CONTRIBUTION
TO THE
HISTORY OF LARYNGEAL PHTHISIS.

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PART I.—A SHORT NOTICE OF THE HISTORY OF
LARYNGEAL PHTHISIS.

PREVIOUSLY to entering upon my own observations of this disease, I beg leave to retrace shortly the main features of the history of laryngeal phthisis, premising that after searching through various medical periodicals for a series of cases of laryngeal phthisis, carefully examined and reported in a practically useful form, my inquiries have been fruitless. This probably results from the fact that the present affection has not been universally acknowledged as a specific disease, and has therefore attracted, at all events in England, less attention than it appears to me to deserve.

It may also be added that owing to the comparatively recent discovery of the laryngoscope we have hardly yet had time for reaping its full usefulness in the study of laryngeal phthisis. It was only in 1857, during the summer months,

that Türck, of Vienna, endeavoured to employ the laryngeal mirror in the wards of the general hospital of that town. He was not successful, however, at first, and at the end of the autumn appears to have abandoned his fruitless attempt. In the month of November of the same year (1857) Professor Czermak, of Pesth, borrowed Dr. Türck's little mirror and overcame all difficulties; thus it was that Czermak created the art of laryngoscopy.¹

The 'Dictionnaire Encyclopédique des Sciences Médicales' (1868), under the heading of *Larynx*, contains a full and interesting monograph of laryngeal phthisis by Doctors Krishaber and Peter.

The affection is defined as an ulcerous disease of the larynx of a tubercular nature, occurring during the progress of pulmonary phthisis, and occasionally preceding it. Its essential character is a tendency to destruction (of tissue) from a mere epithelial erosion of the mucous membrane to caries or necrosis of the cartilages.

This affection may be superficial or deep-seated; in the former case it assumes the features of a catarrh in which the epithelium undergoes destruction, leaving the mucous surface uncovered and secreting a large quantity of muco-pus. This muco-pus is in all probability the substance I have called white mucus or milky secretion² for want of a better name to give it, and wishing to defer calling it pus until I could obtain positive evidence as to its nature.

As the disease progresses it burrows into the tissues and affects the mucous glands, which become inflamed and are frequently destroyed, leaving an ulceration, even where no tubercles can be detected in the larynx. When the ulcerations have made their way through the mucous membrane, the cellular tissue, muscles, and cartilages become inflamed and necrosis sets in; the vocal cord may also ulcerate and even undergo complete destruction, when the whole laryngeal cavity is bathed in pus, mixed with portions of expectorations often tinged with streaks of blood.

¹ 'Mackenzie on the Use of the Laryngoscope.'

² 'Clinical Notes on Diseases of the Larynx.'

The above changes may take place without the formation of tubercles in the larynx, although tubercles are usually met with in that organ in the latter period of the disease; moreover, the ulcerations from the destruction of the mucous glands may become the seat of tubercular granulations. These tubercles assume either the form of granulations or that of ulcerations. When after death such granulations are incised they exhibit a grey tissue, nearly always yellow in the centre (their microscopical description is given); they are seated immediately under the epithelium by which they are covered. Soon afterwards the epithelium is shed, and the elementary constituents of the tubercle become atrophied and disorganized, being converted into a small indurated and ulcerated mass (*petite nodosité cratèriforme ulcérée*); occasionally the mass stands out, exhibiting an ulceration round its base. When several of the ulcerations are near to each other they join by extension of the tubercular formation. Thus originate larger ulcers usually superficial and with festooned margins. Their edges and centre are commonly the seat of small vegetations or vascular bodies, which, however, are very seldom developed to such an extent as to bear the name of polypus. At the same time the mucous membrane becomes vascular and covered with muco-pus. In conclusion, the anatomical changes met with in laryngeal phthisis may be divided into three stages. In the first the mucous membrane and its small glands are affected, although but superficially, a desquamation of the mucous membrane takes place, and an erosion or superficial ulceration occurs round the orifice of the ducts of these glands. The second stage is attended with destruction of the mucous membrane, causing considerable loss of substance; but the ulcerating process is also continued in depth and extends to all or nearly all the different parts of the larynx (muscles, cartilages, ligaments, and articulations), thus constituting the third stage.

After alluding to the laryngoscopic appearances of the larynx, the authors proceed to describe the symptoms of the disease. With respect to the voice, it is more or less changed

according to the stage of the pulmonary affection in which the laryngeal disease has supervened. If the larynx is affected at an early period of the pulmonary mischief the voice is but little altered, but if it begins at a later stage of the disease of the lungs the changes in the voice—hoarseness and aphonia, are most marked. Whenever, in the course of pulmonary phthisis, aphonia sets in rapidly, it may be concluded that the laryngeal disease is likely to be of the severest kind.

Aphonia may be cured, the laryngeal disease proving a mere simple catarrh of the mucous membrane, as happens in simple laryngitis, and quite independent of consumption; consequently laryngitis in phthisis should not always be considered as incurable. The cough in laryngeal phthisis has a peculiar sound, due to the fact that the larynx cannot close completely in the act of coughing, and a certain quantity of air passes between the vocal cords instead of its causing them to vibrate by means of a series of succussions as in ordinary coughing.

The expectoration has, of course, a double origin, being derived from both the larynx and the bronchial tubes. It is very difficult, if not impossible, to ascertain which portion of the expectoration is to be ascribed to either one or the other of these two sources.

Hæmorrhages from the larynx are very rare.

Pain is not often met with, and ulcerations of the laryngeal cavity are painless. When, however, the disease affects the epiglottis, then the throat begins to feel painful. There are, however, exceptional cases of pain in the larynx, these being met with where the laryngeal and the pulmonary disease run a rapid parallel course.

Deglutition, according to our authors' observations, was a painful act in at least one quarter of their cases of laryngeal phthisis, and in nearly all of them proved troublesome. The pain experienced while swallowing is occasioned by the pressure on the larynx exerted by the constrictors of the pharynx, as they contract while the larynx is raised in the act of swallowing. The dysphagia is sometimes such that the efforts to swallow bring on vomiting.

As to the nature of the disease, MM. Krishaber and Peter are convinced that it is invariably connected with pulmonary tubercles. They discuss Cruveilhier's statements, who, in 1834, did not believe the tubercular diathesis to be the only cause of laryngeal phthisis; he considered that chronic laryngitis neglected or unskillfully treated might become ulcerous, and that laryngeal phthisis could exist by itself independently of any pulmonary affection.

With respect to the diagnosis there are three points to determine—1st, is laryngitis present? 2nd, is the laryngitis ulcerous? 3rd, the ulcerous laryngitis, is it tubercular? The two first questions are readily answered by laryngoscopic examination; the last by an inquiry into the state of the chest.

If the larynx be affected, although without any sign of mischief in the lungs, the affection is perhaps what the authors call initial or preceding the pulmonary disease, or it may be simple chronic laryngitis or a syphilitic inflammation. Should unmistakable signs of phthisis be found in the lungs the laryngeal disease is not on that account to be considered necessarily as tubercular, it may be simple chronic laryngitis, but the larynx will probably become ulcerated when the laryngeal mischief is tubercular. An ulcerated larynx may, however, result from a syphilitic affection, and in order to place the diagnosis beyond doubt, an inquiry is to be made into the history of the case so as to ascertain whether there be any syphilitic taint present. In the absence of any such predisposition there can be no doubt that the laryngeal disease is tubercular.

Our authors next proceed to examine nervous aphonia in phthisis, and conclude that it is independent of tubercular disease, merely resulting from the state of debility connected with the pulmonary affection; it is consequently curable.

The prognosis is always very serious. The initial form is that which is likely to last longest in the latent stage; in cases where a remission of the pulmonary disease takes place the consecutive or secondary form may also exhibit a latent progress, and even become arrested. The ultimate or late

form is the last blow given to a constitution shattered by pulmonary disease. In short, laryngeal phthisis, Messrs. Krishaber and Peter observe, may become modified, arrested, or even undergo temporary improvement, but is never cured.

As to the influence of the expectorations from their direct contact with the laryngeal mucous membrane, our authors consider it as merely favouring disease (*condition d'opportunité morbide*), although many distinguished writers, such as Silviu, Louis, Trousseau, Belloc, and others, believe the sputa to exert a morbid action.

Treatment.—That adopted by MM. Krishaber and Peter appears somewhat bold in its application, although carrying much weight with it from the authors' experience and standing. Holding the laryngeal mirror by the left hand, with the right they introduce into the larynx a piece of fused nitrate of silver fixed to a caustic holder, and cauterise *deeply* (authors' italics) the affected surface. This operation is perfectly painless and is remarkable in that respect, the more so that solutions of nitrate of silver, even weak, produce much pain when applied to the diseased larynx. After a few days a superficial sequestrum falls away, when a fresh cauterisation is effected, and so on until the ulcerated surface becomes modified, which occurs in the course of a few days. Thus, the ulcerating process is arrested, the pain in the larynx lessened, and deglutition becomes easier.

A narcotic solution may be introduced into the larynx having the following composition :

℞ Extracti Opii,
Extracti Belladonnæ aa. 50 centigrammes (gr. $7\frac{1}{16}$),
Aquæ Laurocerasi 28 grammes (3j).

The result of this application, it is stated, is certain (constant) and immediate; the means is truly a mere palliative, but it sometimes proves extremely useful, especially where it is necessary to relieve intense pain in the act of swallowing.

Inhalation of narcotic vapours is also beneficial; towards that object an infusion is prepared with 2 grammes (31

grains) of belladonna or stramonium leaves in 200 grammes (7 ounces) of hot water; the water is kept at a temperature of 160° or 170° F. in a special kind of inhaler, and the patient inhales the vapour for some minutes several times a day.

The acute laryngeal pain may also be relieved by hot fomentations applied to the larynx externally.

Dr. Pollock ('Elements of Prognosis in Consumption,' 1865) observes that he has found 8·66 per cent. of the whole number of (phthisical) cases to have their larynx affected,¹ but of those who attained the duration of four years only (19 out of 300) 6·33 per cent. had symptoms of laryngeal irritation. The extension of the disease to the organ of voice, although a much less frequent complication than diarrhœa, is a bad prognostic indication.

The question relating to the frequency of laryngeal phthisis in consumptive individuals is of much interest, and I regret that my own data with respect to this subject should be hardly sufficiently correct to allow me to express a result in figures; I should be inclined, however, to agree with Dr. Pollock's statement. My observations would give, perhaps, a rather higher proportion of cases of laryngeal phthisis, but, on the other hand, a certain number of these cases were sent to me at the time by my colleagues at the Hospital for Consumption, who, knowing I was engaged on that subject and took a special interest in it, directed some of their own hospital patients suffering from the throat to come to me. I am extremely obliged to these gentlemen for having thus promoted my object.

Niemeyer, 1868 ('*Traité de Pathologie Interne*,' translated into French under his directions), under the heading *Tubercular ulceration of the larynx*, admits two forms of laryngeal phthisis; one he calls infiltrated tuberculosis, the other miliary tuberculosis. He concludes that the disease (laryngeal phthisis) is never met with, but when the lungs themselves are tubercular, and observes, "we do not doubt

¹ Louis, from post-mortem examinations, places this proportion at 20 per cent. (Watson).

that tuberculosis of the larynx even in its diffuse form is of the same nature as pulmonary tuberculosis itself."

The seat of the affection, he observes, is usually the mucous membrane covering the transverse arytenoidean (probably arytenoideus) muscle, but tubercular ulcers may also be found in other places, such as the posterior aspect of the epiglottis. On that spot may be seen occasionally, though but seldom, little masses about the size of a millet seed, grey, and rounded, later turning yellow, then softening and undergoing disaggregation, and leaving in their place little rounded ulcerations of the size of a grain of semolina or hemp seed (the primary tubercular ulcer of Rokitansky). The union of several of these ulcerations from subsequent deposits becomes the source of a loss of substance irregularly defined and exhibiting raised and lacerated margins (the secondary tubercular ulcer of Rokitansky). It happens much more frequently, that the mucous membrane of the part affected first assumes a yellow tinge in consequence of its infiltration by innumerable small cells; the tissue becomes gradually relaxed, and a loss of substance takes place, first superficial, and by degrees spreading considerably in extent and depth. It is not unfrequent that the destruction is continued from the posterior walls of the larynx to the vocal cords, when it may happen that the posterior insertion of those ligaments is destroyed or undergoes considerable loss of substance. The tubercular ulcers of the posterior wall of the epiglottis in some rare instances perforate the whole thickness of that organ, the margins of the epiglottis remaining usually unaltered, while in syphilitic ulcerations this is not the case.

