the primary source of infection is thoroughly dealt with and cut off. To recapitulate for a moment the conditions which were met with here :—

Sinus thrombosis, with subdural abscess.
Premature delivery.
Suppurative tonsillitis.
Two attacks of erysipelas.
Jaundice.
Abscesses in the shoulder, leg, and kidney.

Pathology.—Pure cultivations of the streptococcus pyogenes were obtained from all the foci from which tubes were inoculated, namely, mastoid, neck, shoulder, and calf, but in the mastoid, in addition, there was present a bacillus, but this appeared quite an accidental contamination, and not of a pathogenic nature. Hence it was obviously a rational procedure to give the streptococcus antitoxin here, and a true test of its value, as contrasted with the indiscriminate use to which it has been put in any temporary pyrexial state, such as that met with in the puerperium, and often due to a mere sapræmic condition, and dependent on other organisms rather than the streptococcus.

It is interesting to note that, although the streptococci obtained entrance into the blood-stream, as evidenced by the secondary abscesses, yet none settled in the uterine wall, which had undergone the traumatism in connection with delivery ; and also that cutaneous erysipelas—which, likewise, is dependent on the streptococcus—complicated this case on two occasions.

Now, the streptococcus is usually associated with virulent and rapidly-spreading inflammatory conditions ; but the abscess in the calf, although due to this organism, was characterised by its extremely slow progress, the calf hardly being perceptibly larger at the end of a month of the abscess than at the end of the first week. I believe this to be due to the effect of the antitoxin administered during the course of its formation.

After noting the organism obtained from the abscess, I forwarded an agar culture to the Clinical Research Association ; and their report was to the effect that the tube contained "an extremely feeble growth of pure streptococcus pyogenes." The Association were quite unaware, when they sent their report, that the patient had been treated with antitoxin. Its effect, I believe, was to inhibit the growth and attenuate the virus of organism present. The possibility that the second attack of erysipelas was due to a different variety of streptococcus may be mentioned ; and this may explain the severity of the attack, although antitoxin was administered. I have never seen, however, an erysipelas of the face subside so quickly as it did in this patient.

The position of the fistula in the posterior wall of the external auditory meatus, as in this case, is extremely rare. The usual site for bony perforation in mastoid disease is, as is well known, on the outer aspect of the process—especially in young people. But if the mastoid disease spreads

https://doi.org/10.1017/S1755146300167149 Published online by Cambridge University Press

to the "border cells," in the process, which abut on to the external auditory canal, then perforation into this channel may occur. In their work on mastoid abscesses, Broca and Lubet-Babon have found only three such instances in one hundred and forty-six mastoids operated on by them—*i.e.*, two per cent. I have shown, in the photograph, the position of these "border cells" in a horizontal section that I made in the temporal bone of an adult. It will be noted what a thin layer of bone separates some of them from the surface.

I have during the last few weeks had another case of suppuration in these "border cells" in a man of fifty, who was admitted into the Norfolk and Norwich Hospital, with septic meningitis, secondary to otitis media, of one month's duration. The meningitis was the result of the infection spreading through to the internal ear, and then along the internal auditory meatus to the meninges; but in operating on the mastoid, a collection of pus about a quarter of an inch in diameter was found in the cells adjacent to the meatus, but the antrum was free. The man subsequently died with purulent lepto meningitis on both sides and over both base and vertex of the brain.

Diagnosis.—This patient had the headache, vomiting, repeated rigors and optic neuritis of sinus thrombosis, as well as the local signs of mastoid disease; but the history of discharge for only one week, which I made out to be quite genuine, certainly put me off the correct diagnosis at first.

However, sigmoid sinus thrombosis is known as a rare complication of acute otitis media, and MacEwen mentions a case in which it occurred after only eight days of aural discharge, complicating a case of diphtheritic sore throat.

