

POSTDIPHThERITIC STENOSIS OF THE LARYNX
(RETAINED INTUBATION INSTRUMENTS
AND RETAINED TRACHEAL
CANULÆ).¹

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INTUBATION and tracheotomy are the two operations for the relief of the acute stenosis of laryngeal diphtheria. In this country, tracheotomy, though it still has some advocates, has been practically abandoned in favor of the less dangerous operation of intubation. But whether intubation or tracheotomy is employed, there are a certain number of cases in which, after recovery from the original disease, the stenosis will persist indefinitely. The number is probably less with intubation than with tracheotomy, but the mortality after the latter operation is so high that a fair comparison is difficult. My experience has been limited to cases which were originally intubated, and therefore this paper will consider more the subject of what the late Dr. O'Dwyer called retained intubation tubes than retained tracheal canulæ. But several of the cases described below, after first having been intubated, were subsequently tracheotomized, and so placed in practically the same position as those cases which have been tracheotomized at the outset. As both conditions (that of retained intubation tube and retained tracheal canula) can be easily cured by the same method of treatment, and as I think I can show that the anatomical cause of the stenosis is likewise the same for both, there seems nothing to be gained by considering them as separate and distinct forms of stenosis. Indeed, there is a loss. Dr. O'Dwyer

¹ Read before the New York Surgical Society, February 14, 1900.

has proved the inestimable value of his invention for the cure of retained tracheal canulæ. But he considered retained intubation tubes another matter, and incidentally one which was more or less the fault of the operator. Hence, when the condition has not been soon overcome, as he advised, by repeated careful intubation, avoiding all traumatism, tracheotomy, or thyrotomy, and excision of the obstructing tissues have seemed the only two resources. Neither will succeed. If, however, both conditions are regarded as the same and treated virtually on the same principle, success can be guaranteed.

The chief difficulty in the management of cases of retained tube has been the ignorance or uncertainty as to the usual anatomical cause of the persistent stenosis. The literature on this subject is scanty and unsatisfactory. O'Dwyer gave the results of his experience in a paper read before the American Pediatric Society in 1897. Without describing individual cases, he stated that the most common obstruction is an œdematous, chronically inflamed condition of the subglottic region. When the swollen tissues are relieved from the pressure of the tube they immediately come in contact and block the lumen of the larynx. He mentions as a less common cause of the stenosis cicatricial tissue. The dyspnoea then does not occur immediately after the removal of the tube, but only after the lapse of some hours or days. The clinical histories of cases of retained tube are very suggestive of these two conditions. But without actual demonstration of their existence, and in view of other possibilities, notably, exuberant granulations, the average surgeon to whom these patients may be referred, after a prolonged course of repeated, careful intubation has failed to effect a cure, will generally attempt an exploratory thyrotomy and "give it up." As exceptional causes of the stenosis, O'Dwyer cites two cases in which granulations sprang from erosions produced at the base of the epiglottis by the head of the tube. These effectively blocked the lumen of the larynx when the tube was withdrawn, and also threatened to overtop and close the upper opening of the tube while it was in place. This difficulty was met by changing the shape of the

head of the tube, which relieved the ulcer of irritation and allowed it to heal. The hard-rubber instruments now in use, by the way, probably could not inflict such a traumatism.

He described one other case in which breathing without the tubes was impossible; and after death, which followed an unexpected autoextubation, it was found that there had been a slough of the cricoid cartilage. The loss of this cartilage he considered irremediable except by tracheotomy. Dr. O'Dwyer mentioned the possibility of a postdiphtheritic paralysis of the laryngeal abductor muscles, but called attention to its improbability, as such paralyzes usually occur some time after the onset of the disease, whereas the stenosis exists from the beginning.

Galatti (*Jahrbuch für Kinderheilkunde*, xlii, 1896, p. 340) reports two cases, in one of which (not tracheotomized) there was found at autopsy a cicatricial thickening of the mucous membrane and perichondrium on the inner surface of the cricoid cartilages. In the second, respiration at the end of ten days was impossible without the tube, and therefore tracheotomy was performed. As the condition did not improve, a month later the larynx was opened by a thyrotomy. Just above the tracheotomy wound a cicatricial stricture was found and dissected out. The remainder of the larynx showed only a general swelling and redness. The raw surface left after excision of the cicatrix was skin grafted, and subsequently unsuccessful attempts were made to keep the larynx open by a tracheotomy canula fitted with an attachment which projected upward through the larynx.

Baer narrates a case in which there was repeated autoextubation for twenty days, and finally tracheotomy. The laryngeal stenosis persisted, and, as intubation was then impossible, thyrotomy was performed. This revealed some granulations near the tracheal fistula, and above, a fold of mucous membrane which apparently caught the tip of the intubation tube, and so prevented its insertion. There was no ulceration in the larynx. The granulations and the fold of mucous membrane seem to have had nothing to do with the stenosis, because, after their excision, intubation was immediately accomplished and

the tracheal canula removed; yet even then respiration without the aid of the tube was impossible. On this account, some weeks later a second tracheotomy was performed and death followed. The autopsy revealed a firm swelling just below the vocal cords, occupying the anterior and lateral parts of the larynx and markedly narrowing its lumen. This description corresponds so closely with the cause of the stenosis in other cases, namely, hypertrophic subglottic laryngitis, that there seems to be no reasonable doubt of its identity. The fold of mucous membrane interfering with the insertion of the tube, and the granulations near the tracheal fistula, for the reasons given above, could not have been the real cause of the stenosis.

