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THE SUBMUCOUS AREOLAR TISSUE OF THE LARYNX, AND ITS SIGNIFICANCE IN THE SPREAD OF OEDEMA.

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(Plates VI.–IX.)

The subject of oedema of the larynx has been brought somewhat prominently before my notice by the opportunity recently given to me by my friend, Dr. Harvey Littlejohn, of studying a fatal case of this condition. The sudden onset of acute oedema producing symptoms of a grave nature, which may terminate in death, is an occurrence of sufficient surgical importance to merit more than a passing notice being given to its consideration. Indeed, a rapidly developing oedema in this region must be regarded as a matter of vital importance, and the possibility of its sudden fatal termination should always be kept in mind. Cases are reported in which death has taken place from asphyxia, not only within a few hours, but within a few minutes of the first onset of symptoms of dyspnœa.

From a perusal of the text-books and monographs dealing with oedema of the larynx, it is evident that various classifications have been adopted by different writers. It is not my intention to enter into this part of the subject. It is sufficient for our purpose to bear in mind that oedema of the larynx is merely an objective clinical phenomenon, and not a disease. It consists in a fluid exudation into the loose submucous areolar tissue, and it may be of a simple serous or of a sero-purulent character. The loose connective tissue is present to a greater or less extent in different parts of the larynx, and the object of this paper is to demonstrate the arrangement of this tissue and to point out its clinical significance.

Oedema of the larynx may be either inflammatory or non-inflammatory in origin, the former being by far the more frequent; it may be secondary to previously existing disease in the larynx, or it may arise in connection with inflammations at the base of the tongue and in the fauces and pharynx, while more rarely it may result from an inflammatory process arising in the cellular tissue or glands of the neck. The non-inflammatory form may occur as a local manifestation in renal and cardiac disease, or it may result from obstruction of the veins of the neck, produced by various causes.
The three following cases present some features of interest, and serve as a suitable introduction to the more anatomical portion of the paper:—

Case 1.—The patient, J. A., aged 34, a stone-mason to trade, had apparently enjoyed good health until the onset of his present illness. In the early part of 1899, he began to suffer from slight hoarseness of voice, and in the month of April of that year he sought special advice. It is unfortunate that no facts concerning the local appearances then observed in the larynx are obtainable, nor is there any reliable information to be had concerning the progress of the patient during the following summer. His friends, however, were under the impression that he suffered from "consumption." On the morning of Monday, September 4, 1899, about six months after the examination of the larynx had been made, the patient suddenly died. The facts regarding his condition immediately prior to his death, as related by his friends, are briefly as follows:—Up to and upon Friday, September 1, he was able to carry on his work as a mason in the neighbourhood of Edinburgh, apparently without personal inconvenience. On the following day (Saturday) he came into town with the object of paying a visit to the Royal Infirmary on the Monday following. He appeared to be in fairly good health and spirits, complaining of nothing save hoarseness. During Sunday night, however, for the first time, his rest was somewhat broken by occasional attacks of difficulty in breathing. At breakfast on Monday morning he did not feel well, but expressed himself as quite able to go out. Within half an hour of breakfasting, the patient was dead, a sudden and severe attack of dyspnœa having supervened, death from asphyxia taking place before any surgical assistance could be obtained.

A post-mortem examination was made by Dr. Harvey Littlejohn, who has kindly furnished me with the following short note of the general appearances which presented themselves:—The body was fairly well nourished; the heart was normal; the left pleural cavity showed some old adhesions, and in both lungs there was evidence of tuberculous disease, although in neither was there a cavity present. The liver, spleen, and kidneys were normal. The brain was not examined.

The larynx presented the following features of interest (Plate VI. Fig. 1):—The upper aperture was almost completely occluded by great tumefaction, so that in the recent state a probe could only be passed with some difficulty downward into the interior of the larynx. The swelling and consequent occlusion was due to an oedematous infiltration of both ary-epiglottic folds, and of the mucous membrane covering the arytenoid cartilages. In the latter situation the oedema extended downwards for some little distance upon the posterior surface of the two cartilages, its lower limit reaching as far as the upper part of the posterior surface of the cricoid cartilage. On each side the swelling extended outwards from the ary-epiglottic folds into the pyriform sinuses, while mesially the inner surfaces of the two folds lay in contact.1

1 This larynx was shown at the meeting of the Laryngological Society of London in May 1900.
In this way the respiratory channel was so completely occluded that the ventricular bands and the true vocal cords, with the intervening glottic chink, were invisible from above. The epiglottis maintained its normal appearance and contour.