Ossification of the cartilages often coincide with tuberculosis of the larynx, and ossified sequestra occasionally form. Tubercular ulcerations may also perforate the laryngeal cavity, causing a fistula and cutaneous emphysema.

Niemeyer considers hoarseness in old-standing phthisis as a very probable sign of laryngeal phthisis, although it may also be due to muscular paralysis of the larynx without any organic change. The more the ulcerations approach the

vocal cord, the greater the hoarseness and more complete the aphonia. In other cases symptoms of hyperæsthesia are observed, characterised by great irritability and excessive cough and often terminating by nausea and vomiting; but, adds our author, "the symptoms we have just stated do not allow of the diagnosis of laryngeal tuberculosis so long as the existence of the same kind of affection in the lung cannot be determined; they can all of them be caused by other morbid changes in the larynx; it is therefore important, whenever a case of laryngeal disease is under consideration, to make a physical examination of the chest as carefully as possible, and put off expressing a positive opinion as to the tubercular nature of the affection, until the laryngeal symptoms can be connected with changes in the lungs as determined by percussion and auscultation.

"Cases of cure of laryngeal phthisis which have been reported, and the virtue of certain medicines called specifics in such instances are due in general to errors in the diagnosis. On the other hand, however, it is positive that a small number of recoveries have actually taken place."

On a few very rare occasions, œdema of the glottis supervenes, which proves fatal in a very short time.

With respect to the treatment, Niemeyer recommends the waters of Obersalzbrunnen or Kränchen of Ems, mixed with equal parts of warm milk. He advises the use of a strong solution of nitrate of silver applied to the larynx, and also frequent gargling with a solution of alum. The medicines, he adds, which prove most useful are narcotics; he prefers in this respect preparations of hyoscyamus and belladonna to those of opium. He also recommends living in a warm and slightly damp air, and in a place enjoying an equable temperature; he prohibits much talking and sometimes enjoins complete silence for weeks.

Niemeyer's description resembles that of MM. Krishaber and Peter in many respects, but all three authors appear to me too much to consider ulceration as the rule, as laryngeal phthisis may certainly exist for a considerable period without exhibiting any ulceration. Niemeyer, moreover, gives, I

think, too little attention to the state of the epiglottis, which is so frequently altered in its shape, and also to the intense pain in deglutition which accompanies the swollen condition of that organ. As to the topical use of solutions of nitrate of silver of which he appears to think well, I cannot refrain from observing that this means of treatment has never given me any satisfactory result. The local irritation this solution produces at the time is very great, and I much question whether any ultimate good follows.

Walshe ('Practical Treatise on Diseases of the Lungs,' 1871) observes that ulceration of the epiglottis gives rise to great dysphagia, especially of liquids, which frequently return by the nostrils. In proportion as ulceration destroys the vocal cords the voice degenerates into a hoarse whisper; he has never observed absolute aphonia; pain in the throat, stinging, pricking, or shooting, is more or less constant; if the epiglottis is free there is either no dysphagia or but very trifling difficulty in swallowing.

The physical signs are rough coarse respiration in the larynx, and sonorous, sibilant, or thin gurgling rhonchus, according to the dryness or moisture of the diseased surfaces.

Sir Thomas Watson ('Lectures on the Principles and Practice of Physic,' p. 204, 1871, vol. ii) observes that he knows of no such disease as laryngeal phthisis existing by itself, meaning that *scrofulous* (author's italics) ulceration of the larynx and trachea occurs only when the lungs are affected with tubercles, although it is sometimes a very early symptom. He also suggests the question as to whether the affection can be influenced by the contact of the matter which is expectorated and its frequent passage over the mucous membrane; and to this he answers, "Probably the fact, that the little mucous glands, wherewith the membrane is provided, are most numerous in the posterior part of the trachea and bronchi, and that these glands are especially prone to ulcerate, furnishes the truer explanation." Watson remarks that Louis, among 180 patients who died non-tubercular, only met once with ulceration of the larynx, while, of those who died of consumption, about one in every

five had ulceration of the epiglottis or larynx, and nearly one in three had ulceration of the windpipe.

Drs. C. J. B. and Ch. T. Williams, in their treatise on consumption, 1871 (p. 373), observe that some of the worse symptoms of the disease (phthisis) are those which indicate the various stages of ulceration of the larynx—pricking pain in the region, difficulty of swallowing, owing to the swollen or ulcerated epiglottis, hoarseness, gradually amounting to aphonia, and from time to time convulsive dyspnœa. These gentlemen have known of great comfort being given by the use of sulphurous acid in the form of spray, either alone or diluted with an equal amount of water. They state that Dr. Powell finds that a tincture of morphia, chlorate of potash, glycerine and soap, soothes the swollen epiglottis and renders swallowing more easy; but the authors conclude by remarking that little progress has been made in the treatment of the disease, and by hoping that the efforts of practitioners will some day be more successful than they are at present.

PART II.—CONTRIBUTION TO THE HISTORY OF LARYNGEAL PHTHISIS.

So much has been written upon the larynx in phthisis that there appears, at first sight, but little to add to it. It is only, however, since the comparatively recent discovery of the laryngoscope, and its introduction into general use, that the condition of the larynx, in cases of consumption, could be accurately investigated. Under the impression that there was still left a field for laryngoscopic inquiry into the present subject, I have submitted to a careful examination a fair number of cases of consumption attended with laryngeal disease; and these observations, together with the results they have yielded, form the ground-work of the present communication.

An inquiry of this kind is so far satisfactory that the pathological changes in progress can be seen at any

time, and their various stages investigated with the eye. The exploration of the chest is certainly the best means within our reach of ascertaining the condition of the lungs at any given time; but we cannot see into the lungs, and our hearing, educated by practice, must supply this want. We can now, however, by looking, satisfy ourselves of the state of the vocal cords, false cords, and epiglottis, together with the working of the laryngeal muscles. We can, moreover, by direct applications to the larynx, relieve the intense laryngeal pain which occasionally accompanies consumption; and we are able, I believe, to retard the progress of the disease in the throat, and in some cases prolong life by enabling patients to swallow where, from excessive pain and swelling of the part, deglutition is no longer possible. In cases of partial or total aphonia the state of the voice may be improved, although this satisfactory result is, as a rule, not easy to obtain.

Laryngeal phthisis is both a symptom and an extension of a pre-existing disease. It is a symptom, because, when clearly established by laryngoscopic examination, there is no doubt that the lungs either are, or will become tubercular if not obviously so at the time; it is an extension of a disease because, as a rule (I dare not yet say it is an invariable one, although it may be so), the pulmonary tissue is the primary seat of tubercular growth, mischief appearing subsequently in the larynx.

My connection with the Brompton Hospital gave me a wide field for the investigation of laryngeal phthisis, and I gladly availed myself of it. Consumptive patients, with throats in every conceivable stage of disease, applied for treatment in the out-patients' room of the hospital: some I examined on the spot; when pressed for time others were appointed to call at my house, where I inquired into their cases. Notes were taken carefully while the examination of the patient was in progress.

Predisposition to Laryngeal Phthisis.

There is, apparently, according to my experience, a very great difference between the predisposition to laryngeal phthisis in hospital and private practice; it is much greater in the former than in the latter case. I mean, that, in a given number of cases of consumption, the larynx, I believe, will be attacked much more frequently among those who attend hospitals than others; moreover, as a rule, the laryngeal disease will run a more severe course amongst hospital patients. I was prepared to find, by inquiring into the occupations of the patients I attended at the Brompton Hospital, that those who made most use of their voice, such as hawkers, would be most liable to laryngeal phthisis; but on examining into the subject this turned out to be hardly the case. The trades or employments were recorded in fifty-eight cases of laryngeal phthisis; most of the patients were engaged upon indoor labour, including shopmen, domestic servants, and clerks; there were two cases of butchers' assistants whose duty it was to call out the weights of the meat as it was taken off the scale, which appears to be an occupation very laborious for the vocal organ. There were one mattress maker, four needlewomen, including one machinist, one naval captain, one reader to the press, and only one labourer. I cannot find any case of a hawker recorded.

Of 309 phthisical patients I attended at the hospital in 1869, none of whom were recorded as suffering in any way from the larynx and who may be safely considered as free from laryngeal disease, about eighty were engaged in sedentary or indoor work, giving a proportion of 26 per cent.; while, out of the 58 cases of laryngeal phthisis, about 30 patients were employed on sedentary or indoor labour, yielding a proportion of about 52 per cent. of the total number. There appears to be consequently, a decided predisposition to laryngeal phthisis amongst those engaged upon indoor work, and who failed, therefore, as a rule, to take a sufficient amount of exercise

in the open air, and whose breathing was carried on in an ill-ventilated atmosphere.

I could not clearly trace the affection to overwork of the vocal organ, the two cases of the butchers' assistants calling out the weights are, however, interesting to record, the more so that out of the 309 phthisical patients without any laryngeal complication there does not occur one butcher. The case of the reader in a printing-office is also worth noticing.

From the above statement it appears that deficient exercise and want of pure air, and, I might add, neglect of hygiene, with which is often united mental anxiety, are the main predisposing causes of laryngeal phthisis. This explains how it is that those consumptive patients who can live in healthy districts, take exercise in the open air, procure good wholesome food, enjoying certain comforts, and freedom from anxiety, are less liable than others to suffer from an extension of the disease to the throat. In fact, laryngeal phthisis is apparently a low form of consumption, or a feature of phthisis occurring in a debilitated individual; hence the importance of attending as much to the general state of health in these cases as to the local mischief.

The task I set myself to work out was to examine together the physical signs of the chest and the state of the larynx where affected, in order to ascertain whether the progress of the disease could remain limited to either of these organs, or whether a change in one part was attended or not with some similar change in the other. I also endeavoured to trace the local alterations occurring in laryngeal phthisis as the disease ran its course, by following up such cases for a considerable length of time; and, finally, every possible attention was given to the treatment.

Before entering strictly upon the subject of laryngeal phthisis, I must beg leave to begin by drawing attention to that state of the larynx, which is occasionally met with in consumption, characterised merely by weakness of the voice, and sometimes aphonia, without there being any inflammation or

tubercular growth in the vocal organ. The patient finds out that after speaking for a length of time the voice drops, or it fails ; while singing, reading aloud, or teaching has to be discontinued, and the act of phonation becomes an effort. This affection may remain stationary for months, quite independently of the course taken by the pulmonary disease ; in some cases it becomes worse ; by degrees speaking may require a considerable effort, when the sound of the voice assumes a high and unnatural pitch, but at that stage the patient finds it so laborious to utter sounds that words are usually spoken in a whisper. A laryngoscopic examination shows at first sight no apparent mischief, but on the patient attempting phonation it will be seen that the vocal cords do not move freely. In most cases the tension of the cords is observed at fault, while their adduction is unimpaired ; in a few others the adduction is incomplete. Where tension is deficient, the larynx, instead of exhibiting long straight vocal bands occupying the whole antero-posterior diameter of the larynx, as in the normal state, shows them to be shortened and somewhat shrivelled, the whole larynx contracting round its centre in its attempt to produce a sound ; while sometimes the cords entirely disappear from view.

In the other case the length of the cords does not appear altered, but they fail to approach each other sufficiently to vibrate.