Boulay, at the Twelfth International Medical Congress in Moscow, reported two cases of prolonged intubation. In the first, no trace of ulceration and no granulations could be found after the stenosis had persisted some three weeks without improvement, in spite of repeated careful intubation. Above the vocal cords the parts appeared normal, but below them there was plainly visible a reddened, rounded swelling which occupied the lateral parts of the larynx and nearly obliterated its lumen. A diagnosis of intense subglottic laryngitis was made. This corresponds very closely with the condition I have found in other cases. The subsequent history is interesting, as it shows the futility of the ordinary methods of treatment. Tracheotomy was performed, as there was constant danger of asphyxia should the tube have been unexpectedly coughed out.

Laryngoscopic examination after the tracheal canula had been worn for about a year showed that the subglottic tissues approached towards the median line from each side like a funnel, leaving between them only a narrow antero-posterior chink. This small opening was dilated by a laminaria tent, and though this was followed by repeated intermittent dilatation for months, respiration at the time of the report (several months thereafter) was only possible through the tracheal canula.

In Boulay's second case the tube had to be retained for about two weeks, and then, on account of the frequent auto-

extubation, tracheotomy was performed. A year later, as the stenosis was very marked, the interior of the larynx was exposed by a median division of the cricoid and thyroid cartilages, and was found to be filled with a dense mass of cicatricial tissue. This was dissected out, and an unsuccessful attempt made, as in Galatti's case, to keep the larynx open by the use of a tracheal canula fitted with an arm projecting upward.

Dr. William Northrup, of New York, has informed me in a personal communication that he has in his possession a larynx illustrating a condition somewhat similar to that found in Boulay's second case. The necessity for intubation continued for some weeks, but the frequent autoextubation finally necessitated a tracheotomy. Death followed about a month later from pneumonia, and at the autopsy the larynx was found completely occluded at its centre by a cicatricial diaphragm. There had evidently been a narrow ulcer extending circularly all the way around the interior of the larynx, and Dr. Northrup believed that it was produced by the intubation.

Dr. Somerset, formerly the resident physician at the Willard Parker Hospital, has kindly supplied me with the history of the following case, which illustrates not only the condition of the larynx, but also the prognosis of surgical operation for the relief of the stenosis.

CASE I.—M. M., three and one-half years of age, was admitted to the hospital with diphtheria on June 2, 1895. Intubation had been required six days previously. The stenosis persisted, and, as autoextubation followed and increased in frequency to such an extent as to threaten death from asphyxia, a tracheotomy was finally performed on July 13. Subsequently, intubation was again accomplished, but with the tracheal canula left in place in case of accidents, as the tube was still often coughed out. Frequent careful intubation with tubes so made as to inflict the least possible traumatism, as advised by Dr. O'Dwyer, was of no avail. Astringents, like alum, applied on the surface of the tube to the laryngeal mucous membrane, and afterwards curetting the larynx, were tried without success. The stenosis

did not improve, and the child could breathe only through the tube or the tracheal canula. In this condition she was transferred in October to Dr. Clinton Wagner, at the Metropolitan Throat Hospital. He opened the larynx by a median division of the cricoid and thyroid cartilages, and found the following condition (*New York Medical Record*, January 4, 1896): "The entire lining membrane of the larynx from the epiglottis to the cricoid cartilage appeared to be thickened and covered with a mass of what seemed to be granulations. After removing a portion with scissors and forceps, an attempt was made to pass an ordinary laryngeal sound upward and forward into the mouth, but without success. It encountered an obstinate resistance. Upon carrying the index-finger into the mouth and below the epiglottis, I could distinctly feel the point of my sound below this dense mass of adventitious tissue. Finally, I succeeded, with the aid of the knife and my finger, in forcing an opening sufficiently large to permit the sound to pass through into the mouth." The larynx was subsequently kept open and packed for several days, and then drawn together; but the tracheal canula was left in the lower angle of the wound. The result was not entirely satisfactory, and about two years later, Dr. G. B. Hope, in the same institution, again opened the larynx in the line of the old scar. In a personal communication he informed me that the soft parts were much thickened, and at the superior aperture there was a cicatricial band, which, together with thickened tissue below it, had rendered the larynx almost impervious. He divided and excised the obstructing parts, and subsequently employed intermittent dilatation to keep the larynx open. At present, however, the tracheal canula is still worn most of the time.

The stenosis in this case was evidently dependent at first either upon simple or inflammatory hypertrophy, or else upon cicatricial thickening of the soft parts. The granulations mentioned could have had little or nothing to do with any obstruction described as a "dense mass of adventitious tissue." It is conceivable that granulations may have resulted from the curetting to which the larynx was subjected before the first thyrotomy, but it is easy to mistake for them the vascular, thickened laryngeal mucous membrane which bleeds freely at

the slightest touch. In talking with other surgeons to whom such cases have been referred, I have found that granulations *per se* are considered the usual cause of the obstruction to respiration in any case of retained tube or tracheal canula, and operation for their excision seems generally to be advised. This is the only case, however, among those I report, in which I can find any ground for even a suspicion of their presence as an obstruction within the larynx. This fact should therefore be emphasized to prevent useless operations.

