is no strong reason against the adoption of intubation in selected cases. Any surgeon who intubates must be prepared to do tracheotomy at once if necessary.

[Reference was then made by the lecturer to the more recent results and opinions on the subject in America and on the Continent.]

ULCERATION OF THE LARYNX.

Ulceration of the larynx in children may be due to tubercle, syphilis, typhoid, scarlet fever, or measles, or it may be the result of the impaction of a foreign body, or occur without obvious explanation as to its cause. Illustrative examples were cited.

FOREIGN BODY IN THE AIR PASSAGES.

The symptoms attending the introduction of a foreign body into the air passages, and its lodgment there, are so well known that a recapitulation of them is unnecessary. On most occasions the diagnosis is clear, but occasionally, in the absence of history, and from special peculiar features in the case, it is very difficult to arrive at any certain conclusions. On the other hand, the history of swallowing a foreign body may be imaginative on the part of the friends, and have nothing to do with the case under consideration. When we remember how constantly children are running about at play with some foreign body in their mouths, and how often they laugh or cry quite suddenly in the midst of eating their food, it is remarkable that the accident does not more frequently occur.

When a foreign body is impacted in the larynx after the initial paroxysm has subsided, the symptoms may be very slightly marked, though usually there is alteration of voice, some cough and dyspnoea, with pain and tenderness about the larynx. The difficulty of diagnosis in very young children may be very great. Occasionally even in children the foreign body may be seen with the laryngoscope. If a foreign body remain in the larynx it may at any time prove fatal by giving rise to inflammatory oedema of the glottis, or, as shown by cases narrated, the ulcer may occasion pneumonia. The body may be expelled by coughing, or change its position, and pass into the trachea or one of the bronchial tubes. If it remains for a prolonged period in the larynx the voice is likely to be permanently affected, and such stenosis of the larynx may result as to necessitate the wearing of a tracheotomy tube.

When a foreign body is lodged in the trachea there are usually signs of its presence on auscultation, and the recurrent attacks of dyspnoea, as the body changes its position, are very characteristic.

A small smooth body, especially if heavy, is more prone to descend into one of the bronchial tubes than an uneven light body, which is likely to remain in the trachea. The tendency of the body to pass into the right bronchus rather than the left has generally been ascribed to the fact that the septum at the bifurcation is somewhat to the left of the middle line. Dr. Alby, of Rome, has demonstrated that the right bronchus more closely follows the line of the trachea than the left. It is probable that foreign bodies find their way into the left bronchus much more often than is generally supposed. There appears to be a good deal of difference of opinion amongst the compilers of statistics as to which bronchus has the preference.

When a foreign body enters one of the bronchi, the symptoms produced must vary with its size, position, and character. Thus, if it completely blocks the main bronchus, the proper respiratory sounds are absent. If it partially obstructs, then a loud musical sound is likely to be heard, with diminished breathing in the lung of the affected side. There is generally pain, cough, and perhaps some blood-tinged expectoration. The affected side is at first resonant on percussion. If the body be impacted in one of the secondary divisions, then only a limited area of the lung will at first be affected.

If a foreign body remain impacted in a bronchus, inflammatory symptoms generally supervene, and may be localised or general. The results are very variable. Both the bronchi can be easily explored from a low incision in the trachea, and many cases are on record where various substances have been thus removed successfully in children, and much ingenuity has been exercised to meet the mechanical dif-

iculties in particular instances. Death may result both after the removal by operation and after its spontaneous ejection. In several recorded cases where an unsuccessful search has been made, the tracheotomy wound has been left open and the body ejected at a later period. When an abscess forms in the lung, recovery is very rare. The usual mode of death with obstruction of bronchus by foreign body is pneumonia with abscess or gangrene. Death may, however, take place quite suddenly from the body getting displaced from the obstructed to the sound side.

ON A CASE OF POLIOENCEPHALOMYELITIS WITHOUT ANY ANATOMICAL LESIONS.