In order to ascertain the condition of the interior of the larynx, it was necessary to divide the posterior wall vertically. Examination then revealed the presence of an ulcer of considerable size upon the mesial aspect of the left ary-epiglottic fold, and also upon the left ventricular band. The left true cord was destroyed throughout almost its entire length, while a small granulation formed an excrescence close to its posterior extremity. The right true cord also showed evidence of some superficial ulceration. There was no glottic or subglottic oedema.

Although I was unable to discover the presence of the tubercle bacillus in a small piece of tissue removed from the edge of the ulcer situated upon the left ventricular band, the history of the case, the macroscopic appearances of the larynx, and the associated pulmonary condition, sufficiently established a diagnosis of laryngeal tuberculosis. Death, undoubtedly, was due to asphyxia resulting from a rapid serous exudation, which produced sudden and marked oedema of the loose submucous connective tissue in the ary-epiglottic folds and over the arytenoid cartilages.

The case, thus somewhat briefly recorded, presents certain features of considerable interest. A localised chronic oedema, occurring in cases of laryngeal tuberculosis, is, as we are aware, by no means infrequent; indeed, it is a common manifestation in that disease. We have no reliable information regarding a previously existing chronic oedema in this case, as no examination of the larynx had apparently been made for some considerable time before death. The mere fact, however, that the patient was able to carry on his work and move about up to the last, without any apparent respiratory trouble, sufficiently justifies us in concluding that there was an absence of any marked swelling from that cause. On the other hand, the sudden onset of great dyspnoea points to the development of an acute oedema, which more or less completely occluded the respiratory passage, and rapidly terminated in death. It is possible that some slight oedema of a chronic nature was already present, and that a sudden acute exacerbation supervened upon it.

It is necessary, therefore, to bear in mind the possibility of so grave a complication arising in the course of laryngeal tuberculosis. While it is a well-recognised fact that acute inflammatory oedema may develop in a larynx affected with tuberculous, syphilitic, or cancerous disease, some diversity of opinion apparently exists amongst different authorities as to its frequency. Trousseau states that oedema of the larynx is the most frequent termination of laryngeal ulceration. In tuberculous cases, he says,
death results from œdema before the last stage of marasmus is reached, but it is not apparent whether he implies that it is usually of such a nature as to bring about a sudden termination, as in this case. A perusal of the cases of acute œdema, reported during the last ten to fifteen years, leads one to conclude that in tuberculous ulcerations, at any rate—and it is this disease which we are at present considering—a fatal complication of this nature is comparatively rare. The study of the literature of this subject has been greatly facilitated by the publication of an interesting paper by Clarence Rice of New York. This writer has tabulated a series of forty-one cases of acute œdema, as reported in the journals during the decade 1887 to 1897. In twenty-two instances, or in one-half of the cases, the dyspnœa is described as being of a severe or of an acute suffocative type. Of these, six died during the acute attack, while three other cases proved fatal, but not apparently from asphyxias. In the remaining thirteen cases, in which the respiration was seriously interfered with, recovery took place. If we examine into the possible etiology, not only of the twenty-two acute cases, but also of the remaining nineteen, of a rather less severe nature, we find that tuberculosis of the larynx is described as only being present in one instance, and in this case the exciting cause of the œdema was stated to be iodide of potassium. In one other case, where the œdema produced an acute suffocative dyspnœa, the patient, who was suffering from laryngitis, was described as being "a tuberculous subject." This fact must, however, be kept in mind in studying the tables published by Rice, that in many instances merely the term "catarrhal" is employed to describe the pre-existing laryngeal condition. In the absence of more definite information on this point, we cannot, of course, altogether exclude the possibility of the catarrh being of a tuberculous nature. Since the publication of Rice's paper, I have been able to obtain reference to fourteen additional cases of severe dyspnœa from laryngeal œdema, published previous to 1902, and in none of these was tuberculous disease of the larynx present. These facts would tend to show that the rapid onset of severe dyspnœa, terminating in sudden death, or necessitating the immediate performance of tracheotomy, is not a common complication of laryngeal tuberculosis. The case here recorded and figured must therefore be regarded as one of comparative rarity.

Case 2.—A male, æt. 49, presented himself at Dr. M'Bride's department in the Royal Infirmary, on May 8, 1896, with the history that on the previous day his throat had commenced to give him pain. Up to that time he had enjoyed good health. The pain was considerably aggravated on swallowing, and he experienced the sensation as if a foreign body was in the throat. He had no dyspnœa or hoarseness, the voice remaining unimpaired. The patient's general condition was fairly good.