I am indebted to Dr. F. Tilden Brown for the description of the operation upon, and the conditions found in, the following case:

CASE II.—H. P., three and one-half years of age, was intubated for diphtheria at the Willard Parker Hospital on June 28, 1896. The child recovered from the diphtheria, and nothing noteworthy followed except that the laryngeal stenosis persisted, and the tube, after the elapse of the usual length of time, could not be removed except for a few moments for cleansing. About a month after admission autoextubation began, and Dr. Somerset, who was then the resident physician, informs me that frequently, after coughing out the tube, the child would run to him with the instruments, and, though deeply cyanosed and in extreme dyspnoea, would aid in the operation all he could by standing quietly and in the proper position while the tube was reinserted. Hence, the closure of the larynx could not have been complete and instantaneous; and therefore the lesion was probably vascular, thickened tissue, which became more or less rapidly engorged, and so blocked the lumen of the larynx when relieved of the pressure of the tube. A cicatrix would not contract enough to produce obstruction for several days, and granulations would close the larynx immediately.

No improvement occurred with intubation, and therefore he was transferred to the Presbyterian Hospital, where, on July 31, 1897, Dr. Brown opened the larynx by median division of the cricoid and thyroid cartilages. Careful inspection showed an even thickening of the soft parts throughout the whole interior of the space inclosed by these cartilages. There was no sodden or watery appearance, such as might be produced by œdema. Fur-

thermore, the thickened tissue seemed firm and vascular. Bleeding was easily excited by sponging, yet there was nothing like ulceration nor any sign of cicatrization. The soft parts within the larynx were apparently covered everywhere by epithelium. The even thickening of all the tissues made it difficult to distinguish the vocal cords and ventricles from the subglottic area. With a view to decreasing the amount of soft tissue, which appeared the only obstacle to free respiration, and at the same time to replace the vascular tissue by non-vascular, cicatricial tissue, a vertical strip was cut from the whole thickness of the soft parts on each side of the inner surface of the larynx. The hemorrhage was checked by packing, which was removed after the bleeding had stopped, and the larynx then closed. A few hours later dyspnoea occurred, and a tracheotomy canula was inserted through the lower angle of the wound. Healing was uneventful, but the laryngeal stenosis was never relieved sufficiently to allow of breathing without the canula, and the patient finally left the hospital in this condition, to die a few months later of pneumonia.

The report of the pathologist upon the excised strips of tissue stated that they showed "infiltration with many small round cells. In places apparently granulation tissue. Over some thickened areas there is thin mucous membrane."

It will be noted that at the time of operation the stenosis appeared to be caused solely by simple thickening or hypertrophy of the soft parts. There was no cicatricial tissue, and nothing in the pathologist's report indicates its presence. As no ulceration was seen, granulations could not have been the obstruction; and the "granulation tissue" described by the pathologist must have been a part much infiltrated with the round cells of the chronic inflammatory process which gave rise to the general thickening of the tissues. The excision of this tissue was of no benefit.

I am indebted to Dr. Dillon Brown for the history of the following case:

CASE III.—J. G., fourteen months of age, was intubated by Dr. Brown during the first week of February, 1897. The child had had a pharyngeal diphtheria for several days, and in-

tubation was required thirty-six hours after the first symptoms of involvement of the larynx. The diphtheria gradually subsided, but the laryngeal stenosis persisted, and necessitated retention of the tube. The tube began to be coughed out some three weeks after the first intubation; but respiration, though possible for an hour or two without the tube, could never be carried on for any length of time through the natural channels. Finally, on the twenty-eighth day, the tube was coughed out, and, before it could be reinserted, the child was asphyxiated. At the autopsy, the intralaryngeal soft parts were found to be evenly thickened to such an extent that the lateral portions of the larynx were in contact in the median line. The tissues were moderately soft, and compressible enough to permit the introduction, without undue force, of the tube ordinarily employed for a child of this age and size. There were no ulcerations and no cicatrices. The specimen was not examined under the microscope until December, 1899, when Dr. W. N. Berkeley, the pathologist of Dr. Brown's laboratory, very kindly cut some sections transversely through the centre of the larynx and furnished me with the following report:

“The mucous membrane, submucosa, and remaining soft parts within the larynx appear much swollen and also thickened by a growth of new cells. The vessels are numerous and congested. There is an irregular increase in the superficial epithelium and a hypertrophy of the subepithelial papillæ. There is no evidence of ulceration with loss of substance. The striated muscle fibres, both in front and behind the arytenoid cartilages, are present in the usual amount, but, like all the other soft tissues, are infiltrated with columns of small round cells. In all probability this infiltration would interfere with or abolish the function of the muscles. The cartilage is normal.”

The stenosis in this case was clearly dependent upon a thickening or hypertrophy of the intralaryngeal tissues, accompanied and probably caused by the continuation of the original exudative inflammation which seems to have become more or less chronic. The results of the microscopical examination of the tissues taken from this patient and from H. P. are practically the same. Involvement of the muscles in the in-

flammatory process, and their consequent loss of function, might permit the tube to be easily coughed out. At any rate, when the tube is retained for any length of time, frequent autoextubation seems sooner or later to occur. That it is a dangerous, and may be a fatal, complication is evident.

CASE IV.—C. S., five years of age, required intubation in the course of a diphtheria at the Willard Parker Hospital on May 20, 1899. She gradually recovered from the diphtheria, but not from the laryngeal stenosis. The tube was first removed at the expiration of five days, but had to be immediately reinserted. Thereafter it was taken out and a clean one substituted about once a week until June 15, when breathing through the natural passages, though somewhat labored, was found to be possible. But the dyspnoea increased, and on June 23 intubation was again required. Careful removal followed by the immediate reinsertion of a clean tube every week or two followed, but without success. Dr. O'Dwyer's "special" tubes for such cases were faithfully tried with a similar result.

