

less, her breathing entirely thoracic, and so noisy that nothing could be determined with the stethoscope. Her abdomen was distended with flatus, and painful on pressure; its walls were rigid and motionless. Sinking rapidly, she died the same morning at nine o'clock. Mr. Wood, to whose kindness I am indebted for this history, adds, "I could not help feeling that her mode of death resembled that from peritonitis from ruptured intestine;" and he suggested that this might be a case of spoiled and fatty diaphragm.

On making the post-mortem examination, twenty-four hours after death, the body was found tolerably well-nourished, but the integument was of a dusky purple, as with people dead from engorgement of the right heart and lungs. On opening the thorax the heart was seen considerably enlarged. The pericardium was adherent, as also were the pleuræ opposite the diaphragm and the lower parts of the chest. The cavities of the heart were dilated, and the left ventricle was also hypertrophied (thick-walled). The cusps of the mitral and of the aortic valves were thickened and fringed with deposits. The lungs and the right cavities of the heart were filled with dark fluid blood. The folds of the peritoneum were laden with fat, which tissue was present in only small quantities in other regions of the body. The remaining abdominal and pelvic organs presented a natural appearance. The skull and its contents were natural.

The diaphragm was pale, with the exception of those portions which arose from the bodies of the vertebræ, and retained their ordinary aspect. On closer inspection, the remainder of the muscle was seen to be mottled with pale-yellow specks; these resembled the markings often noticed in the muscular walls of a fatty heart. When examined under the microscope, this tissue was found degenerated into fat, the granules of which destroyed and took the place of the muscular structure. The heart was the seat of ordinary fatty degeneration; but the muscles of the body, such as the intercostals, the pectorals, and the psoæ, which were purposely examined after the condition of the diaphragm had been observed, were quite natural in appearance; and the same may be said of the voluntary muscles generally in the cases which remain to be described. A drawing of this diaphragm is in the museum of the hospital.

CASE 3.—In this instance, death resulted from a combination of causes, but the disease of the diaphragm had made considerable progress. A. M.—, male, aged fifty-three years, was admitted into the hospital, under the care of Dr. Hue, on March 18th, 1857. It was learned from his friends that evidence of chest mischief had existed for seven months. The day before his entering the hospital symptoms of coma were first noticed; they rapidly became confirmed and persistent. When seen, the patient was in a profound coma, which continued until death. Treatment availed nothing.

The body was greatly emaciated, and the muscles generally were pale, but otherwise natural. The arachnoid was opaque, and raised from the pia mater by a quantity of serous fluid. The brain had its ventricles greatly dilated and filled with clear fluid, but there was no evidence of tubercular deposits on their lining membrane. The lungs were laden with tubercles, and were riddled with cavities. The heart was pale, flabby, and the seat of fatty degeneration. The diaphragm presented appearances like those of the heart, the crura being the parts least affected. The yellow mottling and the granular fatty change in the fibres were distinctly marked. All the other organs presented a natural appearance.

CASE 4.—In September of the same year, J. A.—, a male, aged fifty-two, died in one of Dr. Farre's wards from extravasation of blood into the pons, medulla, crus cerebri and crura cerebelli of the right side, with a history of apoplexy of twenty-four hours' duration. Here also, in brief, the arteries, heart, diaphragm, and liver were spoiled by fatty degeneration.

It would be useless to enumerate other cases similar to those just related. The following history and post-mortem examination is, however, interesting as differing in many respects from those thus far referred to.

CASE 5.—E. C.—, female, aged thirty-two years, was admitted on February 12th, 1856, into one of Dr. Burrows's wards, having suffered for five months from the ordinary symptoms of phthisis. From time to time, and more frequently of late, she had been subject to attacks of urgent dyspnoea, attended with pain, such as that supposed to characterise angina pectoris.

She was labouring under one of these when she was brought to the hospital. She held her hand to her heart region, was unable to lie down, and her breathing was entirely carried on by the upper part of the chest (superior intercostal). A cough,

short and abrupt, was evidently affected by her inability thoroughly to expand the thorax. Her abdomen was retracted and fixed. Despite stimulants, her breathing became more and more laboured, the pain persisting, and she died seventeen hours after her admission.

The body was emaciated. The muscles were pale, but their tissue was natural. The several organs and viscera were natural, except the following:—The right heart was dilated, and both it and the left heart were affected with fatty degeneration. The bloodvessels were atheromatous. The diaphragm presented the following appearance:—Its muscular tissue was pale and wasted; it was flabby, and easily torn. On looking closely at it, a number of minute yellow specks were seen scattered throughout its tissue: for the most part arranged one after the other in little lines, which took the course of the muscular bundles. Most noticeable around the central tendon, this change became less marked towards the crura; and in the latter appeared to have made but little progress. The diaphragm was firmly adherent by the pleuræ to the lungs, which in their turn were fixed by adhesions to the remainder of the walls of the chest. It was in the portions speckled, as above described, that the fatty decay had made the greatest advance. The lungs were filled with tubercles in various stages.