1 Trans. Am. Laryngol. Soc., N. Y., 1898.
On examination, the fauces and pharynx presented a normal appearance. With the laryngoscopic mirror, however, the epiglottis was seen to be of a bright red colour, greatly swollen and rigid, and from its size and rigidity practically obliterating any further view of the larynx. So far as could be ascertained, the inflamed oedematous condition of the mucous membrane appeared to be localised to the anterior aspect of this structure, and the absence of dyspnœa and any impairment of the voice pointed to the fact that the inflammation had not attacked the upper aperture or the interior of the larynx. The swollen epiglottis was scarified with Heryng's knife, and the patient very soon began to feel relief from his symptoms. On the following day the redness and swelling had somewhat subsided, and the condition continued to improve from that date. When examined again with the mirror at the end of three weeks, the epiglottis was found to have regained its normal contour.

To this condition the term *angina epiglottidea anterior* has been applied. In all probability the infection takes place through the lingual tonsil at the base of the tongue, and the inflammation invades the area lying between the tongue and the epiglottis. Owing to the presence of a quantity of loose submucous cellular tissue in this region, a considerable serous exudation takes place, and oedema of the anterior aspect of the epiglottis is rapidly formed. In this case the first symptoms were only complained of on the previous day. In the concluding part of the paper these points will be considered in greater detail.

**Case 3.**—The third case also presents certain features of considerable interest, and may be studied in conjunction with that just narrated. J. F., a policeman, æt. 22, complained on the morning of January 31, 1900, of a sore throat, which caused him some pain on swallowing. He experienced no difficulty in breathing at this time. He remained out on duty all day in severely cold weather. In the evening he experienced for the first time slight difficulty in respiration, and on the following day he remained in bed. During the night of February 1, severe dyspnœa supervened, producing marked cyanosis, the members of his family describing him as becoming "black in the face." On the following morning he was seen by Mr. Alexis Thomson, to whom I am indebted for the description of his condition. He was found speechless, perspiring, and in great distress, his inspirations being of a noisy stridulous character. His pulse was full and bounding; the temperature was 100° F. There was nothing to be felt on external palpation of the neck. With the aid of a spatula a mass of a livid hue was visible at the base of the tongue. With the laryngoscopic mirror the epiglottis and ary-epiglottic folds were seen to be enormously swollen and partially occluding the upper aperture of the larynx. The true cords were only seen with great difficulty on attempted phonation.

The treatment adopted consisted in scarifying the epiglottis and ary-epiglottic folds with scissors, after spraying with cocaine. A large poultice was applied to the neck, and a steam spray and tent erected. There was free bleeding and expectoration, and considerable relief was
obtained in a few hours. Compound jalap powder was administered. On the following day the breathing was much easier, there was no crowing inspiration, and the patient was able to swallow and speak without much difficulty; the oedema had considerably subsided. Examination of the urine at this stage showed an acid reaction, specific gravity 1035, some albumin, and a trace of blood; also urates and granular and hyaline casts.

I had an opportunity of examining the patient on February 28. He was then in good health; the larynx showed no oedema, infiltration, or ulceration; but there was a slight redness of both vocal cords. The urine was normal. It is evident both from the history given in this case, and from the local appearances observed, that the inflammation commenced in the region of the fauces. The sore throat and pain on swallowing were the first symptoms complained of, and the objective examination disclosed an acute oedematous inflammation of the region immediately in front of the epiglottis. At a later stage, difficulty in breathing supervened, undoubtedly consequent upon the infiltration of the ary-epiglottic folds, which partially occluded the upper laryngeal aperture. In this case, therefore, in all probability, the inflammatory process had spread downwards so as to involve the larynx proper.

THE ANATOMICAL ARRANGEMENT OF THE SUBMUCOUS AREOLAR TISSUE OF THE LARYNX.—The development of inflammatory oedema of the larynx is, in the majority of cases, dependent upon three main factors,—First, upon the intensity of the inflammatory process producing it; secondly, the site of the infection; and, thirdly, the anatomical arrangement of the loose submucous cellular tissue of the larynx. It is to a study of the second and third conditions, and especially to the last named, that we now wish to draw the reader's attention. As oedema is essentially a serous exudation into the submucous areolar tissue, it naturally follows that the extent of the swelling thus produced, and the degree of development which may be attained by it, must depend both upon the limitations and upon the amount of such tissue in any given area. This loose cellular tissue varies somewhat in amount in different parts of the larynx, in certain areas being very considerably developed, in others being sparse and thin, while in other situations again, septa or partitions of a denser nature are met with, which appear to demarcate these areas the one from the other: in other localities again it is absent. We find that these anatomical facts have their clinical counterpart in the cases of oedema of the larynx that are from time to time met with, the exudation being sometimes small in amount or reaching very considerable proportions, in some cases being localised, in others being more or less diffuse.
In 1852, Sestier, in his classical work, demonstrated upon the cadaver the importance of studying the arrangement of the submucous areolar tissue in different parts of the larynx. One of the methods adopted by him for this purpose consisted in peeling off the mucosa, and defining the different areas in which this could be accomplished readily or with difficulty. In more recent years, Hajek of Vienna has investigated the subject from its anatomical standpoint, by carrying out a number of submucous injections and observing the distribution of the fluid which was thus introduced. The interest aroused by a study of Case 1, described and figured in the earlier part of this paper, has led me to carry out a series of injection experiments, the results of which I find bear out, in the main, the conclusions already arrived at by Hajek. I shall now endeavour briefly to describe the arrangement of this tissue demonstrated in this way. The accompanying plates illustrate the more important of the experiments.