This last state of the laryngeal muscles is usually met with in hysterical aphonia, and, indeed, it appears to exhibit in consumption the peculiar intermittent character it is found to have in hysteria, the voice leaving and returning from time to time without any apparent cause. Still the present laryngeal affection may be met with in phthisical male patients who can in no way be considered hysterical.

Now, although these two functional changes in the larynx might appear to have different origins, the tensors or cricothyroid muscles being supplied exclusively by the superior laryngeal nerve, while all the other muscles are supplied by the inferior laryngeal, still both nerves arise from the pneumogastric which is also distributed to the lungs,

and they all three meet in the ganglion on the root of the track of the pneumogastric. Therefore it appears to me possible that the deficient state of nutrition or abnormal condition of the lungs should also weaken the functions of the laryngeal nerves, by a reflex action through the ganglion. In hysterical aphonia the mischief appears more local, and by faradisation of the laryngeal muscles a complete cure can often be affected; while in phthisical aphonia without organic change, electricity, although it may be of use, often fails to restore the voice or even to improve it materially. On the other hand, in these cases, an improvement in the state of the lungs and the general health will often be attended with a simultaneous favorable change in that of the voice.

Trousseau ('Clinical Med.' vol. iii) alludes to this particular form of aphonia in consumption, unattended by any organic change. He observes ". . . but likewise and without any material lesion, as is shown by examination with the laryngoscope, pulmonary tuberculisation sometimes produces nervous aphonia." This affection is also noticed, as stated in the first part of this paper, by MM. Krishaber and Peter.

The following is a case illustrating the usual form of nervous aphonia in phthisis, that which is due to want of power of the *tensor* muscles of the vocal cords.

(CASE 46.)—Mary Ann T—, middle-aged, pregnant (and has a large family), consulted me on the 25th June, 1869. The physical signs of the chest were—on the left side in front respiratory murmur, with humid crepitus just below clavicle, falling in at right apex with some dulness, and deficient murmur on that same side, abundant mucus crepitus and tubular breathing under the third rib. She had an attack of hæmoptysis, which lasted two days, two months before applying.

I examined her with the laryngoscope on the 23rd July; no organic change was to be seen in the larynx; the cords looked healthy. On attempts at phonation the arytenoid cartilages moved freely towards each other, showing a proper

adduction of the cords, but they were drawn out of the field of the mirror and appeared deficient in tension.

She lost her voice three months before coming to me, a word being, however, spoken out now and then. I understood her to say she had a previous attack of aphonia, from which she had recovered.

On applying faradisation to this patient's larynx I thought I observed a want of sensibility of the organ, as it was not conscious of a fairly powerful electric current, which, however, was felt when stronger. At first faradisation caused but a slight improvement, but she also took cod-liver oil and iron, and was eventually benefited considerably by the treatment. On the 21st September her voice was much improved, though still weak and hoarse.

In the following case it was not the tensor muscles, but the adductors, which were at fault.

CASE 47.—Henry G—, æt. 22, a labourer.

Physical signs.—Left and right side (in front) shade of dullness from apex to middle. Left in front crackling and sibilus, no obvious respiratory murmur. Posteriorly shade of dullness on both sides. Left supra-spinous fossa crackling and metallic sounds. Right posteriorly respiration bronchial with crepitus. Voice very weak and hoarse.

Laryngoscopic examination.—Throat so irritable that the mere introduction of the glass into the mouth caused spasmodic contractions. Recourse was had to a waterproof bag filled with a mixture of ice and salt tied round the patient's neck and in contact externally with the larynx; a few minutes later the examination proved quite easy. The laryngeal mucous membrane and that of the arytenoid bodies was rather relaxed and red; there was some congestion of the cords, epiglottis somewhat pendulous but during the act of coughing the larynx became plainly visible. It was then distinctly observed that on attempting phonation the vocal cords failed to approach each other, although both of them moved about half-way towards the median line. This patient had two attacks of pleurisy thirteen months before (August twelve

months), the second being rather a relapse, and he never quite recovered his health since then. There was nothing defective in his voice at that time, but just before the following Christmas he became hoarse and by degrees nearly lost his voice completely. In the following month of May he had a very bad cold, with an attack of hæmoptysis. He began to suffer from a fistula in ano about the time when his voice became affected.

I will now relate an interesting case, in which both the adductors and tensors failed in their action, there being extensive disease in the lungs.¹

John F—, æt. 21 (Case 36), has been subject to aphonia for the last two months. His speech is now (19th March, 1869) delivered in a cracked tone of voice, and some words are dropped in a whisper; he has never suffered from hæmoptysis; one of his brothers died of consumption.

Physical signs of the chest.—At right apex in front, shade of dulness, cavernous respiration with crackling to third rib; at base respiration vesicular with some crepitus. On that side posteriorly unmistakable signs of cavity and consolidation. Left lung tolerably healthy.

With the laryngoscope the posterior halves of the vocal cords are seen to have lost their natural hue and acquired a red glistening appearance. The cords fail to stretch properly, as shown by the middle of the chink between them remaining open when a sound is attempted, while their ends come into mutual contact.

The arytenoid cartilages are observed to be drawn anteriorly, in the act of phonation, and to approximate so close to each other as to meet, or very nearly so, the cords being at the time much shortened. On abduction the cords do not move quite symmetrically, the left assuming more of a diagonal position than the right, they appear uneven. Supraglottic region red and relaxed.

The patient was treated with cod-liver oil, syrup of phosphate of iron with quinine, and croton oil liniment rubbed

¹ 'Clinical Notes on Diseases of the Larynx,' p. 104.

occasionally on the right side of the chest. Faradization was applied repeatedly to the vocal cords.

The progress of the case was quite satisfactory, and on July 16th both vocal cords moved freely and symmetrically in the act of phonation; they were not shortened and came tolerably well in contact throughout their whole length, showing a marked improvement in their power of tension. The arytenoid cartilages were no longer displaced in an anterior direction, and their lateral movement was quite free.

The patient could sing the scale without any difficulty, and the sound of his voice was much improved. I saw him next on the 8th July, when his voice might have been considered as quite natural.

From the interest of this case I may perhaps be allowed to reproduce it on the present occasion.

We may conclude that hoarseness, or aphonia, may result from phthisis, either directly or indirectly, without any organic affection of the larynx; and in these cases electricity may be productive of much benefit, and even bring on recovery, although the treatment can hardly be considered so successful as it is in simple hysterical aphonia.

I shall now enter more directly upon the subject of my communication, namely, *laryngeal phthisis*; but it is necessary that some explanation be first given as to the meaning this denomination is proposed to convey.

I propose to include under that name both what might be called *tubercular laryngitis* and *laryngeal phthisis*. The former would mean of course laryngitis or inflammation of the larynx connected with tubercles, the latter would mainly convey the idea of an adenoid growth (tubercular deposit) in the larynx independently of inflammation. It often happens that when inflammation accompanies laryngeal phthisis it possesses no peculiar appearance beyond what is observed in common laryngitis; but it differs in this respect—that while in common laryngitis it yields readily to treatment, in tubercular laryngitis it frequently goes from bad to worse, whatever be the mode of treatment adopted. There is, apparently, in

laryngitis accompanying phthisis, a local source of irritation acting incessantly and keeping up the inflammation; which is not the case with ordinary laryngitis. The usual signs of laryngeal inflammation may last for a considerable time in phthisis and sometimes merge into a permanent thickening without any tubercular granulations being visible. It is obvious to me, however, that the thickening is of a tubercular nature; the parts forming the vocal organ, which are naturally soft and flexible, become stiff and indurated, and on being punctured with a scarifier, convey the impression of a hard gritty body. Moreover, a white milky secretion (probably purulent), varying in thickness, is observed on the walls of the larynx, and sometimes oozing out between the arytenoid bodies, in nearly every case of inflammation of the larynx accompanying phthisis. It is so frequently met with under those circumstances, that its presence has enabled me to diagnose tubercular disease of the lungs where the pulmonary symptoms were, indeed, very questionable.

In many cases, however, perhaps in most, a granular appearance is seen in various parts of the larynx; it may be visible either on the arytenoid bodies or epiglottidean folds, the false cords or the epiglottis; the part looks as if speckled over with a number of small white dots the size of a pin's head or millet seed. The tissue in which these little masses are imbedded is red and congested, but if scarified the part feels resistant, and yields much less blood than would have been anticipated. It may happen that the granulations are concealed by the white secretion I have referred to, but if that be wiped off with a brush the state of the laryngeal mucous membrane becomes at once visible.

Where the epiglottis is thus affected it becomes very hard and altered in shape, assuming a shrivelled up and contracted appearance. The pain felt in the throat in these cases is sometimes excruciating, especially during the act of deglutition. I have seen patients dreading to swallow, throwing the head into every possible position, and applying the hands tightly against both ears with the vain hope of obtaining some relief during deglutition, while intense pain

was marked upon their features. When the epiglottis is much contracted it fails to protect effectually the orifice of the larynx, and the discomfort of liquid food not finding its way readily into the œsophagus is added to the pain. Those extreme cases are fortunately but seldom met with. I have, however, seen a sufficient number of them to be thoroughly impressed with their serious and extremely painful character.

It sometimes happens towards the termination of the disease that growths or little tumours form on the larynx; they are seen, perhaps, mostly on the arytenoid bodies, but may be met with on other parts of the larynx, and are accompanied by softening and ulcerations of the laryngeal mucous membrane. These changes indicate, of course, a most unfavorable condition.

It is remarkable that, although in some instances the soft parts of the larynx should become so thoroughly indurated, still the vocal cords continue moving apart tolerably freely in the act of respiration, so that actual dyspnœa beginning at the larynx is seldom met with. The detection of the very first occurrence of softening of the laryngeal mucous membrane is not always easy to determine. When this change has set in, the white secretion becomes thicker, and on removing it with a camel's-hair brush the surface of the mucous membrane is seen to be pitted, or, if I may be allowed the comparison, somewhat like moth-eaten cloth; or the follicles on the posterior aspect of the larynx, lying between the arytenoid bodies appear enlarged and somewhat excavated at their base. In other cases positive ulceration is met with, the posterior ends of the vocal cords being not unfrequently involved in this change.

I have often attempted to ascertain whether the succession of changes the larynx undergoes follows a determined course; whether weakness of the vocal muscles is always the first change, next, irritation and inflammation of the part, then swelling and induration with a granular condition, and, finally, softening and ulceration.

I cannot say, however, that I have succeeded in doing

this, and question whether those changes invariably follow each other. I have observed, for instance, that a merely weakened condition of the muscles of the vocal cords may often exist throughout the whole course of a case of consumption without its passing into inflammation. Indeed, it is very seldom, in a case of confirmed phthisis, that the power of phonation is not more or less weakened; this may not be apparent, but the patient will be conscious of it. Tubercular disease in the larynx may, however, also commence with a want of power in the production of sounds, the earliest signs of inflammation being superadded weeks or months later. A weakened state of the voice is a sign of no little importance, especially when it has lasted for a considerable length of time. Patients are often aware of this, and it does not unfrequently happen that a lady with consumption in her family, although in apparently perfect health, suddenly finding her voice beginning to fail when singing, will at once anxiously seek for medical advice.

Suspicious symptoms may be heard in such cases at the apices of the lungs, such as some harshness, slight vocal resonance, and deficient respiratory murmur, without, however, any appreciable dulness on percussion. These cases may do well under treatment, the power of singing returning, although care should be taken by such patients to use the voice as little as possible for some time.

Inflammation of the larynx, with the presence of the white milky secretion, may be accepted as a very strong evidence of phthisis, although, of course, if unaccompanied by any physical signs of the chest, one should pause before giving a positive opinion. I have related a case ('*Brit. Med. Journ.*,' 24th October, 1874,—"*Consumption, a form of septicæmia*") of a gentleman in apparently perfect health, in whose throat I detected the presence of the white secretion alluded to above. There was no sign of mischief in the lungs beyond a slight dulness on percussion at the left apex; yet, twenty-six days afterwards, this patient was seized with hæmoptysis, which proved the beginning of acute phthisis, and he died a month later.

