

## REPORTS

OF

MEDICAL AND SURGICAL PRACTICE IN THE  
HOSPITALS AND ASYLUMS OF GREAT  
BRITAIN AND IRELAND.

## EAST LONDON HOSPITAL FOR CHILDREN.

THREE CASES OF MEMBRANOUS LARYNGITIS.

(Reported by T. E. HAYWARD, M.R.C.S.E., Resident Medical Officer.)

[Under the care of Dr. EUSTACE SMITH.]

CASE I.—Elizabeth C., a well-nourished child four years old, was admitted on September 25th, 1878, with distinctly stridulous breathing, and well-marked recession of the walls of the chest and cyanosis. There was distinct false membrane on the pharynx and one tonsil, while the glands under the jaw were enlarged. The child was restless, and struggled for her breath. Four days previously, she had become a little hoarse; two days later, breathing became stridulous, and it became urgent shortly before admission. Chloroform being given, the trachea was opened above the thyroid, the incision extending into the cricoid, the wound was dilated, and then a thick piece of membrane, one-and-a-half inches long, and presenting a cast of the trachea, was expelled. The trachea was then cleansed with a feather passed through the wound and from the glottis. A large silver tube was inserted, and the child was placed in a steam-tent, the steam being impregnated with carbolic acid; subsequently, solution of thymol was substituted. The tracheal tube was frequently cleaned with a feather dipped in solution of bicarbonate of soda (ten grains to an ounce), while at intervals the spray of the same solution was inhaled, with the object of partially dissolving the diphtheritic membrane and aiding its expulsion. There was no albuminuria; the lungs were almost clear, and remained so; the temperature varied from 100 to 102 deg. Fahr. A mixture of iron and quinine was ordered, with half a teaspoonful of brandy every two hours.

The next day, the child was quite comfortable, and took food well. Much viscid mucus and some membrane were expelled through the tube, which was frequently cleaned.

September 28th.—Vesicles appeared around the wound, and resulted in distinct white patches of membrane. Considerable inflammatory œdema of the neck and upper part of the sternum followed; this was painted with a solution of liquor ferri perchloridi, and subsequently a mixture of liquor plumbi subacetatis and milk was applied, and the swelling subsided.

October 4th.—The tube was removed for a time, and she could breathe freely. Convalescence proceeded favourably, and she was discharged in good health on December 1st.

(Under the care of Dr. H. DONKIN.)

CASE II.—Theodore R., a rather fat child, two and a quarter years old, was admitted on September 25th, 1878. He was struggling for breath; the face was flushed, and expressed great distress; there was a frequent croupy cough; the fauces were inflamed, and the tonsils swollen, presenting distinct white patches of membrane. Inspiration was prolonged, with stridor and recession of the chest; respiratory sounds were feeble over both bases, with impaired resonance on percussion. Temperature 100 deg. Fahr.; pulse 128, weak. He had been attacked with hoarseness of voice on September 19th, and respiration became gradually more embarrassed till his admission. Smells from the drains had been noticed at home. Chloroform being administered, tracheotomy was performed with incision of the cricoid cartilage, and the same precautions as in Case I. A large cast of the trachea was extracted from the wound; immediate relief followed, and the patient was placed in a steam-tent. The after-treatment was conducted as in the preceding case. The urine contained one-sixth of albumen.

During the next few days, he had some violent fits of coughing after the soda-spray, with expulsion of membrane; one piece was one-and-a-half inches long, and appeared to have come from the bifurcation of the trachea.

September 28th.—Coarse rales were heard over both lungs; the edge of the wound was covered with white diphtheritic exudation; membranes and some blood-stained mucus were discharged from the wound. This continued occasionally till October 9th.

October 11th.—There was no albuminuria. He could breathe without the tube during the day, but dyspnoea came on during sleep, necessitating the continued use of the tube. There seemed to be some

paralysis of the laryngeal muscles; this was only temporary, and did not extend to other parts. He rapidly regained strength, and was discharged on January 8th.