Again, Erskine, of Glasgow, in 1890, described a case of sinus thrombosis in a man of seventy-two, where there was no perforation of the membrana tympani at all—although disease had long been present, and progressing insidiously had at last involved the sinus, after producing extensive caries in the interior of the bone.

In my case there must have been long existing otitis because of the sequestra found in the mastoid, but this had gone on without external discharge of pus until one week before admission.

Dr. JOHN MIDDLEMASS HUNT. *On the Relation of Fibrinous Rhinitis to Diphtheria.*

At a meeting of the British Laryngological Association in November, 1891, I read notes of a case of fibrinous rhinitis, and showed casts from the nasal passages. The case was in every way a typical one, as the patient remained in good health, had no albumen in the urine, no glandular enlargement, and no subsequent paralysis. The discomfort arising from the nasal obstruction, and the profuse watery discharge, were the only symptoms.

I should mention that no bacteriological examination was made, and the diagnosis rested entirely on the clinical history of the case.

Since then I have met with three cases presenting the clinical characters of fibrinous rhinitis, but all of them have been so related to

https://doi.org/10.1017/S1755146300167149 Published online by Cambridge University Press

diphtheria as to make me thoroughly distrust any case based on clinical evidence alone.

I propose to bring these other cases briefly before your notice.

The first was that of a medical man, practising near Liverpool, whom I saw on February 15th, 1894. He was in good health, and came to me on account of distressing nasal obstruction with abundant watery discharge from the nostrils, which had troubled him for about a week. He had not been attending any cases of diphtheria, but thought he might have conveyed some poisonous material to the nasal cavities while performing a *post-mortem* examination in a case of ulcerative endocarditis. On examination I found both nasal passages lined with well-formed, thick, white membrane, which left a bleeding surface when torn off with forceps.

From the complete absence of all constitutional symptoms, and remembering my former case, I assured the patient he was only suffering from fibrinous rhinitis, and that he would be all right in a few weeks. I did not see him again, and unfortunately no examination was made of the membrane I had removed.

It was six months later before I learned that a fortnight after his visit to me, and before the nasal trouble had gone, membrane appeared on the tonsils, and he went through a severe attack of diphtheria followed by extensive paralysis. I need hardly say that I have not been so confident about the diagnosis of fibrinous rhinitis since.

My second case was one of a little girl, who came to the Royal Infirmary on March 19th, 1896, complaining of nasal obstruction, which had been present about a week. On examination both nasal cavities were found to be lined with membrane, which was thinner than in the former cases, and more friable, so that it was not possible to remove a large piece with the forceps. The mucous membrane underneath was raw and bleeding. There was no fever, no albumen in the urine, no glandular enlargement, and no deposit on the tonsils or pharynx.

Portions of the membrane were sent to Prof. Boyce's laboratory, where cultivations and inoculation experiments were made. The report was that the inoculations gave a negative result, and the cultures showed streptococci and staphylococci, but no Lœffler bacilli.

Subsequent inquiry showed that the patient had suffered from "tonsillitis" a month or two before, followed by paralysis of the palate, of which there was still some evidence when she came under our notice. We also found that several other children, who were associated with her at that time as "fairies" in a local pantomime, had been laid up with bad throats.

The third case was one I saw in November, 1896. The patient, a little girl of ten, was sent to me by the family medical man as a case of chronic nasal catarrh. She complained of obstruction of, and discharge from, the left nostril, which had begun with a cold in the head eight weeks before. She was then for one day in bed, but since had been running about without any complaint except the nasal discharge. During these eight weeks she had never any sore throat.

On examination there was a recent adhesion to be seen between the

https://doi.org/10.1017/S1755146300167149 Published online by Cambridge University Press

anterior end of the lower turbinal and the septum, and behind were some patches of distinct membrane with muco-purulent secretion. There were no enlarged glands, no albumen in the urine, and no paralysis.

On inquiry I found that a week after this patient began to have the nasal trouble a servant in the house had a bad sore throat, and was in bed for eleven days, and that two weeks later a brother, aged two years, died of croup. The servant was subsequently a patient at the Royal Infirmary with diphtheritic paralysis.