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THE following case affords an example of polioencephalitis. The affection commenced with paralysis of the eye muscles, which disappeared after some months, reappeared after some years along with symptoms of bulbar paralysis and while the eye muscles regained their power, the bulbar symptoms, along with symptoms of weakness in some of the spinal muscles persisted till some years after; somewhat suddenly there appeared again complete ophthalmoplegia externa with almost complete paralysis of all the motor cranial nerves and of the upper cervical nerves, with atrophy of some of the affected muscles, whilst there were no sensory disturbances of any kind. At the necropsy a most careful examination of the peripheric and central nervous system showed nothing to account for the symptoms observed during life:

M. A. H., a widow, aged 36, was admitted into the Manchester Infirmary on November 19th, 1891, when the following notes were taken by Mr. C. E. McCarthy, M.B. (Vict.), then Clinical Clerk.

History.—Up to fifteen years ago she was a strong and healthy woman; she then was attacked with drooping of both eyelids, for which she attended the Manchester Eye Hospital, and in about six months completely recovered. For the next five years she remained in good health, when she had a second attack of double ptosis with diplopia; almost simultaneously she began to have difficulty in talking and swallowing, and experienced weakness in her arms. The eye symptoms again improved in about nine months (she was an inmate then of the Workhouse Hospital), but her ability to talk or swallow did not improve, and as regards these symptoms she says that it took her a long time to eat her meals, and the food used to lodge in her mouth between the teeth and the inside of the cheek; she would have to stop eating at times because her jaws ached, and she had to support them with her hands, the swallowing also became difficult; she had difficulty in speaking, as she could not pronounce some letters; she had not been able properly to open her mouth or to protrude her tongue, nor was she able to whistle, whilst before she could whistle well; the weakness in the arms, or rather shoulders, also persisted. In spite of these symptoms she was able to work first as a dressmaker, and latterly in a bakehouse, till five weeks before admission, when she became worse; the eyelids suddenly drooped and she could only see by raising the lids with her fingers; the swallowing became very difficult, and her speech so indistinct that she was not easily understood; the shoulders and arms also became much weaker. All these symptoms gradually grew worse, and latterly she began to suffer from difficulty of breathing, she then sought admission into the hospital. There was no history, and there are no signs of syphilis. As regards her social history, she stated that ever since her marriage (fifteen years ago), she had led a hard life; her husband had been a careless, lazy man, of drunken habits; he died of phthisis five years ago. She had had five children: four born alive, and one (the fourth) stillborn; three of her children were alive and in good health, one died of consumption of the bowels. Her father died, aged 48, cause of death not known; her mother was alive and well; she had three brothers and five sisters, all in good health.

Condition on Admission.—The patient was propped up in bed, as, owing to the dyspnoea, she could not lie in the recumbent position. She had a worn, languid, and (owing to the ptosis) a sleepy appearance (facies Hutchinsonian). She was very emaciated; the skin was dry and wrinkled; temperature 97.8° F. There were no enlarged glands and no oedema. There were no signs of any mental disturbance; she appeared a very intelligent person; there was no vertigo, no headache, no pains in any part of the body. Whilst there was no affection of the special-sense organs or of the sensory nerves, there was marked paralysis of all the motor cranial nerves. Smell, taste, hearing, and vision were perfectly normal, and an ophthalmoscopic examination showed a normal fundus. The internal muscles of the eye were normal; the pupils were equal and reacted to light and accommodation. The external muscles supplied by the third nerve were nearly all completely paralysed; the inferior rectus of the right eye acted slightly, as did the left levator palpebrarum. The right internal rectus and the right levator palpebrarum and the inferior and superior rectus on each side were completely paralysed. The fourth and sixth nerves on both sides were completely paralysed. Of the fifth nerve the motor portion only was affected. The masseters and pterygoids and temporals were much weakened, though not completely paralysed. Of the facial nerve both the upper and lower branches were affected; the patient could not close either eye; she could not wrinkle the forehead or