CASE 6.—Another case was that of a charwoman, aged twenty-two, but old beyond her years; the mother of several children; a broken-down, ill-nourished creature. She was brought to the hospital on the morning of Jan. 12th, 1858, and was admitted into Dr. Burrows's ward. It was ascertained that she had suffered from a cough for three weeks; that she often broke out into a cold perspiration, with faintness, and urgent dyspnoea. She was, when first seen, in a dying state. Respiration hurried, shallow, and entirely thoracic; abdomen tender, retracted, and rigid; pulse 120, very feeble. Despite stimulants, she died four hours after her admission, complaining almost to the last of the great faintness, and of the embarrassed breathing.

On making a post-mortem examination, it was found that she had extensive aortic disease, with a fatty and dilated heart (foramen ovale patent), and a large congested liver. The diaphragm, like the heart, was the seat of fatty degeneration. This change, whilst it involved all that portion connected with the ribs and the tendinous arches, was absent from the crura.

That the muscle which works next hardest to the heart, and under conditions somewhat resembling its mode of action, should be liable to suffer from a similar degeneration of tissue, is sufficiently evident; indeed all which has been proved and argued for the one may with equal justice be said of the other. The unlikeness of the mode of death occurring chiefly from fatty diaphragm as compared with that from a fatty heart lies, as it seems to me, in the distress and difficulty of breathing which, from the onset, attends the former—a condition noticed only secondarily, if at all, with the latter.

But degeneration of the two muscles is apparently co-existent, and it is only a question which shall break down the sooner. When decay has spread through their tissues, a little extra strain on the breathing apparatus or on the circulation, may at any moment determine the issue. These are points, however, for the consideration of the physician rather than of the surgeon. My present object is simply to draw attention to the existence of this muscular degeneration, and to suggest that it may be in itself a cause of death, or may prove, with other diseases, a serious complication.

Queen Anne-street, Cavendish-square, Jan. 1867.

A CASE OF FUNGUS HÆMATODES, IN A PATIENT SIX YEARS OF AGE, DEVELOPED IN A SUBCUTANEOUS LYMPHATIC GLAND ON THE MARGIN OF THE TEMPORAL FASCIA.

By W. BIRD HERAPATH, M.D. LOND., F.R.S.*

MRS. B.— consulted me, in the latter part of 1864, respecting her little daughter, C. J. B., who had had spina bifida in the lumbar region—of course congenitally. The little patient was at that time about four years old. The

* Read at the Bath meeting of the Bath and Bristol Branch of the British Medical Association, on Dec. 13th, 1866.

tumour was small, about the size of a walnut; it had not altered in size. The skin was thin and transparent, and the tumour had not interfered with her movements from the first, as she was able to walk at sixteen months old. It had burst from accidental violence about six months before I saw it, and at that time hæmorrhage took place, but ever since it has wept or discharged a watery fluid; at times this would dry up, then the sac would refill and discharge itself again. Her mother said that the tumour affected her head, and was often the cause of alarming symptoms; these were severe headaches. At these times she shunned the light and disliked noise; but there were no convulsions at any time. She could not even go to church because of the noise of the organ; yet she liked vocal music, and instrumental also, if not too loud. She was generally sprightly and lively in character. Slight internal strabismus existed at the time of her first visit to me, and had only been noticed from the date of the accident. These head symptoms were generally noticed at those periods when the lumbar cyst was refilling and exerting pressure on the cerebro-spinal fluid, but no paralysis of either extremity had ever been observed. I was inclined to attribute these cerebral symptoms to the usual irregularities in diet attendant upon the indulgences always allowed to children with chronic and apparently incurable disease, and treated her accordingly for that paroxysm which had induced the parents to send for me.

In the month of August, 1865, this little patient was again brought to me; and then it was on account of a small glandular swelling which had shown itself at the anterior and superior margin of the left temporal muscle. It was as large as an almond when decorticated, but destitute of pain, and situated above the temporal fascia. It was *distinctly movable*. At that time I was disposed to think it of a strumous character, though, if so, the position was unusual, and no other glandular tumours existed.

I saw her again on the 18th of September, 1865; and in one month it had increased in size, and was then as large as a walnut, but showed no appearance of softening or suppuration. But at that time I strongly recommended its extirpation, as its appearance had become somewhat doubtful, to say the least of it.

Oct. 4th, 1865, was the last time I saw the child for some months; the parents refusing to have any surgical operation performed, although the tumour was nearly double the size when last seen.

On the 25th of April, 1866, she was again brought to me; and now it had grown to the size of a cricket-ball. And from this date no further doubt existed as to its nature. It was decidedly of a malignant character, and of course medullary sarcoma or fungus hæmatodes. The spina bifida was observed to decline and decrease in proportion as this cephalic tumour increased; and at length it disappeared altogether and healed up, so that at the time of her death the scar was very little apparent.