Laryngeal disease in phthisis leads to aphonia. Of course,

where the soft parts of the larynx are thickened and indurated, it is natural that the voice should be affected, but it is often remarkable how little laryngeal mischief may produce this result. In the early stage of the aphonia with thickening of the larynx, the voice may be restored by electricity, but we must not flatter ourselves that the recovery will be permanent. In an old-standing case of aphonia with chronic thickening, electricity will not be of much avail, although a few words, and perhaps some sentences, may be obtained by this means. I question whether ulceration of the larynx be necessarily attended with aphonia, unless the ulceration should be situated on or very near the vocal cords.

The prognosis derived from laryngeal phthisis is, I fear, as a rule, unfavorable. It is specially so when softening of the larynx has positively set in. Still, with proper management, especially respecting climate, I have seen patients with the soft parts of the larynx considerably thickened, and obliged to converse in a whisper, spend a very comfortable and pleasant winter in the south of France, and certainly be no worse in the spring than they were in the preceding autumn. As a rule, the condition of the throat remains chronic so long as the state of the lungs is quiescent, and, consequently, the pulmonary conditions and general health must be always taken into account, together with the state of the larynx, in reference to prognosis. I now have to remark that the results of my observations as to the state of the throat compared with that of the lungs, has led me to conclude that similar changes do not take place simultaneously in both organs. Thus, patients having large cavities in the lungs may continue for a considerable time with a thickening in the larynx, exhibiting no signs of softening; or there may be chronic irritation in the throat, with the secretion of white mucus, and no aphonia, remaining unchanged to the termination of the disease. I do not think that laryngeal consumption is cured any more than pulmonary consumption, and it is obvious enough that if the affection of the throat should be owing to tubercular or adenoid growth,

the cause of irritation cannot be removed. If, however, such a satisfactory result cannot be obtained, we can do a great deal towards relieving the irritation and changing its acute into a chronic form by removing the patient into a proper climate.

There is no contagious or especially morbid quality in the air expired by those who suffer from laryngeal phthisis, although it is frequently possessed of a very disagreeable fœtor. I have found, however, the breath of such persons extremely irritating to my own larynx, and a protracted laryngoscopic examination in cases of phthisis has frequently been productive in me of much discomfort and hoarseness. This influence of expired air when breathed insufficiently diluted, is not, I believe, limited to laryngeal phthisis, although, perhaps, the breath in this affection and, I might add, in consumption, is still more irritating than under other morbid conditions.

All work with the laryngoscope when carried on for a certain time has had an irritant effect on my own throat, so much so that the members of my family have frequently been led to infer, from the sound of my voice, that I had been much engaged with the laryngoscope on that particular day. At times my voice was very hoarse with a constant desire to clear the larynx, which proved extremely troublesome; wiping out my own larynx with a camel's hair brush only removed a small quantity of perfectly clear mucus. This continued for months till, at last, I had to adopt some means to put an end to it, and with this object in view I wore a charcoal respirator every time I had to do any hospital work with the laryngoscope. This plan was perfectly successful. The liability to laryngitis from breathing expired air remains, however, although I am not in the least subject to irritation in the larynx from other causes.

The foregoing remarks certainly show that a state of irritation of the larynx may proceed from breathing too close to the mouth of another person, and the effect will be the same in crowded and ill-ventilated rooms.

Although healthy expired air is irritating to breathe,

air expired from those who suffer from laryngeal phthisis, and also I might say from phthisis, unaccompanied by any laryngeal disease, appears to me, as previously stated, possessed of a still more irritating character. All substances present in the blood under a form that allows of their being eliminated in a volatile state, find their way out of the body through the lungs, so that expired air contains many substances besides carbonic acid. A well-known experiment of Claude Bernard, which I have frequently repeated, consists in the injection of an aqueous solution of sulphuretted hydrogen into the veins of a dog, and testing the air expired by the animal a few seconds later, when it is found to be highly impregnated with sulphuretted hydrogen. This shows how very readily volatile substances are given out of the blood through the substance of the lungs. I have also observed on one occasion a man who was wearing a tracheal canula, and whose breath, therefore, must have come from the lungs without passing through the mouth, expire air smelling strongly of onion or garlic. Now, some of these volatile substances appear to be irritating to mucous membranes; indeed, they are chiefly effete matters, and cannot therefore be taken into the body without producing mischief.

It is not improbable that the tendency to consumption resulting from a sedentary occupation in crowded and ill-ventilated rooms is partly owing to the irritating influence of expired air, but this tendency may be due also to some property possessed by expired air which may be allied to that of a poison, just as urea acts as a poison when injected into the blood, or when its elimination from the body is checked by disease of the kidneys. Now, when pure air is inhaled it becomes charged in the lungs with the volatile constituents of the blood which have diffused themselves through the pulmonary tissue; but if the air inspired be already loaded with volatile substances from other people's blood, these substances will diffuse themselves through the lungs of those who breathe them into their own blood, and act as a poison; and to the present substances must be added carbonic acid, which will also be absorbed for the

very same reason. No wonder then that the nutrition of lung-tissue charged with carbonic acid and volatile substances acting as a poison should become altered and eventually diseased.

Treatment.—The treatment of laryngeal phthisis may be classed under three heads—1st, local ; 2nd, medicinal ; 3rd, climatic ; the second and third including means of treatment which apply, of course, to pulmonary phthisis as well as to the laryngeal disease.

The local treatment is clearly that which should be applied first. The use of solutions of nitrate of silver, perchloride of iron, or of any strongly astringent and irritating fluid, I have discarded. I do not find these applications do any good, and they produce much distress at the time. Solutions of bromide of potassium, and alum in glycerine, or gum water, will answer the purpose much better ; when applied freely to the larynx with a camel's-hair brush, they often produce a temporary relief of the pain. I have, however, found a solution of iodine in olive oil introduced into the larynx to answer the purpose more effectually ; ten to twenty grains of iodine with two or three grains of iodide of potassium dissolved in one ounce of olive oil forms a solution which keeps fairly well. In case of much irritation of the larynx a few drops of this solution may be mixed with an equal number of drops of olive oil before it is used ; it is remarkable to what extent olive oil checks the irritating action of iodine, which it dissolves readily, and the operation is sometimes attended with great relief. The only drawback is the taste of the solution, which with some people is extremely disagreeable ; this objection, however, may be overcome by causing the patient to rinse out the mouth with a little eau-de-Cologne and water immediately before and after the application. I also recommend the same kind of iodized solution, mixed, however, with a little chloroform, to be rubbed into the skin on the front part of the neck twice a day ; the object is not to produce counter-irritation, but to obtain an absorption of the iodine. Where the soft parts of the larynx are much swollen and hardened, scarifying the part with

Mackenzie's laryngeal scarifier sometimes gives relief; this means is especially useful when the epiglottis is enlarged, producing dysphagia and much pain in the larynx. A few punctures of the epiglottis let out a little (though very little) blood, and give much relief at the time. Care should be taken, however, not to repeat the punctures too often, lest waste of substance and softening of the tissue should occur subsequently, at the punctured spot. I have never observed purulent matter discharged by puncturing the larynx in laryngeal phthisis; the small lancet is felt to enter a hard gritty tissue, and a drop of blood will appear at the puncture; so little blood is usually obtained that it hardly tinges the saliva on clearing the throat; on a subsequent examination with the laryngoscope at the same sitting, a little red speck, but no oozing of blood, is all that is likely to be seen of the puncture.

I have never found leeches applied externally to the throat of much use, and as to counter-irritation, I am inclined to doubt whether any benefit be derived from it in such cases. With respect to the aphonia, I am afraid no other means is available but Faradization of the vocal cords although the voice may return for a few seconds after scarification. Where the laryngeal mucous membrane is but slightly thickened, the aphonia resulting mostly from a state of debility of the cords, electricity may be of marked benefit, and often after a few sittings words and sentences can be spoken out at intervals. It is better to place the electrodes of the battery outside, right and left of the thyroid cartilage (a method of applying electricity to the larynx suggested to me by Dr. Krishaber of Paris), than introduce one of the electrodes into the larynx, as is done in cases of hysterical aphonia. I usually move about the wet sponges on both sides of the larynx so as to avoid exhausting the muscles by prolonged excitation. The operation may be repeated from twenty minutes to half an hour every other day; of course it is only in chronic cases that electricity can be applied; where the larynx is greatly inflamed and very painful, no such treatment is admissible.

Climate.—The climate most likely to benefit cases of laryngeal phthisis is of course that which is best suited to the treatment of phthisis. At a certain height above the sea, a height which varies with the latitude, no cases of phthisis, or but very few indeed, are to be met with. Dr. Lombard, of Geneva ('*Climat des Montagnes*'), places that height in Switzerland at a little beyond three thousand feet, an altitude also adopted by Dr. Hermann Weber ('*Med.-Chir. Trans.*,' 1862). Dr. Symes Thompson ('*Med.-Chir. Trans.*,' 1873,) considers that temperature, as well as altitude and latitude, should be taken into account with respect to a place fit or unfit to be a health resort.

We have yet, I think, to test experimentally how far it is advantageous to send patients to winter in the mountains in northern countries at those altitudes where consumption is unknown, although such places may be admirably adapted for summer residence. We have also to find out to what extent the conditions producing this immunity from phthisis in the native population influence the arrest of phthisis where it has clearly broken out.

I am inclined to believe that, as a rule, those who suffer from consumption, in an acute form, wherever they may be living, except when residing at a considerable elevation, will derive benefit from change to a higher situation, although perhaps not distant from their previous residence, and possibly not more than from 100 to 300 feet above it. Thus an obvious benefit is not unfrequently met with by removing merely from London to Norwood. From Geneva, 1200 feet above the sea, patients are frequently sent in the spring-time to Mornex, 500 feet higher, on Mount Salève, where they often improve in a most remarkable way. On the Mediterranean coast I have seen cases of pneumonia, hæmoptysis, and fever, not only get better, but recover completely in a very short space of time by leaving the seaside and resorting to the hills a short distance from the sea, at a difference of altitude ranging from 150 to 300 feet. I have even met with a patient suffering from acute phthisis, at Nice, who derived temporary benefit from being moved from the first floor to

the top floor of a tolerably high hotel; while, on the other hand, I have known patients to be the worse for coming down to the Mediterranean coast in the spring, after enjoying tolerably fair health throughout the winter on the hills.

In cases of chronic phthisis, with the pulse and temperature normal, it may perhaps be an advantage for patients to take up their residence nearer to the Mediterranean coast on a low level; but should the pulse increase in frequency, and the temperature of the body rise, the hills but a little way off should be resorted to at once.

Syphilitic or malignant disease may be mistaken for laryngeal phthisis. The absence of the physical signs of pulmonary consumption will, as a rule, dispel the idea of a tubercular condition of the larynx; still, as the symptoms of phthisis are not always very obvious, it will perhaps be well to pause for a little time before deciding on the treatment. A course of mercurial medicine advisable in one case would be likely to do mischief in the other; indeed, according to my own experience, all mercurial preparations should be carefully withheld in tubercular disease, whether of the lungs or throat. It is not easy to point out any very marked difference between the laryngoscopic appearance of syphilitic and tubercular laryngitis; these two kinds of affections I have seen, indeed, present like appearances; perhaps the syphilitic disease is more liable to spread to the uvula and velum, to produce thickening and ulceration, and interfere with the respiration at the glottis. The history of the case will of course help to "settle the question." Epithelioma of the larynx will exhibit warty excrescences in the part, easily recognised by their angry-looking appearance, and the intense pain felt in the throat; there may be in such cases much dysphagia and pain on deglutition, without any affection of the epiglottis.