In November, I heard of the difficulty, and suggested the long-continued, *uninterrupted* use of as large a tube as could be introduced into the larynx. I was informed that this plan was being carried out; but the mother became impatient, and on January 25, 1900, brought the child to Gouverneur Hospital. The following day, under chloroform anæsthesia, I removed from the larynx a metal No. 4-5 tube; in other words, a tube not larger than that ordinarily employed for a child four or five years old. I found that the larynx was almost completely blocked, and immediately reinserted a No. 11-13 tube, the largest I had. This slipped in place without requiring any force, or at least not more than would be equivalent to a pressure of about three pounds on the handle of the inserter. This case is still under treatment. The tube was removed March 1, and respiration without it was possible for several hours. There was thus marked improvement. But it seemed wise to replace the tube for another six weeks, which have not yet expired. There is every prospect, however, of a permanent cure.

CASE V.—I. B., four years of age, was intubated in the Willard Parker Hospital for diphtheria in December, 1896. She recovered from the diphtheria, but the stenosis persisted. The

tube was changed about once a week for cleansing, but had to be replaced almost immediately. In February, autoextubation began, and gradually increased in frequency until it occurred several times a day. Dr. O'Dwyer's "special" tubes were tried, and also large-sized tubes, even a No. 8-10. But nothing could be kept in the larynx for more than a few hours at a time; and therefore in March, as there was constant danger of asphyxia, tracheotomy was performed. A month later intubation was again accomplished, but the tracheal canula was left in place in case the tube should be coughed out again. As it was not, the canula was finally removed and the tracheal fistula allowed to heal. The tube was changed about once every ten days or two weeks, but the stenosis persisted; and decided expedition in the insertion of a fresh tube was necessary to prevent asphyxia. Respiration without the tube was entirely impossible. In addition to the regulation 4-5 tube ordinarily used for a child of this size, Dr. O'Dwyer's "special" tubes were again employed. Finally, as no improvement occurred, she was transferred to Gouverneur Hospital on October 15, 1897. She was then carrying a rather small tube in her larynx, apparently with the idea that the stenosis was due to traumätism inflicted by the tube, and that if the pressure were made as slight as possible the stenosis would subside. But about a week after admission the tube was unexpectedly coughed out, and death from asphyxia only prevented by an impromptu tracheotomy, performed, I believe, with a penknife, by the house surgeon, Dr. Pafford. Subsequently, intubation was again accomplished, at first with the tracheal canula in place; later, as autoextubation was not repeated, the canula was removed and the fistula allowed to close.

The condition seemed to require operation, and on January 10, 1898, the stenosis having persisted unrelieved for a year, I opened the larynx by a median vertical incision through the cricoid and thyroid cartilages. The mucous membrane presented a thickened velvety appearance, rendering the vocal cords and ventricles indistinguishable from the other parts. The tissues were evidently very vascular, and after sponging bled freely. There were no cicatrices and no ulceration. The epithelium was everywhere intact. On bringing the parts together, it was found that the thickened mucous membrane on the inner surface of each side of the larynx came in contact, and effectually blocked respi-

ration. This vascular thickening or hypertrophy of the soft parts in the interior of the larynx was the sole cause of the stenosis. I therefore passed a silk suture through the cartilage and thickened mucous membrane on each side and tied it on the outer surface of the corresponding thyroid ala. This retracted the tissues enough to allow easy breathing when the larynx was closed. But infection followed, and in the course of the next few weeks the greater part of the thyroid and cricoid cartilages sloughed out. The case was discharged April 20, 1898, apparently condemned to the permanent use of a tracheal canula.

She returned in August with a dense cicatrix and some dermatitis around the fistula. I then found that a probe could be passed with some slight difficulty through the larynx from below up into the mouth. Under chloroform anæsthesia this aperture was rapidly dilated with urethral sounds until a No. 24 F. could be passed. This was followed by the insertion of a 4-5 intubation tube. The tracheal canula was then removed and the fistula closed spontaneously within a short time. The tube was not disturbed until the first of October, when, as there was a noticeable odor about the breath, it was taken out and a No. 6-7 tube substituted. At the time of the change of tube, it was found that the stenosis was still very marked. The 6-7 tube fitted rather tightly in the larynx, and was large for the size and development of the child, who appeared about three years old.

This tube was left undisturbed for about five weeks. After its removal at the end of this time (November, 1898), natural breathing was obtained, except that at first there were several attacks of coughing followed by severe inspiratory dyspnoea. It was somewhat alarming, but gradually disappeared under the administration of a little morphine. Apparently, the abductor muscles had become more or less atrophied in their long period of disuse, while the intubation tube or tracheal canula had been worn, and did not open the larynx quickly at the end of the act of coughing. There is at present (February, 1900) no dyspnoea whatsoever. The voice, however, is harsh and unnatural for a child. So far as can be judged by palpation of the neck through the rather dense cicatrix which is present, the thyroid and cricoid cartilages appear to have been at least partially regenerated.

Thus this case was twice tracheotomized for autoextubation, and subsequently reintubated without improvement of the

stenosis, between December, 1896, and January, 1898. In the latter month the larynx was opened, and the stenosis proved to be dependent upon thickening of the intralaryngeal soft parts. The attempt to bind this hypertrophied tissue to the thyroid alæ by a vertical cicatrix resulted in slough of at least part of the cartilage, and the development of a stenosis which was apparently worse than before. The use of small intubation tubes, frequently and carefully changed with the object of inflicting the least possible traumatism upon the larynx, was of no avail; while the insertion of a tube as large as could be borne without lacerating the parts, and its retention uninterruptedly for a long period, effected a comparatively rapid and a permanent cure.