frown; the lips could not be pressed together, and the patient could not smile; whistling was equally impossible. The azygos uvulae and platysma were also paralysed. The tongue lay on the floor of the mouth, and was somewhat atrophied. It could not be protruded beyond the oral orifice, and its movements in the mouth were very limited; the patient could not touch the roof of the mouth with its tip, nor remove the food from between the gums with it; the depressors of the hyoid, though not completely paralysed, were very weak. The other cranial nerves—glossopharyngeal, pneumogastric, and spinal accessory—were also affected in a marked degree. The soft palate was paralysed, and the palate and fauces showed no reflex movements on being touched or tickled. Swallowing was very much impaired; mucus and saliva collected in the fauces and posterior nares, and, to get rid of the accumulation, the patient closed the nostrils with her fingers and made quick respiratory efforts. The sensation of the mucous membrane of the oral cavity was not impaired. Articulation was very much affected. The words uttered were so indistinct that the patient answered most of the questions put to her by writing, and the voice was low pitched and weak. Laryngeal examination was carried out with great difficulty; it showed the abductors to be weak, as seen on deep inspiration. The sterno-mastoid and trapezius, though thin and somewhat atrophied, acted with some force. The pulse was 130, regular, but weak. The diaphragm acted slightly, the respirations were 40, shallow, and short; the deltoids were atrophied and almost completely paralysed. The muscles of the upper extremities were emaciated, but there was no paralysis and no specially-marked atrophy of any group of muscles. There were no fibrillary twitchings. The various sensations in the upper extremity were normal. The wrist, the radial, and the triceps reflexes were markedly increased; there were no contractures. The muscles of the trunk and of the lower extremities showed no paralysis or atrophy, and the various sensations were normal throughout. No fibrillary twitchings. The superficial and deep reflexes were present, but not exaggerated; there was no ankle clonus, and no signs of any contractures. The examination of the lungs showed extensive bronchial catarrh. The heart sounds were feeble; the heart was not dilated nor hypertrophied. The examination of the abdominal organs showed nothing abnormal. The urine, specific gravity 1020, was free from sugar and albumen.

Progress of the Case.—November 20th. Patient was worse. The diaphragm moved very little, and the patient suffered much from dyspnoea. She was only able to swallow very small quantities of milk, and this with difficulty. The pulse was 135, the respiration 40, and the temperature 97° F. Electrical examination of the facial nerve showed absence of degenerative reaction. The muscles responded to a weak faradic current with quick contractions, and on the application of the galvanic current KCC > ACC, and the contractions were quick. On November 22nd the patient was much worse. There was marked cyanosis. The respiration was of a spasmodic character, and the dyspnoea very great; the diaphragm had ceased to act; swallowing was quite impossible; there was no longer true articulation; the patient could only produce a faint grunt; the pulse was at times intermittent, very weak, and thread-like. During the night of November 22nd the breathing became more and more laboured, the temperature rose to 99.5° F., and the patient gradually sank and died.

Post-mortem Examination (by Dr. Kelynaek, Pathological Registrar).—There was commencing hypostatic pneumonia and generalised bronchitis, a white clot in the pulmonary artery, early atheroma of the aorta, slightly granular kidney, and a small dermoid cyst of the ovary. The muscles appeared healthy and of normal colour. The macroscopic appearance of the membrane of the brain and of the brain itself showed nothing abnormal, the ventricles were not dilated, and the brain substance on section showed no increased vascularity. Sections through the pons and medulla showed, both as regards colour, consistency, and vascularity, nothing abnormal; the several cranial nerves at the base of the brain appeared of normal colour and thickness. The spinal cord and its membranes showed to the naked eye a perfectly normal appearance.

Microscopical Examination (by Dr. Williamson).—The whole of the brain, pons, medulla, and cord were carefully hardened for microscopic examination. Serial sections were made of the pons, medulla, and upper part of the cord, and stained with the various staining agents, most of them after Weigert's method. The sections were carefully examined both by him and by myself, and compared with sections from a normal specimen. Without entering into a detailed description I may say that no changes either in the ganglia cells, or in the nerve fibres, or the neuroglia cells were found. In a few specimens the walls of the blood vessels appeared thickened, and showed slight cellular infiltration in the perivascular spaces, but no hæmorrhages; no degeneration of nerve fibres or atrophy of the ganglia cells could be seen. The nuclei of the third, fourth, and sixth, as well as the nuclei of the other cranial nerves, showed the ganglia cells in all their integrity with their processes, and in some places the network of fibres. Some of the cranial and spinal nerves were examined both fresh and hardened, and showed nothing abnormal. Of the motor cortex and of some parts of the basal ganglia sections were made and stained, and on examination gave equally negative results as to any pathological changes. Unfortunately no muscles had been preserved for microscopic examination.