Post-mortem appearance.—Towards the fatal termination of consumption, softening of the mucous membrane of the larynx sets in, where there has been laryngeal phthisis, and after death the mucous membrane of the larynx

discloses the pitted character observed during life. I have before me the notes of the post-mortem appearances of the larynx of a boy aged sixteen years, who died of consumption with laryngeal phthisis at the Brompton Hospital. Softening had commenced on the right arytenoid body, which exhibited an irregular wasting of tissues, and depressions with ragged edges on the mucous membrane. The surface of the larynx appeared to have been undergoing a process of solution and removal; the epiglottis was studded with translucent specks, as if the substance deposited was undergoing a change preparatory to actual softening; the false cords appeared healthy, the true cords exhibited an injected and swollen appearance. In another case, that of a girl of twenty-two years, the whole of the posterior wall of the larynx was found uniformly tinged with a pink colour; softening had commenced under the epiglottis at the commissure between the anterior ends of the vocal cords.

Softening may extend down the trachea, or perhaps begin in the trachea, reaching the larynx; it is, of course, difficult to tell where the affection commences.

CASES.

Before proceeding to offer a few remarks on the cases recorded at the end of the present communication, I shall beg leave to give an account of two patients, which appears to me of interest with respect to the subject of the present paper. One of them exhibited a very suspicious appearance of phthisis in the larynx, while the physical signs of the chest were not those of perfect health. The other suffered from acute laryngitis, with aphonia accompanying congestion at the base of the lungs. Both patients recovered perfectly.

B. H—, æt. 17 years, had been an inmate of my own house for a period of seven years, and was therefore constantly under my observation during that time. He was tall and well proportioned, and his general state of health was excellent. On the 6th April, 1871, he complained to me that he felt a thickening in his throat as if a ball was lodged there. This peculiar sensation he had first noticed on the 3rd of that month; he had also coughed up a little blood on that same day (the 3rd April). On looking into his throat with the laryngoscope, I distinctly saw that his epiglottis was swollen; he complained of pain in swallowing, which (as usual in such cases) he referred to the inside of his ears, and his voice was rather weaker than usual.

Physical signs of the chest.—Left side harsh, bronchial, noisy expiration; at right apex shade of dulness to second rib and vocal resonance deficient over that space, but no crepitation; respiratory murmur normal. At right supra-spinous fossa respiration very harsh; murmur bronchial and deficient; no dulness; a systolic bruit at apex of the heart. Pulse 76, regular; weight of body 8 st. 11½ lb. Never suffered from rheumatism; was formerly subject to abscesses. When six years old he had, he says, brain fever, and was ten days without speaking a word. Never suffered from inflammation of the lungs, or cold or catarrh of any kind; there is no hereditary predisposition to phthisis. I treated the case with syrup of iodide of iron, cod-liver oil, and his throat was rubbed externally with iodized oil.

On the 12th April, or six days after commencing the treatment, he had gained 2½ lb. weight.

On the 19th his throat was much better, the peculiar sensation he first complained of having much diminished. Coughed a good deal and expectorated; right apex respiration very harsh and some dulness to second rib confirmed. Left in front nothing abnormal; back of right side respiration very harsh, and dulness at apex. Applied acupuncture and a weak solution of croton oil in cajuput oil to right apex.

Only a slight degree of thickening of the epiglottis now remained, and he felt but little uneasiness in the throat on swallowing.

27th.—No longer any discomfort in the throat excepting in a slight degree in the evening when going to bed.

Physical signs.—Questionable dulness right apex; murmur rather bronchial at that spot with somewhat exaggerated expiration; no crepitus; he had lost, however, $1\frac{1}{2}$ lb. weight.

May 15th.—Right apex quite clear on percussion, but a shade of dulness at the back where murmur is deficient and very harsh. Now feels quite well, and can take a long walk without fatigue.

November, 1871, at Nice.—Respiration rather louder than natural in the right side; murmur deficient and harsh with distinct crepitus on the right side at the back; a decided though but slight dulness at right supra-spinous fossa. Had been in very good health since last May; epiglottis was seen to have resumed its normal state, though slightly thickened at the rim. No cardiac murmur whatever, but cardiac impulse rather strong.

December 6th, 1872, Nice.—Had spent last winter at Nice, and the following summer in England. Percussion note on both sides now quite resonant; left and right supra-spinous fossæ respiration bronchial, very harsh, with loud expiration. Complained of sore-throat; had had a chill three or four days before. Pharynx red with some small aphthæ on the pillars. The epiglottis at the tip appeared a little thicker than usual. Weight 9 st. 9 lb., had gained $9\frac{1}{2}$ lb. since the 19th April, 1871.

March 13th, 1874.—Had again wintered at Nice; no abnormal signs in front left and right. At the back very fine crepitation right supra-spinous fossa, and respiration harsh lower down. Left supra-spinous fossa respiration harsh, normal lower down: no dulness. A laryngoscopic examination showed the epiglottis to be still slightly thickened at the rim, but free from congestion or irritation. Said he now felt nothing the matter with his throat; indeed, had

enjoyed excellent health since last examination. He had grown into manhood, and his frame was well developed in every respect. Now weighed 10 st. 5½ lb., having increased 10¼ lb. since December, 1872.

This case is interesting, and certainly looks very much like one of phthisis arrested *ab ovo*. The epiglottis is not, according to my experience, liable to swell from common temporary inflammation, as would be the case with the other parts of the larynx in laryngitis. An enlarged and indurated epiglottis is to me an invariable sign of some serious mischief. In the present instance there may still remain a very slight thickening of the epiglottis. I have no record of ever observing the white mucous secretion in this patient's larynx, or, indeed, of any other laryngeal mischief besides that relating to the epiglottis.

In the following case there were pulmonary congestion and bronchial catarrh in the lower part of the lungs, and in the larynx the vocal cords were mainly affected, the epiglottis remaining healthy.

Mr. B—, aged about 40, arrived at Nice suffering from incessant and very loud coughing, and having entirely lost his voice. He caught cold about eight weeks before from having been detained at sea in a small boat after dark, when he had to row for a considerable length of time, and was probably chilled after perspiring very freely, and when tired in other respects. His throat became sore, and he was treated with the application of nitrate of silver, but his voice became weaker and dropped into a whisper.

January 16th, 1874.—His cough comes in violent noisy fits; there is some little expectoration at night, but none in the daytime.

19th.—Pulse 102; temperature under tongue 101°; much wheezing heard under the sternum. Face red; cough distressing; larynx had been found on a previous occasion red and angry, though not apparently swollen. I could not distinguish the true vocal cords, and as medical treatment

appeared of little use, weak as he was and but just able to stand, I took this patient out of his bed up to a villa at St. Philippe, about 150 feet above Nice, on the slope at the western side of the town. The next day he was already much better, his voice returning at intervals, though weak and hoarse. Appetite returning. Temperature of the body, 99.4° ; pulse, 100.

29th.—Improvement continued. Had walked an hour without fatigue; appetite much better. I then obtained a good view of the throat. On attempting to make a sound there was some difficulty in the adduction of the cords, which sometimes met, sometimes remained apart. During phonation I could plainly see the true cords reduced to a very thin pale lamina, instead of exhibiting their white pearl glistening bands. The false cords appeared rather thickened; voice still very hoarse and rough. On January 19th the wheezing under the sternum had entirely disappeared, but there remained some distinct crepitation with slight dullness over a considerable portion of the base of the left lung.

On January 29th dullness had disappeared, though crepitus continued.

February 3rd.—Rudimentary vocal cords approached each other, but lacked proper tension; they appeared shortened and did not vibrate freely in the act of phonation.

16th.—Vocal cords returning to their normal state, voice much improved; murmur more natural at left base and less crepitus.

25th.—Vocal cords had almost perfectly recovered.

March 4th.—Could take long walks of three or four hours without fatigue; voice normal. Left the villa on the 11th of March.

The following are the weights of this patient as taken with a reliable weighing chair:

January 31.—12 stone	$2\frac{1}{2}$	pounds.
February 3.—12	„	$3\frac{1}{4}$ „
„ 9.—12	„	$5\frac{1}{4}$ „
„ 12.—12	„	$6\frac{1}{4}$ „

February 16.—12	„	7½	pounds.
„ 19.—12	„	7½	„
„ 25.—12	„	7	„
March 4.—12	„	9½	„

A gain of seven pounds weight from the 31st January to the 4th March.

The beneficial effect of the pure bracing air of the hills, although only about 150 feet above the sea, was most remarkable in the present case, the improvement in the state of the larynx progressing together with that of the chest.

I have reported below in an abstract form seventy hospital cases, most of them occurring amongst my own out-patients at the Brompton Hospital, a few others having been sent to me by my colleagues. These cases were followed up as far as possible, but it is difficult to induce hospital out-patients to attend for a lengthened period; so that many were lost sight of at a time when I was most anxious to ascertain their state of health.

Remarks on the Cases.

The number of male patients far exceeds that of the female patients; there being 49 of the former, and 21 of the latter. I do not recollect exercising any selection in my cases of laryngeal phthisis, and my impression is that I noted down all cases of phthisis with laryngeal complication, while engaged with the present inquiry. I therefore believe there is a fair ground for concluding that men are more liable to laryngeal phthisis than woman.

The ages were recorded in 55 cases. They are as follows:

Number of cases aged 20 and under	.	.	.	4
„ between 20 „ 30	.	.	.	26
„ „ 30 „ 40	.	.	.	16
„ above 40	.	.	.	9

This seems to show that age has little or no influence as a

predisposing cause of laryngeal phthisis, as, according to Dr. J. E. Pollock,¹ there is a large preponderance of cases of phthisis between the ages of 20 and 30. Therefore, as far as age is concerned, the predisposition appears to apply equally to consumption, whether the throat be affected or not.

Out of the 70 cases, 8 exhibit no visible organic change in the larynx (slight congestion and redness in 2); these cannot be considered strictly as cases of laryngeal phthisis, but should be regarded as cases of functional defects of the larynx in phthisis. In 1 of these 8 cases the organ was apparently affected long before consumption set in; therefore, 7 only can be really considered as connected with tubercular disease of the lungs. The 8 cases are as follows :

Case No. 1.—Cavern.	Adduction of cords deficient.
„ 13.—Dulness, metallic crackling . . .	Ditto.
„ 21.—Crep., dulness, and crackling . .	Tension of cords deficient.
„ 30.—Cav. respiration and crackling .	Adduction of cords deficient (some redness of larynx, but cords normal).
„ 35.—Harsh respiration and prolonged expiration, shade of dulness; hæmoptysis and strong here- ditary predisposition . . .	Tension of cords deficient.
„ 36.—Cav. and crackling, dulness . .	Tension deficient, adduction normal.
„ 46.—Falling in at right apex, dul- ness, crepitus, hæmoptysis . .	Tension deficient.
„ 55.—Metallic click, coarse crepitus .	Ditto.

Tension of the vocal cords was therefore deficient in five cases, and adduction of the cords was deficient in three.

As to the actual change which had taken place in the larynx, in 31 of the 70 cases, the epiglottis was observed to be affected.

¹ 'Second Medical Report of the Hospital for Consumption,' 1863; and 'Treatise on Prognosis in Consumption.'

The following are the alterations this body was found to exhibit :

Case No. 2. Epiglottis, knotted and atrophied.

„	3.	„	concave at margin.
„	6.	„	thickened and studded with granulations.
„	8.	„	pendulous, misshapen from induration and softening.
„	11.	„	much swollen and pendulous.
„	12.	„	very red and subsequently granular, though not swollen.
„	16.	„	streaked from congested capillaries, but neither swollen nor granular.
„	22.	„	much swollen and curled, and exhibits tubercular granulations.
„	23.	„	small, speckled and appeared white (probably pale).
„	24.	„	swollen and pendulous.
„	28.	„	red, pendulous, and contracted into a cylindrical shape.
„	29.	„	pendulous, notched at apex, much swollen and speckled.
„	32.	„	pale, thickened, and lobulated.
„	33.	„	red, but not swollen.
„	34.	„	thickened and granular, becoming subsequently much swelled.
„	40.	„	swollen.
„	41.	„	indistinctly granular.
„	42.	„	much swollen and red, becoming pale, contracted, and indurated after scarification.
„	44.	„	red, streaked, and granular.
„	47.	„	pendulous and streaked.
„	48.	„	large and pendulous.
„	49.	„	red, granular, shortened, much swollen.