CASE VI.—M. W., aged four years, was intubated for diphtheria at the Willard Parker Hospital in October, 1896. Like the other cases, he recovered from the diphtheria, but not from the laryngeal stenosis. In the first part of November he began to cough out the tube more and more frequently. This difficulty was successfully overcome by increasing the size of the tube up to the largest which could be introduced without much force. But they were never left in place for more than a week or two at a time. After the large tubes had been worn with frequent changes for a month, smaller ones were by degrees substituted, until it was found that a No. 2 tube could be kept in the larynx without difficulty. In May, 1897, as there was no improvement in the stenosis, he was transferred to the Manhattan Eye and Ear Infirmary for laryngeal treatment. But soon afterwards, having contracted scarlet fever, he was sent back to the Willard Parker Hospital, and in October, 1897, transferred to Gouverneur Hospital. Small intubation tubes, or as small as the child could breathe through, were used with a change every two weeks.

In December, the child was one day found suffering with rather marked dyspnœa, and examination showed that the small tube had slipped down out of reach of the finger, inside the larynx; but after repeated trials it was finally extracted in the usual way. The stenosis thereafter seemed to become, if anything, worse. Without the tube no air could enter the trachea, and changing the tube had to be done with rapidity. Beginning with March, 1898, a

No. 4-5 tube, or one suitable to the size of the child, was inserted, and left undisturbed for a month or six weeks. No improvement occurred, and in August a 6-7 tube was employed. Upon the removal of this about seven weeks later, in October, respiration was barely possible for the first time. Acting on the hint given by the change for the better following the use of a large tube, the 8-10 tube was tried, but could not be inserted without considerable force. The 6-7 tube was therefore replaced, and after the elapse of a month, or in November, 1898, this was taken out, and no dyspnoea followed.

During the first twenty-four hours there were frequent attacks of coughing, after which inspiration was very difficult. Asphyxia seemed imminent. But under the administration of morphine this spasm of the adductor muscles gradually subsided, and two weeks later the child was dismissed with the stenosis entirely relieved. At the present time (February, 1900) there is apparently more or less catarrhal laryngitis, and occasionally, after coughing or violent exercise, a stridulous inspiration but no dyspnoea. The only vocal sounds of which the child is capable are peculiar harsh whisperings. Laryngoscopic examination is not tolerated.

The stenosis in this case persisted unrelieved from October, 1896, until October, 1898. The obstruction was complete, and immediately followed removal of the tube, and in all probability was dependent upon the thickening and hypertrophy of the soft parts found in the cases in which the interior of the larynx was seen. Intubation with small tubes frequently changed caused no improvement; while intubation with as large a tube as could be introduced without extreme violence, and its retention in the larynx for a long period undisturbed, brought about a cure.

CASE VII.—M. R., three years of age, was admitted to the Willard Parker Hospital, February 2, 1899, with diphtheria, which had required intubation two days previously. Four days after admission he began to cough out the tube,—at first every day or two, and then several times a day. Respiration without the tube was not possible for more than a few minutes. The stenosis persisted, and autoextubation became more frequent and dan-

gerous, and therefore, on February 18, tracheotomy was performed. On March 31, or a month later, intubation was again attempted; but it was found that the tube could not be retained, and the tracheal canula had to be allowed to remain. Another unsuccessful attempt to substitute intubation for tracheotomy was made on April 18.

At these trials it was found that neither a large size nor a "special" tube could be kept in the larynx. It was always soon coughed out, and the resulting respiratory obstruction followed immediately, and was complete. In other words, the stenosis must have been dependent upon a vascular thickening of the intralaryngeal soft parts, as described in the other cases. A cicatrix would hardly close immediately when relieved of the pressure of the tube, and granulations which might act thus would certainly have bled, and hæmorrhage was never noted. Finally, on June 27, the child was dismissed from the hospital breathing through the tracheal canula. Early in October I examined him at Gouverneur Hospital and found a rather dense cicatrix surrounding the tracheal fistula. When the fistula was occluded, inspiration was impossible, but a small amount of air could be expired; therefore the lumen of the larynx was evidently not entirely obliterated. On October 19, 1899, under chloroform anæsthesia, I succeeded in passing a uterine probe from the tracheal opening up through the larynx into the mouth. This was followed by male urethral sounds, beginning with the smallest until a No. 21 F. was reached. The next larger size met with such firm resistance that it seemed unwise to force it through and lacerate the unyielding tissues. A 4-5 intubation tube was then inserted in the usual way, but required really considerable pushing with counter pressure on the larynx externally before it slipped in place. The tracheal canula was removed. No cough and no pain followed this rather violent treatment of the larynx, and no autoextubation occurred. The tube was changed at the end of ten days under chloroform anæsthesia, and the next larger size, or a No. 5-7, substituted. This was allowed to remain undisturbed for five weeks, during which the child passed successfully through a rather sharp attack of pneumonia. On December 4, 1899, the tube was removed, again under general anæsthesia; and up to the present time (February, 1900) there has been no return of the stenosis and no difficulty with respiration.

The parents inform me, however, that at night breathing is rather noisy, although there seems to be no dyspnoea. At first the voice was entirely absent, but lately it has begun to return, and, though hoarse and harsh, is not unsatisfactory.