Thus, in four cases bearing the clinical characters of fibrinous rhinitis, three were directly associated with diphtheria. In one case pharyngeal diphtheria began in the third week of the nasal trouble ; in another the rhinitis occurred some weeks after convalescence from an attack of diphtheria ; and in a third it was followed within a few weeks by two cases of genuine diphtheria in the same family.

Now, it may be said that of my four cases only the first was one of fibrinous rhinitis, and all the others were cases of nasal diphtheria. But is there any way of distinguishing these two diseases clinically ? Formerly it was thought that primary nasal diphtheria was a very rare disease, and that it was almost always attended by severe constitution symptoms. We now know that in the form of fibrinous rhinitis it is not at all uncommon, and often runs its course with little or no constitutional disturbance

If we turn to the text-books for the clinical characteristics which separate the two diseases, we find Bosworth relies upon one symptom, which he says is pathognomonic of fibrinous rhinitis, namely, that "the false membrane is easily detached from the mucous membrane beneath it without causing hæmorrhage." This, I need hardly say, is contrary to the experience of nearly every other observer. Another American writer, who has seen many cases of fibrinous rhinitis, Dr. Hamilton Potter, says, "the membrane is firmly attached to the parts beneath, and is only detached with violence, leaving a bleeding surface."

Dr. McBride is of opinion that "the intense local discomfort, together with the absence of all constitutional symptoms in typical cases, form an easily recognized combination." As Dr. McBride had only seen one case when he wrote these words he would probably modify them now as the result of extended experience.

Dr. Walsham writes, in his recent book on nasal diseases, "The points to be chiefly relied on in distinguishing fibrinous rhinitis from nasal diphtheria are the absence of constitutional symptoms, of glandular enlargement, of albumen in the urine and of paralytic sequelæ." But it has been repeatedly shown that all these symptoms may be absent and yet the Lœffler bacillus be present in the membrane.

I am afraid there are no clinical characters on which we can rely in distinguishing the two diseases, and our one method is to turn for help to the skilled bacteriologist. The results of bacteriological examination have been to demonstrate the presence of Lœffler's bacillus in an ever-increasing proportion of cases of so-called fibrinous rhinitis.

One of the most valuable contributions to this subject is the paper of Dr. Edmund Meyer in the "Archiv. für Laryngologie," Band IV., Heft 2. In twenty-two cases of fibrinous rhinitis, in which he made bacteriological

https://doi.org/10.1017/S1755146300167149 Published online by Cambridge University Press

examinations with inoculation experiments, he found in nine cases streptococci and staphylococci only, while in thirteen others the Lœffler bacillus was present in virulent form. He also remarked that in their clinical course the cases with diphtheria bacilli showed no difference from those without. He was of opinion, too, that the cases in which the Lœffler bacillus was absent were those in which the disease had been present for a long time, and that the bacillus might have been found in them also if examined earlier.

Prof. Kanthack speaks still more strongly in Allbutt's "System of Medicine." He says, "A curious and important pathological condition is the so-called rhinitis fibrinosa. Clinically such cases are not diphtheria, but pathologically and bacteriologically they are so; in the cases I personally examined I obtained, as others did, large numbers of virulent diphtheria bacilli."

I might venture to summarise our present position regarding fibrinous rhinitis to be as follows :—

1. While admitting that other bacteria besides the diphtheria bacillus may give rise to membranous exudation in the nasal passages, the vast majority of cases of fibrinous rhinitis are due to the Lœffler bacillus.

2. That it is impossible on clinical grounds alone to distinguish fibrinous rhinitis from mild nasal diphtheria.

3. That all cases of fibrinous rhinitis should be regarded as diphtheria until the contrary has been proved by reliable bacteriological investigation.