Case No. 50.	Epiglottis, very red, swollen.
„ 51.	„ pale and thickened.
„ 53.	„ pendulous, swollen and tubercular.
„ 54.	„ red, but not swollen. (Syphilitic taint.)
„ 56.	„ pendulous, red, and much thickened.
„ 57.	„ pendulous, swollen, and granular.
„ 61.	„ pendulous and tubercular.
„ 64.	„ very red, and congested.
„ 69.	„ pendulous, much swollen, and tubercular.

I find only thirteen cases (about 18·5 per cent.) of positive ulceration of the larynx, which appears to be a small proportion. Patients thus affected are, as a rule, older than those whose larynx are not ulcerated; the mean age of the cases of laryngeal disease without ulceration being 29·7 years, while that of the cases of laryngeal disease with ulceration is thirty-eight years. I think it is, therefore, fair to conclude that young consumptive patients are less liable to laryngeal ulceration than their seniors; and that this pathological change is superadded in a swollen and tubercular larynx, to some extent on account of the diminished vitality and want of power of nutrition depending upon age.

The other prominent organic change is, as a rule, a state of swelling of the arytenoid bodies and epiglottidean folds; the vocal cords being often obscured by the hypertrophied false cords, and reduced by congestion to a narrow rim. The white secretion I have referred to is frequently witnessed covering the swollen and tubercular mucous membrane.

As to the dysphagia and the alterations in the voice, and the cough: I have noted difficulty and pain in swallowing in fourteen cases, but this symptom may not have been recorded in others. It is caused, as a rule, by a swollen and tubercular condition of the epiglottis; but deglutition is sometimes interfered with, although the epiglottis remains in the healthy state. During the act of swallowing the larynx is

raised, the root of the tongue resting like a cushion on the epiglottis, which covers the laryngeal opening; thus the tongue, together with the constrictor muscles of the pharynx, presses upon the swollen epiglottis and larynx, and elicits in some cases intense pain. Loss of voice varied from mere weakness of the voice to hoarseness and complete aphonia. In some few cases the aphonia had an intermittent character; in most of them it was either complete or nearly so; but the patients could always speak in a whisper. Of the 70 cases recorded in the table, 59 are stated as exhibiting some alteration of the voice. In by far the greater number it was extremely feeble, in a few it was merely weak, and with a few others it was hoarse or cracked. In the other 11 cases, although the condition of the voice was not recorded, it may be concluded from the appearance of the larynx that the voice was probably either weak or otherwise affected in nine, and there was apparently nothing wrong with the act of phonation in two cases only.

SEVENTY CASES OF LARYNGEAL DISEASE IN PHTHISIS.

(M. indicates male; F. female.)

1.—M., æt. 25.

State of the—

Lungs.—Left apex, cavernous respiration, with loud friction sound and slight dulness; very little natural murmur. Right side, respiration harsh, with crackling.

Larynx.—Weakness of adductor muscles of cords only. Ill for 18 months; aphonia for the last 2 months.

2.—F., æt. 25.

Lungs.—Left, noisy respiration and moist râles, but little natural murmur. Right, cavernous, blowing, and dull at apex.

Larynx.—Epiglottis knotted and atrophied; larynx swollen; impeded movements of glottis.

3.—F.

Lungs.—Right, apex at back, a cavity, left, crackling at the side.

Larynx.—Epiglottis concave at margin; impeded movement of glottis; vocal cords red and swollen.

4.—M.

Lungs.—Cavernous.

Larynx.—Laryngeal mucous membrane wrinkled and relaxed; hoarseness for two months.

5.—M., æt. 30.

Lungs.—Left, respiration very feeble. Right, respiration weak; fine mucous râles. Posterior left, vesicular murmur right supra-spinal fossa; respiration deficient and very harsh, dulness. Hæmoptysis in streaks.

Larynx.—Raised papillæ base of tongue; arytenoid bodies and arytenoidian folds ulcerated; movements of glottis free, but vocal cords pale and lustreless; aphonia, or very hoarse for 8 or 10 months.

6.—M., æt. 43.

Lungs cavernous, with softening and physical signs of chronic second and third stage of phthisis.

Larynx.—Enlarged follicles base of tongue; epiglottis thickened and granular; larynx swollen; aphonia; temporary recovery of voice from treatment.

7.—M.

Lungs.—Left, apex dull, with diffused crepitus in front. Right, some crepitus, with deficient respiration.

Larynx.—Left side, deep ulceration, extending to posterior end of vocal cord; epiglottidean folds red and much swollen; apparent loss of power of tension; epiglottis pendulous; voice affected for 14 months, viz. hoarseness for 10 months, then aphonia.

8.—M., æt. about 26.

Lungs, March 15th, 1872 (Nice).—Left front, humid crepitus; murmur very deficient; shade of dullness at clavicle; posterior left, humid crepitus; some dullness throughout. Right, respiration fair.

Larynx, March 15th, 1872.—Epiglottis pendulous, out of shape from induration and softening; no possible view of larynx; voice returned to some extent under treatment; aphonia for about 6 months. Died March, 1873.

9.—M., æt. 40.

Lungs.—Left quite dull on percussion; crepitus. Right, shade of dullness; no hæmoptysis.

Larynx.—No cords visible; larynx thickened, pain on deglutition, and at one time serious dysphagia; aphonia for 22 months; pendulous epiglottis; suffered much from the throat for the latter 4 months.

10.—M., æt. 26.

Lungs.—Softening; right side dull, with tubular breathing. Left, tubular respiration; respiration very harsh, no dullness.

Larynx.—Rudiments of vocal cords only, the right presented an ulcerated-looking margin; larynx swollen, and the whole cavity contracted on attempts at phonation. Began losing voice about 10 months ago; aphonia for last 7 months.

11.—M.

Lungs.—Right side, some crepitus with bronchial breathing, and a shade of dullness near the sternum; crackling. Left, healthy. No syphilitic taint.

Larynx.—Epiglottis much swollen and pendulous, concealing larynx. Sudden aphonia about three months ago after exposure to a draft; for a fortnight in excruciating pain on swallowing alcoholic fluids or beer; with an effort some words were spoken in a tolerably natural voice.

12.—F., middle-aged.

Lungs, Oct. 31st, 1868.—Right, crackling at apex, with slight dullness. May 3rd, 1869.—Left apex, a large dry cavity; and right, dullness with crackling and very little respiration.

Larynx, Oct. 3rd.—Epiglottis very red; arytenoid bodies, folds and false cords red and swollen; secretion of white mucus in larynx; vocal cords much congested and their lustre lost. Singing power failed 16 months before; voice tolerably clear at this time. May 3rd, 1869.—A speckled appearance of

the larynx. May 22nd.—Epiglottis appeared granular, not swollen; movements of cord free; no swelling beyond slight apparent thickening.

13.—M., æt. 22.

Lungs.—Left and right, shade of dulness; left, supra-spinal fossa, metallic crackling. First signs of phthisis set in with pleurisy about 18 months ago.

Larynx.—Case of deficient tension and some congestion of vocal cords, with red and relaxed mucous membrane.

14.—M., æt. 44.

Lungs, end of Sept., 1868.—Right, shade of dulness; no obvious signs of phthisis. Nov. 9th, 1868.—Right, apparently distant amphoric respiration; right, back, decided dulness. Jan. 18, 1869.—Left, in front, respiration deficient and very harsh. Right, murmur feeble; at the back right, dulness throughout.

Larynx, end of Sept., 1868.—Laryngeal space much reduced from thickening; mere rudiments of vocal cords left, at posterior ends of which were deep ulcerations. January 18th, 1869.—Right epiglottidean folds and arytenoid bodies much swollen; some relief from scarification. Complete aphonia for last three years. (Had partial aphonia 8 or 9 years ago; relieved occasionally, especially in the summer, till 3 years ago.) Pain in larynx till February, 1869, when deglutition became less free than before.

15.—M., æt. 35.

Lungs.—A large dry cavern, extending from apex to third rib on right side (dulness and cavernous respiration).

Larynx.—Back of tongue red and injected; follicles enlarged; epiglottis normal; arytenoid bodies and folds swollen; false cords swollen and true cords less glistening than usual; loss of tension of cords, and speech carried on in a low, harsh tone.

16.—M., æt. 37.

Lungs, 30th Oct.—Mucous râles; no dulness; no cavernous or bronchial breathing. Nov. 7th.—Shade of dulness, left apex. 28th Nov.—Slight but distinct dulness, left apex, in front. 13th Feb., 1869.—Left apex, respiration tubular with slight crepitation and dulness, and amphoric breathing at right supra-spinous fossa.

Larynx, 30th Oct.—Thick and white mucous secretion over epiglottis and folds, visible also in the larynx; no congestion or granular appearance; no pain, no hoarseness, but a constant desire to clear the throat. 13th Feb.—Epiglottis streaked deeply from congested capillaries; walls of larynx red, but no longer any white secretion to be seen; voice occasionally weak, no aphonia.

17.—M., middle-aged.

Lungs, 3rd Nov., 1868.—Bronchial breathing; crackling at apex; right fine crepitus and mucous râles extensive; no dulness.

Larynx, 3rd Nov.—Back of tongue red, with thick white secretion upon it;

larynx particularly pale and secreting thick white substance; vocal cords healthy.

18.—M., æt. 38.

Lungs.—Left, in front, respiration harsh, with thick mucous râles; no other signs; questionable signs of phthisis.

Larynx swollen and vocal cords not seen; on attempt at phonation the folds of mucous membrane project towards median line; white secretion on the posterior wall of larynx, the removal of which brings into view a pale and relaxed mucous membrane. Sudden aphonia 6 weeks ago, and no return of the voice.

19.—F., æt. 22.

Lungs, Oct., 1868.—Amphoric respiration right apex, with crackling and dulness (cav.). 14th Dec.—Symptoms urgent; pulse 144; temperature under tongue 103.2° F.

Larynx, 9th Oct., 1868.—On attempting phonation the cords did not meet well in median line; ulcerated raised patch on posterior wall, studded with enlarged follicles. Aphonia for 3 months; sometimes voice broke out hoarsely for a minute or two. 16th Nov.—Mischiefs in larynx increasing; ulcerations distinctly seen.

20.—M., æt. 51.

Lungs, 18th Nov.—Shade of dulness, left apex. 7th Dec.—Decided dulness; respiration harsh, left and right. 17th Dec.—Left apex dull to third rib; distant metallic sounds. Severe hæmoptysis 4 years ago.

Larynx.—A good view of larynx with assistance of ice bag to throat and ice in mouth; larynx swollen near posterior end of left cord, a raised ulcerated patch stretching across posterior wall of larynx but not reaching right cord; enlarged follicles on the raised membrane. Had a cracked, reedy voice; words often dropped; movement of the cords appeared free.

21.—F., æt. about 28.

Lungs, 19th Nov., 1869.—At upper third left side, in front, deficient murmur; abundant mucous râles. Dec. 9th.—Left, crepitus; right, crepitus and dulness under clavicle. Dec. 23rd.—Left side, crackling. Jan. 22nd.—Physical signs of softening still were obvious.

Larynx, Dec. 2nd.—Voice became natural after an application of a solution of nitrate of silver to larynx. Dec. 11th.—Epiglottis and larynx red and swollen; voice natural. Had aphonia 3½ years previously for about 10 weeks. A severe attack of hæmoptysis 4 years ago.

22.—M.

Lungs.—Left, respiration tubular; right, mucous râles and deficient respiration; murmur; no dulness; hæmoptysis and expectoration considerable.

Larynx.—Epiglottis much swollen and curled; exhibits tubercular granulations; arytenoid body much enlarged, ragged, and speckled; laryngeal space considerably narrowed; such pain that he could hardly swallow; aphonia. Voice

returned a few days after application to me, and deglutition improved, but thinner and weaker in other respects.

23.—F., æt. 27.