The case is by no means isolated. A number of similar cases have been published, though perhaps none in which the paralysis of all the motor cranial nerves was so complete as in the above case, and in all those where a *post-mortem* examination was made no lesions were found to account for the symptoms. Hoppe¹ records one case, and quotes cases observed by Wilks, Eisenlohr, and Oppenheim where the necropsy gave negative results. In Oppenheim's case there was, however, no external ophthalmoplegia, and in Wilks's case the ophthalmoplegia was very incomplete. Senator² records a case in which symptoms of bulbar paralysis—with-out ophthalmoplegia—and of right hemiplegia came on sud-

denly, and persisted more or less for four months, when the patient died, and here again the examination of the nervous system and of the muscles showed no changes.

Eichhorst³ relates the case of a girl, aged 19, who for some months suffered from left facial palsy and diplopia; this was followed by mental troubles, bilateral paralysis of the hypoglossus, paralysis of the glossopharyngeus and vagus, ophthalmoplegia externa, and progressive muscular atrophy. Four days after leaving the hospital she was attacked with severe dyspnoea and died suddenly. The brain and spinal cord were found healthy on macroscopic examination. The microscopic examination had not been made at the time of publication.

Bernhardt⁴ briefly relates a case in which there was double ptosis and paresis of some of the other ocular muscles, with bulbar paralysis and atrophy of some of the muscles of the back of the neck which terminated fatally, but where no necropsy could be obtained.

Other similar cases—which at the time of publication were still under observation, and about the further history, as far as I could make out, nothing has yet appeared—are given by Seeligmüller,⁵ who was the first to use the term "polioencephalomyelitis," and whose patient, a female, aged 23, had been affected for some years with weakness of the extremities, wasting of the triceps, deltoid, sterno-cleido-mastoid, and vastus externus of each side, with almost total ophthalmoplegia externa, and with affections of the facial, motor portion of the trigeminus, the hypoglossus, glossopharyngeus, and spinal accessory; by Schaffer,⁶ and by Guinon and Parmentier⁷ in a series of papers entitled *External Ophthalmoplegia combined with Glossolabio-laryngeal Paralysis and Progressive Muscular Atrophy*. These authors give two cases observed in Charcot's clinic, in one of which, besides the ophthalmoplegia and the muscular atrophy, there were bulbar symptoms—difficulty of deglutition and slight affection of speech.

It is certainly most singular that in those fatal cases so far recorded no anatomical lesion has been found, though the paralysis of the muscles implicated is so very marked. There can be little doubt that some lesion does exist, though it at present has escaped our methods of investigation. Hoppe and Senator are inclined to place the lesion in the higher centres of the brain. I believe, however, that the lesion exists in the nuclei of the cranial nerves either in the ganglia cells or in the fine network of fibres and processes which connect these cells with the nerve fibres. That the primary cause be some toxic agent is not very probable, though such a cause cannot be altogether excluded; yet one would expect, if such were the case, some recognisable changes, considering the chronicity of the disease.

I regret that the affected muscles in my own case were not examined microscopically, yet it is highly improbable that the affection is a myopathic one, for atrophy was noticed only in a few muscles in my own cases, whilst in Hoppe's case the muscles were not atrophied, and in Senator's case the muscles were carefully examined and showed no noteworthy changes. It will be seen that the case which I observed and those which I quoted, with the exception of Oppenheim's and Senator's case, present, although they differ both as regards the onset and as regards the degree of the paralysis, the same principal features, namely, a combination of polioencephalomyelitis superior with polioencephalomyelitis inferior and with spinal paralysis, and more or less atrophy.

It is well known that ophthalmoplegia externa may occur in combination with other nervous affections, thus we may have it combined with polioencephalomyelitis inferior alone, of which several cases have been recorded, or with spinal atrophy or locomotor ataxy. In some of these latter cases distinct changes in the nuclei of the several cranial nerves and fibres have been found, as in those published by Buzzard, Ross, Gowers, Westphal, etc. It is needless for me to refer to these various combinations as an exhaustive account of them has been given by Guinon and Parmentier in the papers quoted above.

REFERENCES.

- ¹ *Berliner klin. Wochenschr.*, 1892, p. 332.
- ² *Neurolog. Centralbl.*, 1892, p. 168.
- ³ *Corresp. Blatt f. Schweizer Aerzt.*, 1889, p. 432.
- ⁴ *Neurolog. Centralbl.*, 1892, p. 189.
- ⁵ *Ibid.*, 1889, p. 153.
- ⁶ *Centralbl. f. Nervenhilfswiss.*, 1891, p. 193.
- ⁷ *Nouvelle Iconographie de la Salpêtrière*, vol. iii and iv.