Dr. PEGLER. *On Bilateral Tumours of the Septum—Lymphoid and Erectile.*

The object of the paper was to call attention to certain simple growths of the septum nasi which had not apparently been hitherto described in rhinological literature.

The tumours were not interesting pathologically only, but were important factors in the causation of

(1) Nasal obstruction;
(2) Paresis of the soft palate;
(3) Affections of speech (sigmatic dyslalia).

Of the lymphoid bodies, the writer's experience had been limited to a single case (some particulars of which have already appeared in this Journal), in which the growths, being attached by a broad tough pedicle on either side, the septum, about three millimètres from the posterior free border, projected into the naso-pharynx and caused almost complete nasal obstruction. They were oval in shape, pale in colour, and mammillated on the surface. Microscopically they consisted solely of lymphoid tissue, encapsuled by ciliated epithelium; lymphoid follicles crowded the entire field of section. There were no concomitant adenoids in the naso-pharynx, but large moriform hypertrophies grew from the inferior and middle turbinates. Some light might, perhaps, be thrown upon the origin of these lymphoid tumours by a study of the histology of the erectile bodies about to be described.

These appeared as parallel longitudinal ridges, one in each nasal

https://doi.org/10.1017/S1755146300167149 Published online by Cambridge University Press

cavity, pink, very broad based, lobulated at intervals, and extending almost the entire length of the septum downwards and forwards at the level of the tubercle.

Microscopically, they were chiefly composed of the normal erectile tissue of the septum, but were remarkable for exhibiting masses of lymphoid cells ranged at intervals close to the periphery in the lymphoid area. They looked much like isolated bodies—true follicles—at first sight, but, as Wingrave had remarked, they were evidently more correctly to be regarded as transverse sections of rod-like prolongations from the subepithelial lymphoid layer of the septum. Might this tendency to outgrowth on the part of the lymphoid element of the septal tissue help to explain the occurrence of the previously mentioned bodies which consisted of lymphoid tissue solely? The etiology of these longitudinal masses was obscure. In Dr. Pegler's case they were associated with extensive ethmoidal disease, and very large granulation polypi, and until these had been partially removed the septal growths were practically obscured. But in a somewhat similar one recently exhibited at the Laryngological Society of London by Arthur Cheatle the concomitants were polypoid hypertrophies of the middle turbinal and paroxysmal sneezing. Wingrave's suggestion that the tumours might be regarded as hypertrophic overgrowths of the tubercle of the septum was a good one, and, whatever the fact might be worth, it was true that longitudinal mucous membrane ridges (representing the tubercle?) were normal appendices to the septum in certain lower mammals.

The *treatment* of the lymphoid tumours had been removal by the cold snare and spoke-shave, aided by a finger in the naso-pharynx. It had required several sittings to remove those described owing to their toughness, but proneness withal to elude the grasp of the instruments. The septum when exposed by their dislodgment appeared at least three times its normal thickness.

The *erectile growths* were successfully detached with a curved probe-pointed tonsil knife, the snare being used to engage what had escaped abscission.

Dr. PEGLER. *An Operation for the Removal of Deformity arising from Fracture of the Triangular Cartilage of the Nose.*

The patient was a male, aged twenty-seven, who had received a blow from a cricket ball a year ago (July, 1897), the ball striking the nose from below.

He gave the following history:—

There had been a great deal of swelling, and also discharge from the nostrils in consequence of the injury. The swelling subsided, but the discharge continued; and he was admitted as in-patient in a provincial hospital. The ridge of the nose was at this time swollen (boggy?) and red. Some operation was performed, and an india-rubber splint was placed in each nasal cavity.

When the patient consulted the writer in June, 1898, there was a scar along the dorsal surface of the nose from the root nearly to the tip. The bridge was thrown into especial prominence on account of the

https://doi.org/10.1017/S1755146300167149 Published online by Cambridge University Press

depressed and sunken position of the triangular cartilage, which, together with the lateral cartilages, were detached in some way from the nasal bones. The deformity was so considerable as to interfere seriously with the patient's prospects in business; and he was willing to undergo any operation that might be proposed for his benefit.