REMARKS.—These cases were treated on the principles advocated by Mr. R. W. Parker in his paper, read at a recent meeting of the Royal Medical and Chirurgical Society. Each case appeared to be one of well-pronounced diphtheria, and their recovery may seem some encouragement to the early performance of tracheotomy, combined with careful after-treatment.

(Under the care of Dr. H. DONKIN.)

CASE III.—Mary R., aged three months, was admitted on September 30th, 1878. She was a fairly well-nourished child, with marked laryngeal stridor, and the aspect of great distress; the lips were bluish; there was great epigastric recession. There was considerable œdema and redness of the submaxillary region, due in part to the previous application of sinapisms. No membrane was seen on the fauces, but the examination could not be satisfactorily made. She had suffered from cough for three weeks, but the urgent symptoms were of only twelve hours' duration. There was no history of bad hygienic conditions. Chloroform being administered, the high operation was performed; some little difficulty was experienced in performing the operation, but there was not much bleeding. Some shreds of membrane were removed, and a large silver tube was inserted.

October 1st.—The child seemed relieved and better; pulse small and rapid; temperature 105 deg. Fahr., with signs of pneumonia over both bases. The tube was changed, some membrane was extracted, and there was a free discharge of mucus from the wound; there was no lividity. Death occurred suddenly at 2 P.M.

Necropsy.—False membrane was found behind the posterior nares, none on the tonsils; small shreds could be peeled off the laryngeal mucous membrane; none was found in the trachea, but its mucous membrane was reddened. At the bifurcation of the trachea, and in each bronchus, were patches of membrane extending into the secondary tubes. Both lungs were partially pneumatic. The right side of the heart was distended with firm clot, extending through the tricuspid orifice.

REMARKS.—The age of the child, and the occurrence of pneumonia, left but little hope of a successful issue. Improvement occurred after the operation, and the sudden death appears to have been due to clotting on the right side of the heart, illustrating the danger of delay in performing the operation.

## NORTH-EASTERN HOSPITAL FOR CHILDREN.

CASES OF CROUP.

(Under the care of Dr. SANSOM.)

[From Notes by Dr. REID, House-Surgeon.]

CASE I.—A female infant, six months old, was admitted January 24th, 1879, about twenty-four hours after it had been seized with noisy breathing and cough, unaccompanied by any intermissions. Collapse of the chest-walls proved laryngeal obstruction, and a fragment of apparently croupous membrane was seen in the fauces, but could not be saved. There was no history of zymotic disease near the child's home. The child was placed in a steam-tent; emesis was at once produced by ipecacuanha, and a solution of ten minims of liquor potassæ in half-ounce of hot water was sprayed into the child's throat. A draught was ordered to be taken every two hours, containing three grains of bicarbonate of potash, a grain and a half of carbonate of ammonia, and two minims of chloroform. The breathing became less laboured; the general condition improved. The third night a solution of lactic acid, ten grains to one ounce, was applied with the laryngeal brush. The child was convalescent in five days, the temperature never having risen above 102.2 deg. Fahr.

[From Notes by Mr. MAJOR GREENWOOD, House-Surgeon.]

CASE II.—A boy, three years old, was admitted May 11th, 1877, with croupy cough and great dyspnoea, having been ill three days. There was no history of zymotic disease or croup in the neighbourhood. An emetic brought away patches of false membrane; his chest was dry-cupped, and he was ordered a mixture containing three minims of liquor potassæ for a dose. There were physical signs of obstruction of the left bronchus, and collapse of the chest was well marked. In a week he was convalescent.

REMARKS.—Dr. Sansom called attention to the effects of treatment in those cases. In Case I, the fragment seen in the pharynx stamped it as membranous tracheitis, and it appeared unlikely that it would have survived tracheotomy. The administration of potash was suggested by its successful employment in a case after tracheotomy, in which it ap-

peared to lessen the adhesion of the membrane. Dr. Sansom suggests the more extended trial of topical applications in the early stages of croup (membranous tracheitis) and the concurrent internal administration of alkalies.

## REPORTS OF SOCIETIES.

### PATHOLOGICAL SOCIETY OF LONDON.

TUESDAY, MARCH 18TH, 1879.

JONATHAN HUTCHINSON, F.R.C.S., President, in the Chair.