The following method was adopted:—The material used for purposes of injection consisted of carmine gelatin, well heated to render it perfectly fluid. Abortive attempts were previously made by injecting water or a mixture of water and glycerin, but, owing to the ready way in which these substances flowed out from the puncture, after removal of the syringe, the results proved unsatisfactory, and therefore the carmine gelatin was substituted. Although this substance in the fluid state is of a thicker consistence than water, it was found to permeate the tissues very readily, and it had the great advantage of quickly setting when cooled. A syringe with a fine pointed needle was used, care being taken to have it thoroughly warmed before use. The injections were made on fresh larynges, and the gelatin was found to pass through the tissues more readily if the larynx was first heated in hot water. The preparations were then fixed in Jores' fluid for twenty-four hours, then immersed in methylated spirits for five or six hours, and finally placed in a mixture of glycerin and water. There was no tendency for the gelatin to shrink when fixed in Jores' formalin solution, a result which certainly takes place when the injected larynx is first hardened in spirit.

The epiglottis.—It is convenient to commence with the study of the arrangement of the loose areolar tissue upon the anterior surface of the epiglottis. If we examine the anterior or lingual surface of this structure in the normal state, we find that, as the mucous membrane is reflected from it on to the dorsum of the tongue, it presents three distinct folds, which from their position and connections are designated the median and lateral glossoepiglottic folds. On each side of the median fold there is a small depression, the glosso-epiglottic fossa or vallecula, situated between the median and external glosso-epiglottic fold on each side.

1 "Traité de l'angine laryngée oedémateuse."
2 Arch. f. klin. Chir., Berlin, Bd. xlii. Heft 1.
(Plate VI. Fig. 2). Passing outwards transversely from each lateral margin of the epiglottis is another fold of mucous membrane, usually described as the *pharyngo-epiglottic fold* (Plate VI. Fig. 2). This structure, as will be presently shown, is of special interest in connection with the subject now under discussion.

The mucous membrane covering the anterior surface of the epiglottis and the glosso-epiglottic fossae is very loosely attached to the underlying tissues. This is due to the presence of a considerable quantity of loose areolar tissue, a fact made readily evident by the ease with which the mucosa can be lifted up by a pair of forceps. A much better demonstration of this anatomical fact can be obtained by means of the injection of fluid, while the boundaries of the area can be more satisfactorily determined by the same method. If a syringe be filled with the warm fluid gelatin, and the needle carefully inserted beneath the mucous membrane at the anterior margin of one glosso-epiglottic fossa—at the point marked by the cross in Plate VII. Fig. 3—the following may be observed. The injection fluid travels without opposition from the base of the tongue into the fossa; as one proceeds to inject more of the material, it passes upwards on to the anterior surface of the epiglottis, readily lifting the mucous membrane before it. In the mesial plane, the median glosso-epiglottic fold forms a slight check to the further passage of the fluid. Beneath this fold the submucous connective-tissue layer is of rather a denser nature, consequently it offers a certain degree of resistance, and under the moderate pressure of the injection it can be made to bulge into the fossa of the opposite side. At the external margin of the fossa little or no resistance is offered at the site of the lateral glosso-epiglottic fold, and the injection passes readily in the direction of the pharyngo-epiglottic fold. If the experiment be repeated on the opposite side of the mesial plane, a similar result is got, and if only moderate pressure be employed, an appearance such as that presented in Plate VII. Fig. 4, is obtained, *i.e.*, two more or less spherical swellings occupy the position of the valleculae, separated from each other by a groove, which indicates the position of the median fold; while with this amount of injection the upper portion of the anterior surface and the free margin of the epiglottis remain visible above the swelling.