This case is noticeable on account of the frequent and uncontrollable autoextubation, which was dangerous, and necessitated tracheotomy. Attempts to substitute intubation for tracheotomy twice failed on account of the same dangerous complication. I am unable to say whether this happened because intubation was tried too soon after the tracheotomy, or because large enough tubes were not used. At any rate, the third attempt, undertaken after the larynx had been allowed to rest for six months, or from April to October, succeeded; and then a tube was employed as large as could be forced into the larynx without rupturing the tissues. The continued use of such a tube, changed only when it became loose, effected a comparatively rapid cure. As stated in the history, the stenosis was probably dependent upon a vascular thickening or hypertrophy of the intralaryngeal soft parts.

As regards the etiology of postdiphtheritic stenosis of the larynx and retained intubation tubes, the views of the late Dr. O'Dwyer are, of course, worthy of the greatest consideration. Nevertheless, I believe they are wrong. He maintained that the condition was the fault either of the operator or of the instruments, which means careless or unskilful insertion or the use of a poorly constructed, and therefore improperly fitting, tube. Formerly, while he was experimenting with and perfecting his instrument, he sometimes encountered ulcerations and granulations; and the two cases he reports of granulations at the base of the epiglottis, where it impinged upon the head of the tube, might properly be counted in this class. At all events, there is no other record of a similar occurrence from the use of the hard-rubber tube as at present made. It must be admitted, however, that erosions and ulcerations are possible with a metal tube, as its surface soon becomes rough from a deposit of what is apparently calcareous matter. But whether

ulcerations and subsequent cicatrices may or may not be thus produced has very little to do with the matter, as they do not seem to be the usual cause of the stenosis in the reported cases.

If lack of skill on the part of the operator could occasion the stenosis, instances of it would be common; whereas, compared with the number of intubations, the necessity of prolonged or permanent retention of the tube is admittedly rare. Intubation is constantly being performed, and if there were often any difficulty with the subsequent removal of the tube, it would be constantly heard of in medical societies. The instance of necrosis of the cricoid cartilage mentioned by Dr. O'Dwyer may, of course, be cited as an example of unskillfulness or bad judgment on the part of the operator in using, during the acute stage of the stenosis, too large a tube or too much force in its insertion. But it is conceivable that such an accident could have happened without intubation. It seems, however, to be the only case of its kind on record; and if it was due to the tube, it might properly be mentioned as an exception which proves the rule. It is not the usual cause of a postdiphtheritic stenosis, and cannot be held to prove anything against the ordinary operator. I mention this matter of etiology somewhat at length because, following the authority of Dr. O'Dwyer, there seems to be a very general belief that either the operator or the tube is at fault. And it is important, from a medico-legal aspect, as well as for the sake of intubation, to show that neither the operator nor the tube ordinarily has anything to do with a possible postdiphtheritic stenosis. It is granted that lacerations and serious permanent damage to the larynx can, of course, be inflicted by extreme lack of skill or care; but to claim that this must have happened in all, or even some, of the cases of retained tube is not borne out by the facts. A certain amount of traumatism is necessarily inflicted at every intubation, and if, by any chance, a chronic stenosis follows, the traumatism is always blamed for it. That this is wrong, at least in the average case, is proved to my mind by the pathology of the condition. It is the same whether the stenosis follows intubation or a primary tracheotomy.

Emil Köhl, in his inaugural address at Zurich in 1884, described very fully the pathological condition of the larynx in cases of chronic postdiphtheritic stenosis with retained tracheal canula. This article demonstrates most conclusively that not the least frequent cause of the difficulty is a chronic hypertrophic subglottic laryngitis, a chronic thickening of the soft parts between the vocal cords and the lower border of the cricoid cartilage. The hypertrophy of the soft tissues was so marked that respiration, except through the tracheal fistula, was impossible. These cases, of course, had never been intubated, and therefore the chronic inflammation within the larynx cannot be charged to the irritation or traumatism consequent upon the insertion or wearing of an intubation tube.

Another and more frequent cause of the stenosis was shown to be granulations and cicatrices in the neighborhood of the tracheal wound or canula. And the nearer the canula was to the vocal cords the worse were these complications. The vicinity of the upper end of the wound was more prone to granulations and cicatrices than the lower, as the upper end generally involved or was close to the larynx where the mucous membrane is more loosely attached than below. This bears upon the cause of the stenosis described in some of the reported cases of retained tubes which have finally been tracheotomized. If the tracheotomy has existed long enough, it, and not the original intubation, may have given rise to the cicatricial tissue.

Incidentally, it may be noted that the number of devices described by Köhl for remedying a postdiphtheritic stenosis well illustrate the difficulties in the way of successful treatment other than by intubation.

Dr. O'Dwyer believed that the usual cause of the obstruction in cases of retained tube was virtually a chronic inflammation of the intralaryngeal soft tissues; but I can find no detailed report by him of cases in which this was found. That it is entirely correct, however, is conclusively shown by the histories of Cases II, III, and V. In each there was a simple thickening of all the soft tissues. No ulcerations, and, of course, no granulations, could be detected. The microscopical

examination of a cross section of the larynx of Case III showed a hypertrophy of the mucosa and submucosa, with a round-celled infiltration of the vascular subjacent tissues. And a similar examination of the strips of tissue excised from the larynx of Case II corresponds very closely with this in respect to the thickening and infiltration, though the remark about there being "in places granulation tissue" is a little confusing. But as the gross appearances of both larynges were so closely similar and no ulcerations were seen, I believe that this must refer to some collection of round cells in the interstices of the tissue, which, as the specimen had been isolated from the surrounding parts, was like the granulations of an ulcer. Furthermore, the report speaks of it as *apparently* granulation tissue. Hence, for all practical purposes, these cases represent the same condition of chronic inflammation and hypertrophy.