Lungs, first examination, about 20th November, 1868.—Shade of dulness, left apex. Dec. 2nd.—Harsh breathing (physical signs not clearly those of phthisis, but the state of throat left little doubt).

Larynx, about 21st Nov.—Has a high-pitch cracked voice; cords approach, though not freely, on phonation; epiglottis small, speckled, looks white (pale); some white secretion on false cords; want of power in adductor muscles of cords (state of larynx symptomatic of phthisis).

24.—M., æt. 38.

Lungs.—Left, in front, consolidation and softening; posterior left, cavern; right, also dulness and cavernous respiration. Father, mother, and brothers died of phthisis. Began coughing last Christmas; in good health before.

Larynx.—With use of ice-bag. Epiglottis observed swollen and pendulous; arytenoid bodies and folds considerably swollen, and secretion of white substance issuing between arytenoids; no visible ulceration; swallowing occasionally produces spasm of the glottis. Voice first failed 3 months ago. At present aphonia, though voice returns slightly at times.

25.—M., æt. 25.

Lungs, 19th Dec., 1868.—Left side; some dulness. 12th Jan., 1869.—Left, loud dry cavernous breathing, with occasional click.

Larynx, Dec. 11th, 1868.—Both sides of larynx very much swollen and covered with white mucus; epiglottis not thickened; swelling punctured. Dec. 16th.—Less swelling, but white mucus continues present; an indistinct ulceration left. 21st Dec.—Epiglottis not swollen, but contracting in some respects; much white mucus. Jan. 25th.—A conical tumour or growth on right arytenoid body. Supraglottic region softening. Loss of voice about 11 months before applying. Had had no food for 4 days when I saw him, from inability to swallow, and was starving; but drank some milk and sherry after scarification of the larynx. Died Jan. 25th, 1869.

26.—F.

Lungs.—(Cavern and softening). Left, some coarse crackling; right, amphoric breathing and dulness at apex to third rib; falling-in of chest at right apex; a chronic case.

Larynx.—Sound of voice imperfect; arytenoid bodies fail to approximate sufficiently in the act of phonation; a raised patch between arytenoids is covered with white secretion, on removal of which follicles like spiculæ are seen.

27.—M., æt. 24.

Lungs.—Consolidation and softening; a large tumour, feeling tense and elastic, but without fluctuation, just under left false rib, measuring in the

erect posture, $4\frac{1}{2}$ inches horizontally and $2\frac{1}{2}$ inches in breadth. This resulted from his being squeezed between a waggon and dust-cart in Sept., 1866.

Larynx, 4th Jan., 1869.—Arytenoid bodies and folds much swollen and secreting white mucus; epiglottis not enlarged; an ulceration on left false cord extending to true cord on that side; aphonia for nearly a year. 16th Jan.—Slight return of voice; ulceration appeared to have a tendency to cicatrize; felt better.

28.—M., middle-aged.

Lungs, 16th Jan., 1869.—Left, respiration deficient and harsh; right, shade of dulness at apex. Phthisis probable.

Larynx, 16th Nov., 1868.—Epiglottis red, pendulous, and contracted into a cylindrical shape; mucous membrane not readily visible on account of epiglottis, but appeared thickened. Lost voice on 8th July last, when he coughed up about half a pint of blood. Voice began improving about end of October following. Stated that since 8th July he had gained 11 lbs. weight.

29.—M., æt. 22,

Lungs, 23rd Jan., 1869.—Left, dry cavernous respiration from apex to third rib, with occasional crackling; from third rib to base distant cavernous breathing. Right, amphoric sounds front and back. Severe attack of inflammation of the lungs about 6 years ago.

Larynx, 23rd Jan., 1869.—Back of tongue very red and follicular; epiglottis pendulous, notched at apex, much swollen, and speckled; exhibited white secretion, which was also seen oozing out of larynx; great pain on deglutition. Jan. 27th.—After scarifying the epiglottis obtained glimpse of larynx, which exuded a white substance in abundance. Aphonia since the end of last August; began with hoarseness for 2 or 3 months. Two years ago, hæmoptysis; was first troubled in singing, but was able to sing till May last.

30.—F., æt. 30.

Lungs, 5th Feb., 1869.—Right apex, in front and behind, cavernous respiration and crackling.

Larynx, 5th Feb., 1869.—Larynx red, with vocal cords healthy; want of power in adduction; weak, nervous, subject to fainting. Voice lost 4 months ago; it returned after Faradization, and continued fair, though rather weak, till March 4th.

31.—F., æt. 23.

Lungs.—Left, base, shade of dulness; right, cavity, frequent hæmoptysis. Mother died of phthisis.

Larynx.—Epiglottis normal; on posterior wall of larynx a cluster of raised follicles. Aphonia for last 15 or 16 months; began coughing about 3 years ago; probably adduction deficient.

32.—F., æt. 22.

Lungs, 17th Feb., 1869.—Abundant crepitus left and right, murmur bron-

chial. (Taking into account laryngeal symptoms, softening probably in progress.)

Larynx, 17th Feb., 1869.—Root of palate pale, and back of pharynx exhibited small superficial excoriations; uvula out of shape and speckled with granulations; epiglottis pale, thickened, and lobulated; much dysphagia; no relief from scarification; epiglottidean folds appeared swollen.

33.—M., æt. 32.

Lungs, 20th Feb., 1869.—Left, tubular respiration and moist crepitus at base. Had suffered from hæmoptysis.

Larynx, 20th Feb., 1869.—Ulceration at base of both arytenoid bodies externally to larynx, so that they look like pedicellated tumours, are granular and very red; epiglottis red, but not swollen; no pain on deglutition. Sudden aphonia on 8th January last; coughs since last November. Treatment—Liquor Arsenicalis; cod liver oil and iodized oil to larynx. 25th Feb.—Throat felt much better; voice returned, though very hoarse.

34.—M., æt. 43.

Lungs.—Began, with uncertain signs of phthisis, on 27th June, 1868. Oct. 2nd, 1868.—No positive signs of phthisis yet. Nov. 4th, 1868.—Metallic crackling, first sign, observed.

Larynx, 27th June, 1868.—Follicles back of tongue red and enlarged; epiglottis thickened and granular; mucous membrane of larynx much swollen. Loss of voice in October, 1867; began with sore-throat, which lasted for 3 months before aphonia became complete. (Never recovered perfectly from pleurisy in 1866; slight hæmoptysis once or twice in March, 1868.) Oct. 16th, 1868.—Epiglottis much reduced in size, but still speckled with granulation. Died 10th Nov., 1869. (See 'Clinical Notes on Diseases of the Larynx,' p. 99.)

35.—F.

Lungs, 5th March, 1869.—Harsh respiration and percussion note not very clear. Two slight attacks of hæmoptysis last 6 weeks. Mother, three sisters, and two brothers died of consumption. 26th March.—Left, harsh respiration; no dulness. Right, harsh breathing, prolonged expiration, and shade of dulness at apex.

Larynx, 14th Jan., 1869.—Cords look quite healthy, and adduction quite free, but loss of tension; the whole larynx contracted in efforts to produce sounds. April 5th, 1869.—Has improved gradually; voice now natural, and can even sing a little. Treated by Faradization.

36.—M. 21.

Lungs.—Right apex, in front, a shade of dulness; cavernous respiration with crackling to third rib; base, respiration vesicular, with some crepitus; back, right side, unmistakable signs of cavern and consolidation ('Clinical Notes on Diseases of the Larynx,' p. 134).

Larynx, 19th March.—Defective tension and abnormal adduction, producing cracked voice. Recovery by Faradization, and could sing the scale without difficulty on the 8th July following.

37.—F., æt. 27.

Lungs.—Cavernous respiration and prolonged expiration, left side, between third and fourth rib; right, falling in (slight) at apex and crackling.

Larynx, 1st May, 1869.—No laryngeal swelling; no swollen follicles; no ulceration; supraglottic mucous membrane red and relaxed, especially right side; none or mere remnants of vocal cords visible; posterior wall of pharynx coated with white secretion; phonation required great effort, and apparently carried on with false cords. 1st Nov.—Left, arytenoid body red and swollen; pain in inside of left ear; epiglottis rather pendulous, but appeared healthy. Only temporary benefit from Faradization.

38.—M., æt. 36.

Lungs, 6th April, 1869.—Left, crepitus; prolonged respiration. Right, dulness and tubular respiration. Hæmoptysis, 18 months ago.

Larynx, April 6th, 1869.—Epiglottis partly pendulous; larynx congested; congestion and loss of power in vocal cords. May, 1869.—Left cord appeared uneven and atrophied; a chink between cords on attempting phonation. Aphonia for 6 months, either total or voice very hoarse. Affection very sudden; patient went to bed quite well, and was voiceless next morning.

39.—F., æt. 37.

Lungs, 18th May, 1869.—Left, fine crepitus; shade of dulness under clavicle. Right apex, breathing rather bronchial. Slight hæmoptysis once or twice. Mother died of phthisis.

Larynx, 18th May, 1869.—Had a string of ulcerations on right epiglottidean fold, which was swollen; adduction too imperfect to allow of phonation. Sept. 21st.—Voice returned at times. Oct. 5th.—Health much improved and voice much steadier. Loss of voice from about 16th April. Treatment with inhalation of iodine.

40.—M., æt. 19.

Lungs, 26th Jan., 1869.—Right apex, tubular breathing; posteriorly right, dulness; left, normal. Hæmoptysis, Dec., 1868; lost 11 lbs. weight in less than 5 months.

Larynx, 14th May, 1869.—Supraglottic region very red, and space contracted from swollen arytenoid bodies and folds. 31st May.—Epiglottis swollen. June 5th.—Adduction incomplete; voice fails now and then in speaking. June 25th—Voice now normal.

41.—M.

Lungs.—Left, in front, distant cavernous sounds; dull posteriorly with cavernous breathing and crackling; no hæmoptysis: no hereditary taint.

Larynx.—Base of tongue follicular; no ulceration; redness and slight

laryngeal thickening; epiglottis indistinctly granular; right arytenoid overlapped left in phonation; cords part when required to vibrate, and voice weak and hoarse in consequence (want of power of tension); no syphilitic taint (an excellent view with the oxyhydrogen light).

42.—M., æt. 27.

Lungs, 29th May.—Left, dull and falling-in, with loud dry amphoric respiration from apex to nipple. Right, dry cavernous respiration.

Larynx, 29th May.—Epiglottis much swollen and red; voice unaffected, indicating normal vocal cords. 2nd July.—Epiglottis secreting white mucus. June 4th.—Epiglottis had become pale, with a tendency to contract. Had been scarified freely on several occasions. June 7th.—Epiglottis smaller, becoming harder and granular. 23rd June.—Epiglottis so far contracted as to allow sight of larynx; cords appeared normal; much white secretion. June 26th.—Voice weak; dysphagia; never any aphonia. Died 11th July, 1869.

43.—M., æt. 37.

Lungs, 29th May, 1869.—Left apex, dulness; slight falling in, absence of murmur and crackling. Right, very deficient respiratory murmur.

Larynx, 15th May.—Right supraglottic region very much swollen; left normal; edges of cord appeared ragged. Complete aphonia since Christmas last. 29th May.—A conical growth seen on right epiglottidean fold; whole larynx very red; no swollen epiglottis; no dysphagia. 2nd June.—Much relief from puncturing right false cord; there appeared a breach of continuity at the seat of a former puncture, as if softening was setting in. June 16th.—Softening appeared commencing on the left of larynx. 25th June.—Left cord much swollen.

44.—M., æt. 31.

Lungs, 29th May, 1869.—Left, crackling; shade of dulness at apex; dulness also right. Hæmoptysis 2 months ago; in ill-health for 12 months. 11th Oct.—A cavity.

Larynx.—Epiglottis red, streaked, granular; larynx very red; arytenoid bodies and folds much swollen; white exudation between arytenoid bodies; voice rather weak, but otherwise natural; occasionally slight pain in swallowing the saliva.

45.—F., æt. 29.