If, however, a larger quantity of fluid be injected and considerably more pressure be employed, the extent of the area of loose areolar tissue becomes more accurately defined, while the appearances above noted undergo considerable change. Thus the temporary opposition offered by the slightly denser layer of tissue in the region of the median glosso-epiglottic fold gives way, the two areas of injection in front of the epiglottis become fused, and there is a very considerable increase in the size of the swelling. Anteriorly it is found to reach to a limited extent on to the dorsum of the tongue for a distance of 3 to 4 mm. beyond the margin of
Fig. 1.—Larynx from case of sudden death, due to oedema of ary-epiglottic folds.

Fig. 2.—Base of tongue and upper aperture of normal larynx viewed from behind.
Fig. 3.—Showing moderate injection of both glosso-epiglottic fossae.

Fig. 4.—View of base of tongue and upper aperture of larynx showing moderate injection of glosso-epiglottic fossae and ary-epiglottic folds.
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PLATE VIII.

Fig. 5.—View of base of tongue and upper aperture of larynx showing forced injection of glosso-epiglottic fossa and ary-epiglottic folds.

Fig. 6.—Antero-posterior vertical section showing interior of right half of larynx.
FIG. 7.—Coronal vertical section of larynx viewed from behind showing occlusion of lumen from injection of ary-epiglottic folds.

FIG. 8.—Antero-posterior vertical section showing interior of left half of larynx.
the valleculæ. Externally the fluid passes towards the lateral wall of the pharynx. In the more important region of the epiglottis itself, the fluid, under increased pressure, spreads over the anterior surface of that structure until it reaches its free margin, where it produces a rounded swelling which conceals from view the normal contour of the epiglottis. Beyond the free margin, however, it will not pass, as along that line the mucous membrane becomes very closely applied to the underlying cartilage, and the loose cellular stratum disappears. It is therefore impossible to force the submucous injection over the epiglottis on to its laryngeal surface, while the cartilage of the epiglottis itself forms an imperious barrier to the further passage of the fluid. If we next examine the area lying between the margin of the epiglottis and the lateral pharyngeal wall, that is to say, the position occupied by the pharyngo-epiglottic fold of mucous membrane on each side, we find that a temporary check is given to the passage of the fluid in that situation. Beneath this fold, as was found to be the case at the median glosso-epiglottic fold, there is a somewhat denser layer of connective tissue, which extends between the deep surface of the mucous membrane and the free edge of the hyo-epiglottic ligament beneath. This, however, will, under somewhat more forcible pressure, give way, and the fluid can then find its way into the loose areolar layer beneath the mucous membrane of the pyriform sinuses and aryteno-epiglottidean folds. We find, therefore, that the swelling which results from forced injection of a considerable quantity of fluid into the loose submucous tissue between the base of the tongue and the epiglottis may be of very considerable extent, and form a rounded mass completely concealing from view the whole of the anterior surface of that structure. It is interesting to observe that, when an experimental injection of this nature is made, the epiglottis is neither pushed backwards towards the upper laryngeal aperture, nor is there any tendency to a backward incurvation of its lateral margins. On the contrary, the epiglottis tends to become broadened out, its lateral margins diverging from each other to a greater extent than is found in the normal larynx. Plate VIII. Fig. 5, shows the large injection mass in front of the epiglottis as viewed from behind; it is limited by the free margin of that structure; the position of the pharyngo-epiglottic fold is also clearly defined on each side. In this case no attempt was made to break down the opposition offered by the somewhat denser tissue beneath these folds. This, however, could have been accomplished with a little additional pressure.

The aryteno-epiglottidean folds.—These two folds which form the lateral margins of the upper aperture of the larynx (Plate VI. Fig. 1) have long been recognised as specially liable to the development of oedema. This is due to the fact that a considerable quantity of loose areolar tissue is present between the two layers of mucous membrane which constitute each fold. The injection experiments
demonstrate very successfully the arrangement of the mucous layers and the subjacent tissue. When the needle is inserted at the posterior-inferior extremity of the fold immediately in front of the cartilage of Wrisberg, and the fluid is directed upwards towards the margin of the epiglottis, the following changes are observed:—The two layers of mucous membrane which together form the fold are very readily separated from each other as the fluid is driven forwards. The inner layer, which is continuous with the mucous membrane covering the interior of the larynx, is raised from the lower and lateral part of the laryngeal surface of the epiglottis, and a swelling is formed in that situation. The outer layer, continued as the lining membrane of the pyriform sinus, is similarly affected, and the sinus readily becomes infiltrated with the injection material. The fluid also passes, without opposition, forwards and upwards to the lateral margin of the epiglottis, while part of it finds its way downwards over the posterior surface of the arytenoid cartilage, so loosely is the mucous membrane attached in this situation. If only moderate pressure be employed, the appearances represented in Fig. 4, Plate VII., are obtained. On the right side the swelling has assumed the pear-shaped form which is so frequently seen clinically in cases of chronic edema of the ary-epiglottic fold. With a moderate injection of this kind the lumen of the larynx remains comparatively free, and normal respiration could doubtless be carried on without any discomfort.