On examining the interior of the nose, a prominent cartilaginous spur, shaped like a spine, obstructing breathing considerably, was seen in the left nasal fossa, but there was nothing specially observable on the right side. The finger, introduced into the nasal cavities, detected what appeared like a splitting of the anterior margin of the triangular cartilage into two lateral portions, which could be squeezed together, but only to separate again when the pressure was taken off. Speculation as to the explanation of this was of little use, as it was evident that the dislocation of the triangular cartilage was accompanied by a general smash up of the non-bony structures; and, in addition, it had been followed by some surgical manipulations. The prominent and pointed cartilaginous spur in the left cavity bore testimony to the force of the original injury.

The patient was shown to the Laryngological Society at its June meeting, and some valuable suggestions were made by the members as to the best way of dealing with the deformity.

On June 17th, Mr. Walsham was asked to see the case in consultation; and, in consequence, an operation was performed on June 22nd. Dundas Grant's suggestion as to the removal of the nasal prominence was carried into effect; but the writer has to express his obligations to Mr. Walsham for many others of great ingenuity, as well as for his valuable assistance at the operation.

Chloroform having been administered, an incision was made along the middle line of the nose, following the original scar, to within a quarter of an inch of the tip. The integuments were dissected back, close down to the bone and cartilage, and held aside by retractors.

The next step consisted in threading a rather stout silver wire through the depressed cartilages from side to side, incorporating the two surfaces that appeared to have separated, and taking care not to encroach upon the mucous cavities by feeling with the tip of the little fingers that the needle did not enter them. The cartilage was now under control, and could be raised by the wire at will.

The second half of the operation consisted in sawing through the projecting angle of the nasal bones from above obliquely downwards and forwards until the cartilaginous articular surface was reached, and at this point the detached piece (which consisted in reality of two portions, a larger on the right, chiefly cartilage, and a smaller, bony, on the left) was turned down, and made to assist in filling up the hollow below. A hole was next drilled through the nasal bone, above the sawn surface, with a centre-bit, and one end of the wire that had been employed to perforate the triangular cartilage was carried through it. In this way gentle upward traction could be made upon the depressed portion, so as to cause it to regain to some extent its old position.

The ends of the wire were next brought together, twisted over the turned-down fragments of bone and cartilage, and trimmed, so as to lie

https://doi.org/10.1017/S1755146300167149 Published online by Cambridge University Press

quite flat on the bridge of the nose. The surface having been thoroughly cleansed with antiseptics, the flaps of skin were united by about half-a-dozen catgut sutures, and sealed down with a painting of collodion.

There was a little inflammatory reaction during the first two or three days, with some œdema in the immediate vicinity of the wound, but this quite subsided under ice bags. When in a week's time the dressings were removed, the upper and lower portions of the wound were found healed by first intention, but the middle stitch or two had burst through, and some pus was escaping. A dressing of red lotion caused the hollow to granulate up, and an injection of Dobell's solution added a great deal to comfort, and to the breathing facilities of the nose. A day or two later the patient was given a clip to wear, which helped the plaster in keeping the edges together. A small central granulation, diminishing in extent every day, was all that remained at the time of writing this abstract.

The tension upon the wires being very considerable, and the pressure upon the turned-down fragments (whose blood supply cannot be great), opens a question as to the ultimate fate of the latter, and as to how long the present improved position of the depressed cartilage may be expected to last. But in any case it seems to the writer that a line has been struck out in the treatment of a rather common deformity, which, with suitable modifications and further improvements in subsequent cases by other surgeons, may lead to a useful operation.

Dr. G. JACKSON. *On Curetting for Suppuration in the Middle Ear, illustrated by a Case of Supposed Malignant Growth.*

It is scarcely necessary at this time of day to insist on the necessity of curetting the middle ear in cases of obstinate suppuration, formation of granulation tissue, bone affection, and the like, but I venture to bring the following case before you as illustrating its desirability, even after the return of growth, which had some appearance of malignancy. I saw the case first in May, 1896, with Dr. R. Simpson, of Plymouth, who has furnished me with the following history.