*Three Specimens of Abscess of the Liver.*—Dr. NORMAN MOORE showed these specimens. The first was taken from a woman who had suffered from chronic dysentery for five years. After death, ulceration of the cæcum was found. The hepatic abscess was partly in the right and partly in the left lobe, and contained eight ounces of pus. The woman had never been further from London than Ipswich. The second case was met with in a man aged 21, who had a sinus in the left leg communicating with a diseased ankle-joint. The presence of the abscess was not suspected until after death. Its walls were thick. He had been unable to find a record of any case of so large an abscess as was met with in this instance as the result of a sinus of that kind. The third case was one of multiple abscess. The portal vein was occluded by pylophlebitis, and in one of the large branches of the vein was a small abscess. The fatal inflammation appeared traceable to disease of the vermiform appendix.

*Piedra.*—Mr. MALCOLM MORRIS showed a series of microscopical preparations of hair illustrating this disease. The specimens exhibited were given him by Dr. Gutierrez. He had ascertained from Dr. Gutierrez that the disease occurs only on the scalp-hairs of women; it never affects the root; the nodes are found at irregular intervals along the hair; the disease is associated with a peculiar acid smell of the hair; it is not contagious; it occurs only in warm places. It is supposed to be caused by the use of a mucilaginous fluid like linseed-oil, with which the women wash their heads to keep it smooth and shiny. Another theory is that it is produced by washing in certain stagnant rivers which contain a mucilage. The hairs are dark in colour, weak, and flaccid. The nodes or piedra masses are intensely hard, producing a distinct noise when beaten against glass. If different specimens be carefully examined under the microscope, the tubercle in the earliest stage of development appears to originate from one cell, which grows by budding in every direction, forming radiating columns of spore-like bodies. As soon as the mass has grown to a certain size, the surface-cells seem altered in shape, becoming darker in colour, forming a pseudo-epidermis. In a transverse section, in addition to the spore-like bodies or hyphæ, rounded spaces of considerable size are seen which do not communicate with the surface, but are enclosed on all sides by the hyphæ. One of the specimens represented some of the node-substance broken up, and containing amongst the debris a number of elongated cells arranged in tuft-like groups, which appeared as if they had escaped from the beforementioned cavities. From these appearances, Mr. Morris believed it could be stated with confidence that the disease was fungoid in character, and that the fungus had attained a higher state of development than any of those hitherto described.—Dr. TILBURY FOX thought that the disease recently described in the journals was not parasitic, but resulted from degeneration of the hair and splitting up of the fibres. True piedra was parasitic, but not in the ordinary way, for the parasite grew on the hair and not in it. He believed that the fungus was identical with that described some years ago by Dr. Hermann Beigel as the chignon-fungus.—Mr. SANGSTER could not think that the fungus mentioned by Dr. Tilbury Fox was the same as that exhibited by Mr. Morris.—Dr. HOGGAN had always considered the supposed fungus described by Dr. Beigel to have resulted from the action of caustic soda upon glycerine.—The PRESIDENT suggested that possibly the real nidus in which the fungus grew was the oil and not the hair, and that its presence in the hair was an accident.—Dr. WILKS said that the specimens showed a condition quite distinct from that which he had described some years ago.—Mr. MORRIS stated that his object in bringing the specimen before the Society had been to establish the difference between real piedra and the condition which had recently been given that name in this country. He believed the disease described by Dr. Wilks was a variety of trichorrhæxis nodosa. He agreed with the President in thinking that the oil probably constituted the real nidus of the fungus.

#### DISCUSSION ON LARDACEOUS DISEASE.

Dr. DICKINSON stated that he should confine himself strictly within the limits laid down by the Council, viz., "Lardaceous disease in reference to its anatomical distribution and pathological relations".