If, however, a considerably greater quantity of fluid be injected into the ary-epiglottic folds, the swelling can be made to assume such large proportions as to completely block the upper aperture of the larynx and to occupy the whole of each pyriform sinus (Fig. 5, Plate VIII.). Fig. 6, Plate VIII., which represents the interior of the right half of a larynx, divided vertically in the mesial plane, shows the extent to which the injection will travel over the inner surface of the larynx. Anteriorly, but only to a somewhat limited extent, the mucous membrane covering the laryngeal surface of the epiglottis is stripped from the underlying cartilage by the invading fluid. Inferiorly, the upper margin of the false cord or ventricular band limits the further spread of the injection in that direction, although under greater pressure this can be broken down and the submucous tissue of the false cord can also become injected. Posteriorly and inferiorly, the mucous membrane covering the laryngeal aspect of the cartilage of Wrisberg and the arytenoid cartilage is firmly united to these underlying structures; consequently this surface remains free from the injection fluid introduced into the ary-epiglottic fold, as shown in Plate VIII. Fig. 6. Owing to this anatomical barrier, one fold cannot be injected from the other in this situation, and thus when both folds are injected a bilateral tumefaction is produced, the two swellings remaining distinct from each other. On the posterior or esophageal aspect of the larynx, the fluid will readily pass from one side to the other, owing to the
loose attachment of the mucous membrane in this situation. The fluid, however, cannot spread over the upper free margin of the inter-arytenoid fold and descend upon the posterior laryngeal wall. It is not out of place to mention here that the presence of a limited amount of loose areolar tissue beneath the mucous membrane of the posterior wall of the larynx permits of the injection of a small quantity of fluid in this situation.

If we again turn our attention to Fig. 5, Plate VIII., and examine the anterior and upper extremities of the ary-epiglottic folds injected under considerable pressure, we find that the fluid has reached the pharyngo-epiglottic folds. The somewhat denser layer of submucous tissue beneath them forms a temporary check to the passage of the fluid into the glosso-epiglottic fossae.

The illustration further demonstrates the fact that the injection fluid has failed to pass from these fossae backwards into the ary-epiglottic folds and pyriform sinuses. If further injection be made, in either of these two situations, the barrier breaks down and the fluid passes uninterruptedly from one area to the other.

When forcible injection of the two ary-epiglottic folds is made, their inner layers approximate so closely to each other in the mesial plane, that they conceal from view the false and true cords. The lumen of the larynx is also occluded to such an extent, that, were the same degree of swelling attained in the living subject, asphyxia would be produced unless relieved by surgical interference. In other words, there is here reproduced artificially the same condition as was found in Case 1 (Plate VI. Fig. 1) which proved so rapidly fatal. Fig. 7, Plate IX., is a coronal vertical section through the larynx, showing the injection-mass occupying each ary-epiglottic fold and filling up the two pyriform sinuses. The two inner layers of these folds are so closely approximated to each other, that the larynx is almost completely obstructed.

It was clearly shown in several of the experiments, that the greater part of the laryngeal surface of the epiglottis could not be injected from the ary-epiglottic folds. In Case 1 this surface remained free from oedema. If the needle of the syringe be inserted beneath the mucous membrane in this situation, it will be found impossible to make the fluid pass underneath it, so firmly is it bound down to the subjacent cartilage. In the earlier experiments upon the glosso-epiglottic fossae, attention was drawn to the fact that the fluid could not be forced over the free margin of the epiglottis on to its posterior aspect. Consequently we find that the laryngeal surface cannot be injected, a fact which the majority of the illustrations clearly show.

It will also be observed that injection of the ary-epiglottic folds produces a marked backward incurvation of the lateral margins of the epiglottis (Fig. 7, Plate IX.); this simulates the infantile type of larynx.1 As the introduction of fluid between the

1 Brit. Med. Journ., London, Dec. 1, 1900.
two layers of the ary-epiglottic folds tends to bring about this infantile form, it follows that in the larynx of the young child and in those cases occasionally met with where the infantile type persists in adult life, a lesser amount of oedema will tend to cause respiratory difficulty. It is probable, too, that the dyspnoea which may supervene in a case of laryngeal diphtheria in a child may be due to a slight amount of oedema of the ary-epiglottic folds before any false membrane has formed in the larynx.