The first case described by Boulay was only seen by the laryngoscope, and in a child this is difficult; but apparently the only cause for the stenosis was a marked subglottic thickening. At all events, no ulceration and no granulations could be detected. Baer's case can only be assumed to be in this class by inference, though this is rather strong. The case was tracheotomized for frequent autoextubation, and, as the stenosis persisted, the larynx was explored by a thyrotomy. The tissues thus seen are described as reddened but not ulcerated. A fold of mucous membrane was cut away, and also some granulations near the tracheal fistula. Nevertheless, the stenosis was unrelieved; and as nothing else is mentioned as a possible cause of the obstruction, it must have been the "reddened" and so probably inflamed and thickened soft tissues.

I am inclined to believe that Case I was another instance of the same condition, but the report of the operator must stand as evidence that the stenosis was dependent upon ulceration and cicatricial contraction. In this case, however, after intubation had been continued for several months, the larynx was curetted, and ulceration, if it had not previously existed, might then have followed.

Boulay's second case seems probably to have been one of

cicatricial stenosis. The larynx was inspected during the course of a thyrotomy after a tracheotomy for frequent auto-extubation, and was found filled with cicatricial tissue. Still, it is not impossible that this could have been an extreme development of the common cicatrix above a tracheotomy wound. Practically, the same history and condition are revealed in Galatti's second case, but the general redness and swelling of the soft parts described as existing above the cicatrix near the tracheotomy wound, makes me believe that the true cause of the original stenosis was this hypertrophy, and not, as given by the author, the cicatrix. There is apparently no reason to doubt, however, that Galatti's first case was one of cicatricial stenosis occurring within the ring of the cricoid cartilage. He was compelled to cease intubating after the tube had been coughed out a number of times, and, as tracheotomy was refused, the child finally died from increasing dyspnoea. Ulceration, as Dr. O'Dwyer has demonstrated, is most prone to occur in this region from the use of too large a tube. But, like the instance of necrosis of the cricoid cartilage, it is an exceptional accident, and can prove nothing against the operator or the instrument. If continued intubation had been permitted in this case of Galatti's, the ulcer would have healed, and been prevented from contracting by the presence of the tube.

Dr. Northrup's case is the only other I have been able to find in which the larynx was plainly obstructed by a cicatrix apparently the result of the intubation. Unfortunately, I am only familiar with the specimen, and so cannot intelligently discuss its history; but this and the one reported by Galatti and the necrosis of the cricoid cartilage narrated by O'Dwyer seem to be all that can be directly traced to the tube, and, in view of the circumstances, should be considered only as among the dangers of a necessary operation. O'Dwyer's cases of exuberant granulations at the base of the epiglottis seem to have occurred before his instrument was brought to its present perfection, and need simply be mentioned as warnings of possibilities. A metal tube which quickly becomes roughened by an apparently calcareous deposit might readily occasion a

similar trouble. It could be overcome, as O'Dwyer suggested, by a rubber tube, with the head built up to relieve the ulcer of pressure, and in addition by curetting.

To sum up: the commonest cause of postdiphtheritic stenosis necessitating long-continued intubation is a hypertrophy of the subglottic tissues accompanied by a chronic inflammation. The intubation is in no way the cause of this, as it occurs irrespective of the operation. Less often there is an ulceration, and subsequently a formation of a greater or less amount of cicatricial tissue and contraction. This likewise is not the result of the intubation except in rare and practically unavoidable instances. But it certainly may follow a tracheotomy, and in a larynx already chronically stenosed it makes the condition worse, but not necessarily more difficult to cure. Exuberant granulations *within* the larynx apparently do not occur with intubation, no matter how prolonged. I should add that in a recently published book on "Tubage et Tracheotomie en d'hors du Croup," by Antoine Sargnon, a half-dozen more cases of retained tubes are cited in which ulceration and cicatrization are mentioned as causes of the stenosis, but without details; and, as I could not find the original references, I cannot well discuss them.

The frequency of the occurrence of a postdiphtheritic stenosis accompanying intubation is a matter of some interest. Dr. Dillon Brown, of New York, informs me that he has encountered it about once in every seventy-five or 100 cases. Dr. C. G. Jennings, of Detroit, with an equally large experience, writes that he has never met with the severer forms of the difficulty, but that in two or three instances he has had to continue the intubation as late as the third week after the first insertion before recovery was complete. His associate, Dr. Shurley, has never had any trouble with delay in the removal of the tube. Galatti, in the article above referred to, states that he had these two chronic stenoses in thirty-one intubations. He reports Ranke as having had one case in many hundred, Hubner, one in 250, and Bókai two in 800. Dr. George McNaughton, of Brooklyn, writes me that he has had but few cases in many

hundred, and these recovered at the latest within several weeks. At the Nursery and Child's Hospital in this city there have been no cases of noticeably prolonged intubation. The New York Foundling Hospital has had six cases in a total of approximately 500. Investigation of the statistics at this institution, by the way, forcibly illustrated the advantages in the use of the diphtheria antitoxin. The house physician complained to me that, before the introduction of this remedy, his predecessors had always averaged at least one intubation a week, and thereby obtained much valuable experience; but about the time he came into the hospital, the rule was instituted that antitoxin should be given to every patient as soon as there was any suspicion of diphtheria. The result was that he had never in a year's service had a single opportunity to practise the operation on a living subject!