Lungs.—Right apex, in front friction sounds, deficient and harsh respiration. Posterior left harsh, with humid crepitation; some dulness right.

Larynx, 4th June, 1869.—Larynx granular and rather pale; secreting white thick mucus. June 12th.—On attempting phonation, posterior part of larynx drawn up, concealing the vocal cords, but arytenoid bodies move freely towards each other (weakness of tension). June 14th.—Remarkable absence of sensibility of larynx to a strong electrical current. June 30th.—Loud whisper

occasionally after Faradization. There had been total aphonia for 4 months. Deglutition difficult at first, but improved under treatment.

46.—F., middle-aged.

Lungs.—Falling in, with some dulness at right apex; humid crepitation, both left and right side. Hæmoptysis 2 months before.

Larynx.—Case of deficient tension of the vocal cords, without organic disease of the larynx (see p. 13).

47.—M.

Lungs.—Physical signs not reported, but case entered as tubercular; pulse 104; temperature 101.2° F. under tongue.

Larynx, 7th July.—Epiglottis pendulous and streaked, but larynx could be seen red, though not exactly thickened, and exhibiting no white secretion. July 12th.—Vocal cords red and appeared atrophied, approximated tolerably well and vibrated to some extent. For about 18 months either very hoarse or only spoke in a whisper.

48.—M., æt. 32.

Lungs.—Loud tubular breathing and amphoric resonance both apices (Cavity in both apices).

Larynx, 24th Sept.—Larynx red; large pendulous epiglottis, concealing glottis, but a round elevation is visible on left arytenoid body. Very hoarse for last 4 months. 1st Oct.—Laryngeal swelling increased, and covered with milky secretion; it receded before scarifier, and bled only a little; acute pain in the left ear; vocal cords seen red and congested. Said he felt something break in throat two days ago, and expectorated much blood and matter. 25th Nov.—Stated by letter that he was worse and apparently sinking. Treatment by scarification and iodized oil.

49.—F., æt. 24.

Lungs.—Left, respiration apparently tubular; right, dull, flattened, and the seat of cavernous breathing.

Larynx, 6th Oct., 1869.—Epiglottis red, shortened, and much swollen, granular; arytenoid bodies and false cords much swollen; left false cord apparently pitted from softening; much dysphagia; could only swallow a little milk; almost starved. Aphonia complete for 6 weeks. 13th Oct., 1869.—Softening progressing in supraglottic region; epiglottis shortening; rather less dysphagia.

50.—M., æt. 39.

Lungs, 1st June, 1869.—Left, deficient murmur and crackling. Sept. 21st.—Left, in front, bronchial, deficient, and harsh murmur; right, distant, dry, cavernous respiration. Oct. 5th.—Left moist, crepitation throughout, no dulness; right, dull at clavicle. Fell ill about Oct., 1868; an attack of hæmoptysis June, 1869.

Larynx, 21st Sept., 1869.—Epiglottis, vocal cords and supraglottic region

very red and swollen; softening appeared in progress; much white mucus secretion; pain on deglutition referred to inside of ears; throat affection had lasted three months, and was worse now. 5th Oct.—Both arytenoid bodies much swollen, and covered with white secretion. 13th Oct.—Voice weak and falling into a whisper. 23rd Nov.—Could only swallow with great pain. Relief from inhalation of iodine.

51.—M., æt. 49.

Lungs.—Cavities in both lungs.

Larynx.—Both arytenoid bodies, especially the left, very much swollen, exhibited white granulations; epiglottis pale and thickened; an ulceration on posterior wall of larynx, and thick white mucus present. Aphonia for 5 or 6 months.

52.—M., æt. 18.

Lungs.—Cavity in both lungs.

Post-mortem examination.—Pharynx somewhat congested; vocal cords softened; laryngeal mucous membrane terminating abruptly with a plug of mucus at rima glottidis.

Larynx.—Complete aphonia for 4 months; on admission into the Brompton Hospital the larynx was seen to be retracted, white mucus present, and paralysis of tensor muscles of cords. (Notes favoured by Mr. N. R. Smith, Clinical Assistant, Brompton Hospital.)

53.—M., æt. 56.

Lungs.—Loud, dry, cavernous breathing right side; left, respiration loud and vesicular. In fair general health.

Larynx, 18th Nov.—Epiglottis swollen, pendulous and tubercular, though not softening. 24th Nov.—Epiglottis larger, exhibited an ulceration; laryngeal mucous membrane appeared thickened and very red; no vocal cords visible. 29th Nov.—Epiglottis much less swollen, and ulceration cicatrising. A small though rather deep ulceration on the posterior wall of larynx. Was suddenly seized with sore-throat after a chill 4 months ago; at present, aphonia; voice occasionally became very hoarse. Pain in larynx only felt on swallowing or coughing.

Treatment.—Scarification and inhalation of iodine; iodide of potassium and syrup of iodide of iron internally.

54.—M.

Lungs.—Both sides, humid crepitus; right, tubular respiration and falling in of chest. A syphilitic sore about four years before, but no secondary symptoms, except night headache.

Larynx (with syphilitic taint).—Pharynx and back of tongue very red. Uvula nearly white from apparent infiltration with tubercular matter; softening at base, and nearly ulcerated through a quarter of its breadth. Infiltration extended to velum; epiglottis red, but not swollen; right vocal cord

healthy; an ulceration at posterior end of the left cord. Pain followed deglutition. Aphonia for the last five weeks.

55.—M., æt. 22.

Lungs.—Left, metallic click; right, coarse crepitus.

Larynx.—A cracked voice so long as he can remember, viz: when about æt. 3, after measles. Arytenoid bodies rather overlapped each other in phonation, but tension deficient. Adductors instinctively compensated for weakness of tension. (This may have been unconnected with phthisis, but is sufficiently interesting to be placed on record.)

56.—M., æt. 22.

Lungs.—Phthisis, cavernus (no notes of physical signs).

Larynx.—Epiglottis pendulous, red, and highly thickened; arytenoid bodies and folds just visible, covered with white mucus, and apparently undergoing softening. Dysphagia acute, and much pain on deglutition felt inside left ear. Great relief from scarification of epiglottis, and could afterwards swallow two wineglassfuls of milk and sherry without trouble.

57.—M., æt. 47.

Lungs.—Left, dulness and cavern under sterno-clavicular articulation; right, loud murmur and vocal resonance.

Larynx, 9th June.—Pharynx red; epiglottis swollen. 21st July.—Epiglottis pendulous and tubercular; pain in larynx and difficult deglutition; voice hoarse, and at times aphonia. 23rd June.—Throat better, deglutition easier, and less hoarseness.

58.—M., æt. 43, butcher's assistant.

Lungs, Dec. 28th, 1870.—Left, front, no murmur, indistinct friction sounds, no obvious dulness back, low indistinct humid crepitation; right, respiration harsh at base. Three or four attacks of hæmoptysis in streaks; coughs and expectorates much; much dyspnoea. Phthisis not confirmed, though probable; at all events, congestion of lungs. Great pulmonary and laryngeal exertion in calling out weight of meat from the scale.

Larynx, June 1st.—Cannot see vocal cords; indeed, nothing but mere edges or rudiments of true cords to be discerned. Is very hoarse. Probable case of simple congestion of vocal cords.

59.—F., æt. 28.

Lungs, July 7th, 1870.—Left, shallow respiration, dulness, with crepitation; right, respiration deficient and bronchial.

Larynx, 22nd Sept., 1870.—Larynx very red and swollen.

60.—M., æt. 30.

Lungs, Sept. 20th, 1870.—Left and right, fine crepitus, no dulness.

Larynx, Sept. 20th, 1870.—Arytenoid bodies swollen and infiltrated with tubercular matter. On attempting phonation, incomplete adduction; cords lost their lustre, and appeared uneven. Ill for eighteen months, and lost voice

twelve months ago. On first applying, aphonia was very complete. In December, 1870, voice improved, but feeble.

61.—F., æt. 17.

Lungs, June 9th, 1870.—Left, dull, loud, cavernous respiration; right, vocal resonance.

Larynx, 9th June, 1870.—Pharynx red; epiglottis swollen; pain in larynx and dysphagia; voice hoarse at the time. 23rd June.—Throat better; deglutition improved. 21st July.—Epiglottis pendulous and tubercular. Iodine inhalation, syrup of iodide of iron, and chlorate of potash.

62.—M., æt. 20.

Lungs, Oct. 12 (1869).—Left, crackling and dull. 24th March, 1870.—Left, dull, no cavity, or crepitation, or click; right, murmur deficient, but otherwise fair. Hæmoptysis in streaks before applying. Brother died of phthisis.

Larynx, 9th Nov., 1869.—Larynx very red; arytenoid bodies and folds swollen and red; voice weak and hoarse. 23rd Nov.—Voice much better, and could also sing. 30th Nov.—Improvement continued. March 24th, 1870.—Apparently no return of the laryngeal affection.

63.—M., æt. 25.

Lungs, Nov. 3rd, 1868.—Left, no dulness, but dry crepitation, very distinct; right, crepitation merging into crackling, no dulness, several severe attacks of hæmoptysis. Nov., 1869.—Left, respiration harsh; right, slight crackling, dyspnoea.

Larynx, Nov., 1868.—Transparent mucous secretion over arytenoid bodies, and false cords; no granular or red appearance; parts rather paler than usual; weakness of the tensor muscles. Nov., 1869.—No laryngeal symptoms.

64.—M., æt. 19.

Lungs.—Left, humid crepitation, murmur very deficient, no dulness; right, loud bronchial wheezing.

Larynx.—Vocal cords could not be seen owing to the congested state of the larynx; epiglottis very red and congested (simple laryngitis, probably passing into laryngeal phthisis).

65.—F., æt. 32.

Lungs.—Very fine crepitation, right; left, normal.

Larynx.—Tubercular spiculæ (follicles) on posterior wall of pharynx, which is thickened; deficient power in adduction of cords, which appeared also relaxed.

66.—M., æt. 33.

Lungs.—Left, very deficient murmur and coarse crepitation; right, a large dry cavity. In ill health for 12 months. No hereditary predisposition to phthisis.

Larynx, March 21.—Pendulous epiglottis; arytenoid bodies very much swollen; aphonia nearly complete.

67.—M., æt. 33.

Lungs.—Crepitus and cavity right apex.

Larynx.—False cords red and swollen; true cords appeared red and had lost their lustre; voice very weak, with a tendency to fall into a whisper.

68.—M., æt. 38.

Lungs, 2nd Feb., 1871.—Some harshness; no expansion or murmur left base; never any hæmoptysis. Mother and two sisters died of phthisis.

Larynx, 2nd Feb., 1871.—Right side of larynx very much swollen, and right vocal cord not visible; left side of larynx congested; aphonia.

69.—F., æt. 37.

Lungs, May 11th, 1871.—Left, some fine crepitus and prolonged expiration; right, respiration fair, though harsh.

Larynx.—Epiglottis pendulous, much swollen, and tubercular; no syphilitic taint; softening apparently in progress on the posterior wall of pharynx (a clear case of laryngeal phthisis, although the physical signs of the chest were still uncertain).

70.—F., æt. 28.

Lungs, Sept. 2nd, 1869.—Right, dull, and bronchial respiration. Oct. 10th, 1869.—Decided large, dry cavity, right apex. Jan. 6th, 1870.—Left apex, a large, dry cavity; right, dry cavity to third rib, sinking in at apex. Hæmoptysis in September, 1869.

Larynx, 28th Nov., 1869.—Both arytenoid bodies were swollen, the left least so; vocal cords healthy. 6th Jan.—Vocal cords healthy, but a raised follicle distinctly seen at back of larynx below the arytenoid bodies; no pain or inconvenience in the larynx beyond a frequent need to clear the throat; the follicles at base of the tongue swollen and red; she thought her voice had been hoarse with occasional aphonia for about ten years. 20th Jan., 1870.—Felt nothing now amiss with the throat; voice quite natural, though perhaps slightly cracked.