Lardaceous disease appeared to have remained undetected until within the last half century. The disorder consists in a widely pervading tissue-change, which is shared by the blood-vessels, and the distribution of which externally to the vessels is regulated by their course, as if they were the channels of the morbid influence. It is nearly certain that the special change is of the nature of an infiltration or penetration of the tissues by a material, foreign to their healthy nature, which the blood-vessels bring to them. Lardaceous matter has not as yet been found in the blood; but the assumption that the blood-vessels convey it is warranted by all the circumstances of the disease. The infiltrating material is first found in the tissues, and in this situation it has been the subject of many conjectures. Rokitansky regarded it as albuminous; Meckel looked upon it as allied to cholesterine, and his views were adopted by Dr. Montgomery; Virchow considered it to be akin to starch or cellulose, and hence the name "amyloid disease". Succeeding observers, amongst whom must be mentioned Pavy, Odling, and Wilks, subjected the affected organs to ultimate analysis, showed that their proportion of nitrogen was the same as that found in the protein bodies, and that, whatever amyloid characters the morbid substance might have, it had not the amyloid composition. By these observations, the amyloid and cholesterine theories of the disease were exploded. The characteristic by which it is chiefly recognised after death is its reaction with iodine, which it absorbs eagerly. Whether it gives a blue colour with sulphuric acid and iodine, as alleged by some observers, is a matter of little importance. If such a tint be ever found, it is probably due to the presence of cholesterine and other fatty products. However well marked the iodine reaction may have been, it vanishes at once upon the exposure of the tissue to a very dilute solution of potash or soda, and this before there has resulted any material injury, even to the microscopic structure of the organ. That which gave the reaction has been dissolved out or modified; but it has been shown by Dr. Marcet that it can be recovered by the action of an acid. There is a point about the iodine reaction which is full of practical suggestion. It is closely associated with the condition of acidity. Fibrin can be made to display it by solution in hydrochloric acid, and to lose it again by being allowed to absorb potash. The question arises then, whether this deposit, which behaves with iodine as though it had come under the action of an acid, may not be wanting in alkali, so as at once to account for its separation in a solid state, and to distinguish it from the normal constituents of the blood, which, in organic composition, it nearly resembles. The answer is, that the new material is wanting in potash as compared with healthy tissue. In the liver, Dr. Dupré found a diminution from .283 to .151 in 100 parts, and in the spleen from .311 to .196. There are other alterations in the mineral constituents of the new material; the phosphoric acid is diminished, but not proportionately to the potash; the chloride of sodium is increased, the earthy matters rather remarkably so; and this characteristic forms an important distinction between lardaceous enlargement and rickety enlargement, which were at one time thought to be identical. In rickets, the lime-salts are diminished in the viscera as in the bones; in the lardaceous disease, they are increased. Lardaceous deposit resembles fibrin which has been artificially deprived of its alkali, not only in the iodine reaction, but also in its insolubility in water, its solubility in alkali, and its ultimate composition. In an investigation made by him at the request of the Society, Dr. Marcet found that an acid solution of fibrine is precipitated by iodine; an acid solution of albumen is similarly precipitated, whereas ordinary albumen is not. Lardaceous disease consists in a general deposition of the fibrin or albumen of the blood, modified by loss of alkali or gain of acid. The disease has been vaguely attributed to cachexia, including in this condition tubercle, cancer, bone-disease, syphilis, rheumatism, and drunkenness. Further investigation showed that two fundamental conditions constitute the general, if not the invariable, causes of the disease, viz., suppuration and syphilis. Out of eighty-three cases of lardaceous disease collected from the St. George's Hospital *post mortem* books, seventy-three were in connection either with protracted suppuration or with syphilis; suppuration occurred in sixty-two cases; syphilis in eighteen. As regards the distribution of the disease, the iodine reaction is found earliest and oftenest in minute vessels, which in most instances are arterial, though perhaps the Malpighian capillaries of the kidney are more early and more often affected than any other vascular structure. The straight arteries of the cones usually participate soon afterwards, or even simultaneously. The lardaceous condition is often succeeded by fibrosis. The kidney is not always the seat of earliest deposit; the liver is often involved at an early period, the terminal branches of the hepatic artery being first affected, and the cells of the middle zone of the lobule infiltrated. The spleen shares in the change, its Malpighian bodies assuming the well-known appearance of boiled grains of sago. The mucous membrane of the small bowel frequently displays the reaction