The patient, a lady, aged about fifty, had, in May, 1894, symptoms of labyrinthine hæmorrhage, produced apparently by violent sea-sickness, which left her with tinnitus, and loss of bone conduction on one side—the left. Not long after this she was in an accident, due to the giving way of some planks of a platform, and her ears got filled up with lime and mortar. It is probable that this was not properly removed at the time. She did not complain of any other symptom than the tinnitus and deafness before referred to, until February, 1896, when she began to get pain in the ear. One day, when in a stooping position, the ear suddenly burst out bleeding ; the bleeding relieved the pain for a time, then it returned. In a few days she had a second attack, which again gave her relief. When examined, after this, by Dr. Simpson, he found the ear (meatus) full of growth.

When I saw her in May, 1896, there were polypoid granulations in the meatus of the ear, with discharge, and very defective bone conduction. As local treatment had been of no avail, I removed all the granulations by curetting, in June, 1896. There was very considerable

https://doi.org/10.1017/S1755146300167149 Published online by Cambridge University Press

thickening of the tissue of the external meatus, near the orifice on the inferior wall, possibly overgrowth of the sebaceous glands ; this I also removed. In spite of subsequent treatment, careful syringing, packing with gauze, the application of chromic acid, galvanic cautery, etc., the growth returned, and in July I again removed it, and also scraped out a sinus which had formed behind the ear, following an abscess, which formed over the mastoid process, just after the first operation. By September, 1896, the sinus was closed, and the tympanic cavity nearly healed, and discharge stopped. I might say that the locality she lived in was damp. The improvement took place whilst away from home. On her return the growth again started, and developed rapidly, in spite of treatment by chromic acid, galvano-cautery, etc.

At this point she consulted Sir William Dalby, who suspected malignancy, and suggested if any further operation was done that it should involve removal of a considerable portion of the mastoid. I might say that the sinus had opened up again. A week after she saw Sir W. Dalby she had two serious attacks of facial erysipelas, beginning at the affected ear, and spreading all over the face. In May, 1896, she removed to a healthier locality, and I performed a third operation, completely curetting out the middle ear and meatus, and also opening up the sinus behind the ear, and introducing a drainage tube through the sinus into the meatus, between the cartilaginous and bony meatus, and bringing it out at external ear. By careful after treatment the discharge stopped, and the whole thing healed up. I should say that a small portion of the growth was examined microscopically before the final operation, and was pronounced to be merely granulation tissue, and probably of a tubercular nature. Subsequently to the operation a larger piece, removed at the time from the deeper part of the meatus, was examined, and was stated to contain evidence of malignancy. I may say that at the present time she is quite well, and there is no return of the growth, although, as might be expected, the tinnitus and deafness continues.

Cancer of the middle ear is rare. The starting point of epithelial new formation is most frequently the auricle and the external meatus, less frequently the tympanic cavity and the mastoid process.

According to the observations of Schwartze, Lucae, Kidd and others, epitheliomata proceeding from the middle ear arise either during an existing suppuration of the middle ear, or after exhaustion of carious processes in the temporal bone. For this reason the growths protruding through the perforation of the membrana tympani are first taken for granulations or polypi. This was so in this case, and their rapid return after repeated removal lent colour to the view that it might be malignant.

Looking at the operation book of the Devon and Cornwall Ear and Throat Hospital, I find that during the last three years about sixty-five cases of middle ear suppuration cases have been curetted, and that with the exception of a few cases in which the after treatment has been neglected, they have all done well.