While post-mortem experiments of this nature cannot be regarded as furnishing exact representations of what may occur in the living body, they nevertheless accurately demonstrate both the amount and the extent of the loose cellular tissue present beneath the mucous membrane. In other words, they show to what extent the tissues in any situation are capable of being invaded by a serous or sero-purulent exudation. Fig. 3, Plate VII., does not portray a fanciful picture; oedema limited to the anterior aspect of the epiglottis is met with clinically, and in Case 2, described in the earlier part of the paper, we have a clinical prototype of the appearances thus artificially produced. That it is possible for the submucous exudation to remain limited to one glosso-epiglottic fossa, is shown by the study of two cases of an acute phlegmonous inflammation described by Caz. In both of his cases there was hyperaemia and swelling of the right vallecula, the epiglottis being pushed somewhat to the left side, while the left glosso-epiglottic fossa remained empty, and thus formed a distinct contrast to the fossa on the opposite side. The question of the spread of the oedema from the region in front of the epiglottis backwards and downwards to the aryteno-epiglottidean folds and pyriform sinuses, is one of considerably greater clinical importance, because of the risk of obstruction to the larynx that may at once arise when these folds become oedematous. So long as the swelling is confined to the anterior aspect of the epiglottis, the only clinical symptoms that may be met with are sore throat, pain on swallowing, and perhaps the sensation of the presence of a foreign body. When, however, the oedema involves the aryepiglottic folds, the additional grave symptom of dyspnoea supervenes. These points are also illustrated in the three cases which form the text of this paper. In Case 2 the inflammation and oedema were confined to the region in front of the epiglottis, and the patient only suffered from sore throat and painful deglutition. In Case 1, again, the edema was limited to the upper laryngeal aperture, and dyspnoea alone was complained of. In Case 3, on the other hand, both these situations were involved in the inflammatory process, and a combination of symptoms was accordingly met with. The sequence of events in this last case clearly suggested that the inflammation had commenced in the area in front

1 Arch. f. Laryngol., Berlin, 1898, Bd. viii. Heft 2.
of the epiglottis, and thence had spread downwards to the aryepiglottic folds.

A study of these cases, and the knowledge derived from the injection experiments, leads us to a consideration of the two following points. To what extent does the layer of denser tissue beneath the pharyngo-epiglottic fold of mucous membrane play a part in preventing the spread of ðæðema in the living body? Secondly, was the further spread of the ðæðema downwards into the larynx in Case 2 prevented by this barrier, or only by early treatment, and would the same result have been achieved in Case 3 had the same early treatment been possible? These two questions cannot be separately discussed. Recognising, as we must, from injection experiments repeatedly performed, that a difference exists between the density of the submucous areolar tissue in the glosso-epiglottic fossa and ary-epiglottic folds on the one hand, and that lying beneath the pharyngo-epiglottic folds on the other, there is some justification for assuming that a serous exudation may be at any rate temporarily checked by it. If that be so, the delay thus given may admit of timely treatment preventing the further spread of the ðæðema towards the larynx. That this structure cannot act in the same definite way in the living body as it does in the dead, must be evident, because in the former the inflammation can naturally spread by continuity of mucous membrane. ðæðema may consequently be thus produced in the area beyond the barrier. At the same time, I think we are justified in assuming that even in the living tissues a temporary check may thus be offered to the onward spread of the ðæðema, and so may retard the onset of the graver symptom of dyspnoæa and permit of treatment proving more effectual. It is very improbable that ðæðema will find its way upwards and forwards from the ary-epiglottic folds to the glosso-epiglottic region, at any rate before tracheotomy is rendered necessary. In Case 1 these folds had become infiltrated to such an extent as to completely occlude the upper laryngeal aperture before any ðæðema had reached the anterior aspect of the epiglottis.

The false cord or ventricular band.—Under the mucous membrane of the false cord, the areolar tissue is somewhat sparsely developed. If the syringe be inserted close to the anterior end of the cord, at the spot indicated by the cross X, in Fig. 8, Plate IX., the fluid finds its way fairly readily beneath the mucosa, though the injection is made with less facility than in the ary-epiglottic region. Under moderate pressure the fluid is confined to the area of the false cord, but if this be increased the intervening barrier is broken down, and the fluid passes beneath the inner layer of the ary-epiglottic fold, and may be gradually forced backwards and upwards, diffusing itself over a considerable area of this fold, if the injection be continued. The swelling produced by injection of the false cord is somewhat limited in size, but if the experiment
be carried out on both sides of the larynx, its lumen thus becomes very considerably narrowed. As the ventricular band forms the upper and inner boundary of the ventricle of Morgagni, both the entrance to the ventricle and the dimensions of the space are considerably restricted by the presence of the injection fluid in it. In Plate XI, Fig. 8, the injection has not passed quite so far as the inferior border of the false cord.