In the Willard Parker Hospital, about 900 cases have been intubated in the last six years; and the same experience with antitoxin was noted as in the Foundling Hospital, but not so impressively, as the patients are generally brought in after the disease has existed some time. The internes of several years ago, however, had far more experience with intubation than those at present on duty. In these 900 cases there have occurred eight of chronic stenosis. A reasonable estimate of its average frequency would be therefore not far from once in every 100 intubations.

Their treatment by surgical operation, which has generally implied opening the larynx by median division of the thyroid and cricoid cartilages and excision of the obstructing tissues, has been noticeably unsuccessful. The various ingenious devices, such as skin-grafting the resulting raw surface or the insertion of some form of tracheal canula made on the principle of Dupuy's T-shaped instrument to keep up a more or less constant dilatation, and at the same time allow respiration through the fistula, have all failed.

Many of the less troublesome cases will recover after two or three or four weeks with intubation as it is ordinarily practised, or with the O'Dwyer "special" tube. This has the re-

taining swell transferred from the middle to the lower end, and is so made as to relieve the supposedly irritated and swollen tissues of all the pressure possible. A local application of an astringent like alum powdered on the outside of the tube previously coated with gelatin, as advised by O'Dwyer, will occasionally succeed. But in spite of these measures, coupled with very careful intubation, some of the patients will not improve, and there seems to be no way of differentiating these at the outset from the other less persistent cases. The lesion is apparently the same, and therefore I believe that nothing is to be gained by treating them differently after the acute stage of the inflammation has subsided. Before that time, anything but the most gentle interference would be dangerous. If only the chronic postdiphtheritic stenosis is not believed to be dependent upon previous intubation, the course is clear. On the other hand, if one clings to the idea that the stenosis is in consequence of the intubation, and that therefore something else must be tried, a failure will result. Dr. O'Dwyer long since proved that the continued uninterrupted use of as large a tube as could be crowded into the larynx was an almost infallible cure for the stenosis necessitating indefinite retention of a tracheal canula. It applies just the same to the stenosis accompanying intubation, and is proved in Cases V, VI, and VII. To these I am enabled, through the kindness of Dr. Kennefick, of this city, to add one more. He had in his service at the Manhattan Eye and Ear Infirmary a child with a postdiphtheritic stenosis, which had necessitated a retention, with frequent changes, of the ordinary-sized tube for five months. At my suggestion, this small tube was removed and one substituted for it as large as could be introduced without lacerating the tissues. This was not disturbed until the elapse of about six weeks, when the stenosis was found to have disappeared, and has not since recurred. In Case VI, which was similar in never having been tracheotomized, there was no improvement in the stenosis from October, 1896, until August, 1898. Then this principle of using as large a tube as possible was adopted, and a cure resulted in eleven weeks, during which

time the tube was changed once. Case V suffered from the stenosis and several operations, alternating between intubation, tracheotomy, and laryngotomy, from December, 1896, until August, 1898, and then, with a large tube changed once, finally recovered in about ten weeks. In Case VII, the stenosis which had existed from February until October, 1899, was not relieved by the ordinary method of intubation, and, of course, not by tracheotomy. The use of a large tube, changed once, and that as soon as it became loose, for a still larger one, effected a permanent cure in about six weeks.

As tracheotomy in these cases seems often to add a cicatrix to the stenosis, even if it had not previously been the cause of the obstruction, it is interesting to note that the gravity of the prognosis, so far as the stenosis is concerned, is not materially increased. That a tracheotomy may be necessary is evidenced by the history of J. F., who died before relief could be obtained after coughing out the tube. I am unable to say why autoextubation is so frequently a complication. It seems to occur about the third or fourth week after the first intubation, and may be dependent upon a loss of power in the laryngeal muscles owing to their involvement in the chronic inflammatory process. It is not apparently from a true paralysis; at least, no evidence of this could be found in the only specimen examined microscopically. But when it occurs repeatedly, and is not checked by increasing the size of the tube, tracheotomy is certainly indicated. Several weeks or even months may have to elapse before intubation can be safely resumed. Then, if the larynx has undergone cicatricial contraction, and it is the more likely to occur the nearer the wound is to the larynx, this contraction should be dilated with urethral sounds passed from below up, until a tube can be introduced as large as the larynx will bear without rupturing the cricoid, or, if this has been divided in the tracheotomy, without producing a slough. It may be impossible to fully dilate a cicatrix at one sitting, and in that case the tube should be increased in size as soon as the tissue has yielded, or in about ten days or two weeks. This tube, then, should not be disturbed for five

or six weeks. Then, whether the stenosis is dependent upon hypertrophied or cicatricial tissue, or whether it originally accompanied an intubation or not, at least marked improvement can be confidently expected. It is possible, however, that another five or six weeks may be needed to effect a cure. The metal tubes cannot be used, as they soon become roughened, and so may cause ulcerations, or even become clogged. The hard-rubber instrument is ideal, as it never becomes foul or loses its smoothness, and therefore can be carried for almost an indefinite length of time in the larynx.

I have always employed general anæsthesia whenever it was necessary to intubate or change the tube in these chronic cases, as I am not an expert in the operation and desired to have the patient perfectly quiet and ready for any contingencies. In skilled hands, however, this should not be required except when forcible and rapid dilatation has to be accomplished.

From the experience noted at the Foundling and Willard Parker Hospitals, there seems good reason to believe that, as the diphtheria antitoxin comes into earlier and more general use, the need of intubation for acute stenosis will gradually disappear. But for chronic laryngeal obstruction, nothing can take its place.

All honor to the name of Dr. Joseph O'Dwyer.

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