In May last her mother caused a photograph to be taken of this little patient; but although the tumour at that time must have been much larger than a cricket-ball, yet, as the carte de visite was taken from the healthy side, nothing whatever of the disease is perceptible from this view. But in July it had grown to be larger than her own head, and was very irregular in form, distinctly nodulated, and the surface traversed by large venous trunks. It had attained its maximum at that time, and it occasioned considerable distortion of the left eye, by elongating the lids, and pulling them outwards.

On July 31st we were hastily summoned, as alarming hæmorrhage had taken place from a large vessel having given way; yet the ulceration was only superficial, and much hidden by the hair. During the first two weeks in August ulceration progressed extensively; a large ulcer opened upon the centre of the fungoid mass, and several of the secondary tubera also made deep and ragged ulcers with everted edges; whilst early in August she became paralysed on the right side, losing the use of both the arm and leg. During the last month frequent loss of blood occurred, blanching her body, and reducing her strength exceedingly. She never complained of much tenderness or pain, but an intense itching of the surface annoyed her. Occasionally she would scream out as if a sudden pain had seized her, but these attacks never lasted long; they were probably shooting or lancing pains, but she was too young to describe them. During the whole of the last two months she was wholly confined to the bed, as the weight of the tumour (about 8 or 9 lbs.) prevented any other position than the recumbent one. Her power of deglutition failing her, as well as her speech, she became incapable of taking any nourishment, and during the greater part of last month she took

nothing daily but a few tablespoonfuls of beer, which she had always relished better than anything else, and she gradually sank from exhaustion and anæmia, death closing the scene very tranquilly, without convulsions or loss of consciousness, on August 31st, 1866. She was at the time of her death six years and two months old, and, for her age, tall and well formed, being four feet two inches long when measured for her coffin.

The treatment consisted throughout in the administration of sedatives occasionally to promote sleep and quiet, and the application of styptics to control the hæmorrhage. The tincture of sesquichloride of iron, and slight pressure with dossils of lint, succeeded effectually. Occasional tonics, with quinine, were at first given, but latterly no medicine was employed. The offensive odour was controlled by the use of a dilute solution of hypochloride of soda as a local disinfectant, and when sufficient was employed it was perfectly effectual.

The friends were very averse to any post-mortem examination being made, and only with difficulty were prevailed upon to have even a photograph taken of the case. This is a very successful effort, and for which I am indebted to Mr. Barton, of this city. It gives a very clear and faithful view of the figure and the extensive ulceration at the time of her decease. It would have been very interesting to have examined the perforation of the skull, and to have traced the existence of secondary deposits in the viscera; but if any such deposits existed they must have been formed very latently, as no symptoms of their presence existed throughout the case. I apprehend there can be no doubt that the primary seat of the malignant disease was in a subcutaneous lymphatic gland, situated above the temporal fascia, and at the anterior border of the temporal muscle; and the case is further remarkable for the extreme youth of the patient in whom this extraordinary growth was developed.

Bristol, Dec. 1866.

ON THE ADAPTATION OF ARTIFICIAL PALATES.

By GEORGE PARKINSON, M.R.C.S.,
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HAVING formerly been house-surgeon at King's College Hospital, under Sir William Fergusson, my attention, soon after taking up the dental specialty, was directed to those cases of fissure of the palate, whether congenital or morbid, which were not amenable to surgical treatment, and having now had the experience of forty-eight cases treated on the principle hereafter described, I feel justified in introducing the apparatus to the medical profession. It sometimes happens in cases of congenital fissure of the palate, that the margins of the velum are so far apart as to preclude the possibility of uniting them by surgical operation, and the case then comes fairly into the hands of the dental surgeon.

There are three evils susceptible of remedy by artificial palates: first, defective enunciation; second, the escape of solids and fluids through the nasal passages; third, difficulty of swallowing.

As congenital cases require a special arrangement of the apparatus to be employed, it will be better to consider these cases first, and afterwards the more simple treatment required, where the palate or portions of it have been destroyed by sloughing of the soft and necrosis of the hard tissues. It is not my intention to occupy time and space in describing the various mechanical arrangements which have from time to time been introduced, but merely to give a sketch of the method of treatment I have found the most simple and successful, at the same time acknowledging I was originally indebted to a paper by Mr. Stearns, which appeared in THE LANCET of the year 1845, for valuable suggestions in the treatment of similar cases.

In a case of congenital fissure of the palate extending through the hard tissues and alveolar ridge, after having taken a correct model of the parts in wax or plaster of Paris, I commence by fitting a thin plate of gold over the vault of the palate, as far back as the posterior margin of the palate bone would have extended had the bony arch been perfect. To the posterior margin of this plate, by means of a hinge, is attached a velum, constructed of hard, well-polished, vulcanised india-rubber, formed in such a manner as to fit the palatine surface of the remnants of the soft palate and allow them to glide over it in