The case I have related shows the importance of the operation, even in suspicious cases, as it is very doubtful if any operation would be successful if cancerous disease had really invaded the bone.

https://doi.org/10.1017/S1755146300167149 Published online by Cambridge University Press

Dr. STCLAIR THOMSON. *Nasal Hydrorrhœa.*

Attention appears to have been first attracted to this subject by Bosworth in 1889 by a chapter devoted to nasal hydrorrhœa in his well-known "Diseases of the Nose and Throat." He therein collected and published eighteen cases, of which he considered the details were sufficiently clear to warrant them being embraced under the heading of "Nasal Hydrorrhœa." A critical examination of these eighteen cases has forced the author to the conclusion that six of them were undoubtedly instances of other morbid affections, and that nine were most probably dependent upon pathological conditions quite unconnected with the nasal mucosa; so that of the original eighteen cases, only three really justify their association under the title of "Nasal Hydrorrhœa." The author has been led to this view by his studies in connection with a case shown before the Laryngological Society of London, where, in an otherwise perfectly healthy subject, cerebro-spinal fluid, with rare intermissions, escaped day and night from one side of the nose. This case—together with the reports by other observers of seven undoubtedly similar cases, and twelve cases which were, most probably, identical—will be found fully recorded in Vol. LXXXII. of the "Medical and Chirurgical Transactions," 1898. It is claimed that of Bosworth's eighteen cases of "nasal hydrorrhœa," no less than thirteen were most probably instances of what should be called "cerebro-spinal rhinorrhœa." In the communication to the "Medical and Chirurgical Transactions" each of these thirteen cases is considered separately.

The writer deals at length with the chemical and clinical signs which distinguish cerebro-spinal fluid from intranasal secretion, and considers the question of the possibility of the hydrorrhœa originating in the accessory sinuses of the nose. Excluding all cases in which the discharge simply traversed the nose on its way from other cavities, and all those in which it is due to direct or reflex irritation, the conclusion is arrived at that what has been regarded as a distinct morbid entity is, in the majority of cases, but a symptom of various affections. Yet it is held that the term of "nasal hydrorrhœa" may still be preserved, if we limit it by defining the affection as one in which there is profuse watery discharge secreted by the nasal mucosa, and not dependent on intranasal or neighbouring sources of irritation. The amount of the fluid may vary from only, what the patient would term, "a slight running" up to as much as a pint in the twenty-four hours. The clinical picture of nasal hydrorrhœa shades off in one direction into cases of what are generally called "hay fever" or "paroxysmal rhinitis," with symptoms of intense local irritation; while in the other direction they may consist of a passive and almost painless watery discharge from the nose.

It appears to be an affection of adult life, affecting males and females indifferently. Although it may be more marked on one side than on the other, the flow usually takes place from both nostrils. When handkerchiefs are soaked with it, they generally dry stiff.

The author's contribution only claims to have advanced the matter one step by distinctly differentiating those cases where the nasal watery

https://doi.org/10.1017/S1755146300167149 Published online by Cambridge University Press

flow is really an escape of cerebro-spinal fluid. The importance of making this differential diagnosis is obvious. When such a character of the fluid is suspected, it is important to avoid any local interference from the risk of infection. In the cases presenting the conditions to which it is proposed to limit the term "nasal hydrorrhœa," the treatment can be only such as we have all tried in cases of hay fever—that is to say, quite empiric, with occasional brilliant results and frequent failures. A plea is entered for moderation in the energy with which so many practitioners use the galvano-cautery. The mucous membrane is easily destroyed; and, while the formation of scar tissue may give a sense of immediate relief, the after results may be worse than the original disease. Careful general treatment—hygienic, dietetic, and climatic—with, possibly, a visit to a suitable spa, will generally secure very satisfactory results.

Dr. FREDERICK E BATTEN. *On the Pathology of Diphtheritic Paralysis.* A paper based on the examination of six cases of that disease by the Marchi method.[1]

The author of this paper has examined the cranial nerves, the spinal cord, the anterior and posterior nerve roots, the posterior root ganglia, the vagi, the phrenics, and some of the nerves to the upper and lower extremities by Marchi's method. He has also examined the spinal cord and the posterior root ganglia by Nissl's method, and the nerves by Pal's method. Of the six cases which he examined five were cases of well-marked diphtheritic paralysis and one was a case of death on the fifteenth day after diphtheria, in which there was not actual paralysis.