The true vocal cord and subglottic area.—Oedema of the true cord depends mainly upon a serous infiltration of the areolar tissue which assists in forming that structure. This has been shown by Hajek in his experiments. If the needle of the syringe be introduced beneath the mucous membrane covering the upper surface of the cord, at a point immediately in front of the vocal process, the injection passes forwards without opposition. It finds its way also outwards into the inferior and outer wall of the ventricle, and in doing so tends to considerably occlude the entrance to that space. If only moderate pressure be employed, the fluid is confined to this aspect of the cord, and does not pass below its free margin. If, however, further injection be made in the same situation, the under surface of the cord and the subglottic area also become injected, a rounded swelling being formed, such as is represented in Fig. 8, Plate IX. If the injection be made beneath the inner free margin of the cord, similar changes are observed, that is to say, the fluid is at first confined to the subchordal area, but under increased pressure will extend to the upper surface of the vocal cord. If a transverse section be made of a cord thus injected, it will be seen that the fluid has infiltrated the whole thickness of that structure. It will further be observed in Fig. 8, Plate IX., that the subglottic edema is confined to a somewhat small area, the lower limit of which is not defined by a horizontal line, but presents a convex margin with the most dependent part of the curve in the centre.

If both vocal cords become oedematous, the glottic chink is readily closed by the bilateral swellings coming into contact, and great difficulty in breathing is produced. It is to this condition alone that the term “edema glottidis” is really applicable. Though this term is frequently used, it is in the majority of cases incorrectly applied. Oedema of the true vocal cords is comparatively uncommon, the aryteno-epiglottidean folds on the other hand being the most usual site of this condition, and the application of the term oedema glottidis to a serous exudation in the latter situation is anatomically incorrect. Risch reports the case of a man who died from asphyxia, and in whom he found, on post-mortem examination, that both true vocal cords were oedematous, and were flattened against one another, completely closing the glottic chink. Morell Mackenzie only twice met with edema of the true cords, and looked upon the condition as a rare one.

1 Berl. klin. Wchnschr., 1866, No. 33.
Subglottic oedema may be entirely confined to the area below the cords, and it certainly occurs more frequently than the previous variety. It has been described by Sestier, Cruveilhier, and others. Gibb has reported a case of dyspnœa in which the oedema was confined to the subglottic region on each side of the larynx. He emphasises the importance of performing tracheotomy instead of laryngotomy in cases demanding surgical interference, as by the former operation one gets more effectually beneath the site of the obstruction. Extension of the oedema to the submucous tissue of the trachea is apparently not common. Amongst 132 cases of oedema of the upper air passages, Sestier found oedema of the trachea only seven times. One can only with some difficulty raise the mucous membrane of the trachea in the blades of the forceps, and it is by no means easy to force the injection fluid beneath it.

In conclusion, we should point out that oedema originating in the region of the tonsils will pass downwards to the glosso-epiglottic fosse, and thence will find its way to the ary-epiglottic folds. Similarly, oedema of the lateral wall of the pharynx will spread to the pyriform sinus, and from there reach the ary-epiglottic fold. In this way dyspnœa may supervene as a grave complication of faucial and pharyngeal inflammation. It is interesting to observe, in connection with the injection experiments made in these two situations, that beneath the mucous membrane of the posterior pillar of the fauces there exists a barrier of dense tissue, which interferes with the fluid spreading from the tonsillar region to the lateral pharyngeal wall lying posterior to it.

ON THE SYMPTOMS AND DIAGNOSIS OF CARCINOMA OF THE HEPATIC FLEXURE OF THE COLON.

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Increasing experience in the diseases which affect certain anatomical regions of the body, tends to teach that many more differences exist in the symptoms which may arise than was at one time supposed. These differences doubtlessly owe their origin to the particular locality of the region involved; and so diverse are they, in certain instances, that it is a mistake to attempt to include them in any combination, however much general anatomical considerations might indicate or suggest that they should be so dealt with. Thus, while it is frequently the custom to speak of carcinoma of the colon as a disease per se, there is little doubt that much difficulty and confusion would be removed, and greater accuracy in diagnosis attained, if the colon were looked upon, not so much as an anatomical and physiological entity, but as a more or less complex part of the organism, whereby disease at one part