The result of the examination is as follows: He finds degeneration of a parenchymatous nature in various cranial nerves in the anterior and posterior nerve roots, and in the nerve fibres as they pass through the white matter to the grey matter of the spinal cord, in the vagus, phrenic and peripheral nerves, and also on both sides of the posterior root ganglia. By Nissl's method he examined the spinal cord and the posterior root ganglia, and the cells in both places were found perfectly normal. By Pal's method no change could be demonstrated.

Of the six cases so examined four gave a positive result, while two gave a negative result, the one being the case above referred to that died on the fifteenth day after infection, the second being a well-marked case of diphtheritic paralysis with hemiplegia due to softening in the lenticular nucleus. In the first case he thinks that sufficient time had not elapsed from the time of infection to allow of changes manifesting themselves in the larger nerve trunks, and in the second case he is unable to give any reason for the negative result.

The paper gives a short digest of the work of various authors on the same subject; he refers to the work of Martin, who has shown that the finer nerve branches of the peripheral nerves are primarily affected, and to the more recent work of Mouravjeff, which tends to show that the primary alteration occurs in the cells of the anterior horn (demonstrated by Nissl's method), the parenchymatous change in the nerves being a secondary and later change; the same had also been shown by Crocq.

[1] From the Pathological Laboratory of the National Hospital, Queen Square, London.

https://doi.org/10.1017/S1755146300167149 Published online by Cambridge University Press

The affection of the posterior roots was first shown by Meyers in 1881, and he found that degeneration existed on both sides of the posterior root ganglia, and this has again been more recently noted by Crocq, Priez, Mouravjeff, and others. Bikeles described the occurrence of fat granules in the posterior roots at their entrance into the cord, the condition described is probably a normal condition commonly present in children, and to a lesser degree in adults. In conclusion, the author regards the dominant lesion in diphtheritic paralysis as a parenchymatous degeneration of the myelin sheath affecting both sensory and motor elements.

BRITISH LARYNGOLOGICAL, RHINOLOGICAL, AND OTOLOGICAL ASSOCIATION.

Friday, July 22nd, 1898.

Dr. DUNDAS GRANT, M.D., F.R.C.S., *President, in the chair.*

OTOLOGY.

The PRESIDENT. (1) *Deep-Seated Cholesteatoma of Petrous Bone with Facial Paralysis. Recovery after Operation.*

(2) *Alcoholic Auditory Neuritis.*

Along with nerve deafness there was pronounced anæsthesia of the feet and fronts of the legs. Great improvement followed administration of strychnia and abstention from alcohol.

Mr. STGEORGE REID. *Pure Cultures of Organisms Separated from the Discharge in Chronic Suppurative Otitis Media.*

Dr. FURNISS POTTER. *Subacute Myringitis Affecting both Membranes.*

A man, aged thirty-seven, a packing-case maker, came to the London Throat Hospital on the 24th of June, 1898, complaining of deafness in both ears and noises in the left ear. He stated that he could hear quite well eight weeks previously. A fortnight before this he had an attack of epistaxis, for which the nose was plugged. He had had no pain or discharge. Bone conduction was increased on both sides; he heard the watch at half an inch on both sides, and both Eustachian tubes were freely patent. On examining the external meati, the appearances were such as to make it doubtful whether the membranes were present or whether it was not a case of complete perforation. The subsequent progress, however, showed them to be intact, but hyperæmic and indrawn. The points of interest in the case were the complete absence of pain and the appearance of the membranes.

Drs. DUNDAS GRANT and PEGLER, and Mr. BARK remarked on the case.

https://doi.org/10.1017/S1755146300167149 Published online by Cambridge University Press