

the mastoid process without any involvement of the middle ear. There are eight centres of ossification in the temporal bone, one for the squamous, two for the styloid process, one for the tympanic ring, and four for the petro-mastoid portion. These centres have nearly coalesced about the fifth month of intra-uterine life, but during the remainder of foetal life the squamous is easily separable by maceration from the petro-mastoid. During the first few years of life also the petro-mastoid and the masto-squamosal sutures are more vascular than the rest of the bone and separation by maceration is occasionally possible. On the outside of the skull of a newly born infant the masto-squamosal line of suture can easily be detected. It commences at the point of junction of the mastoid, parietal, and squamous bones, and then passes downwards and forwards, making with the posterior root of the zygoma an angle of about 45 degrees, until it reaches a point in a line horizontally with the centre of the outlet of the bony meatus—that is, the apex of the supra-meatal triangle. From that point the line drops vertically downwards to the lower surface of the bone. On the inner surface of the infant skull the line of the petro-squamosal suture can be distinctly seen running along the junction of the petrous with the squamous. When a foetal temporal bone is macerated and the squamous is separated from the petro-mastoid it will be seen that the outer wall of the mastoid antrum is formed by the descending plate of the squamous, and that nearly one-third of the masto-squamosal suture line is above, and posterior to, the mastoid antrum. Thus, the line of the masto-squamosal suture can be divided into two parts: (1) an upper part which is above and behind the antrum; and (2) a lower, most of which runs along the posterior border of the descending plate of the squamous—i.e., along the posterior border of the outer wall of the antrum. The point on which stress is to be laid is this—viz., that if a spot of caries occurs along the line in portion (1) it is possible for a perforation exposing the dura mater of the middle fossa to occur without the mastoid antrum being opened up. Two cases which have been under my care illustrate the importance of this point.

CASE 2.—The patient, a boy, aged six years, was admitted into the Birmingham Children's Hospital on May 27th, 1900, complaining of the presence of a swelling over the right mastoid process. In this position was a soft, fluctuating swelling with the skin just becoming red. The history given was that the patient had never had ear-ache or any discharge from the ear and that the swelling had been forming for the past three to four weeks. The membrana tympani on examination appeared quite normal, there being no bulging, perforation, or visible scar. There was no displacement of the auricle. The patient's temperature was normal and he complained of no pain beyond a little tenderness on pressure over the swelling. On June 1st the swelling was incised and proved to contain pus. In a position corresponding to the extreme upper end of the masto-squamosal line was a patch of carious bone of about the size of a split pea. This was scraped away and a sinus was found to perforate through so as to expose the dura mater of the middle cranial fossa. The remainder of the surface of the mastoid and of the descending plate of the squamous appeared quite healthy and so the antrum was not opened. The wound was packed and healed satisfactorily, there never being at any time a discharge from the auditory meatus.

CASE 3.—The patient, a girl, aged five years, was admitted into the Children's Hospital, Birmingham, on Nov. 13th, 1900, complaining of a swelling over the right mastoid which had been forming for three weeks. There had never been any otorrhoea and there had been no complaints of pain. The membrana tympani appeared to be normal. During the next three days the patient's temperature varied from 98.6° to 99.5° F. There was no displacement of the auricle. On Nov. 16th the abscess was opened. At the upper border of the mastoid, just in the position of the upper end of the line of the masto-squamosal suture, was a small patch of carious bone. This was scraped and was found to penetrate almost, but not quite, through to the dura mater. The remainder of the mastoid process appeared to be perfectly healthy. The wound was packed and rapidly filled up.

Here, then, were two cases of what may be called "quiet mastoid abscess," in which the abscess was clearly due to the formation of a patch of caries, presumably tuberculous, in the exact position of the upper part of the masto-squamosal line; in that part of the line, in other words, which has on its deep surface the dura mater of the middle

fossa and which is altogether above and behind the position of the mastoid antrum. Had this patch of caries occurred a little lower down in the course of the masto-squamosal line, then, when perforation of the bone took place, the antrum would have been opened up and it would then have been impossible to say whether the disease began as a tuberculous otitis media, which led to secondary caries of the mastoid process, or as a primary caries of the mastoid process which had perforated through the masto-squamosal line into the antrum. In fact, Cases 2 and 3 appear to give the clue to the morbid anatomy of this class of mastoid abscess.

From a study of these cases it seems justifiable to draw the following conclusions: 1. That in young children a form of mastoid abscess not uncommonly occurs (in 10 out of 61 cases) in which there is no history of otorrhoea and in which the membrana tympani appears to be normal. 2. That in these cases the abscess is of slow and painless formation, the patient's temperature not being raised and the ear not being displaced. 3. That in these cases the abscess is due to primary caries of the mastoid, the patch of caries occurring somewhere along the line of the masto-squamosal suture, this being in a young child the softest and most vascular portion of the bone. 4. That if the patch of caries forms in the upper part of the masto-squamosal line and if perforation of the bone occurs the dura mater of the middle cranial fossa is exposed and the mastoid antrum is not opened up. 5. That if the patch of caries occurs in the middle or lower part of the masto-squamosal line the antrum is quickly opened up and then the caries spreads rapidly through the mastoid.

When the patch of caries is found to be situated in the upper part of the masto-squamosal line and the rest of the mastoid is healthy, then it can be taken that the abscess was caused by a primary caries of the mastoid. When, however, the patch of caries is found to be situated in the middle or lower part of the masto-squamosal line—i.e., in that part which runs along the posterior boundary of the supra-meatal triangle—it must remain uncertain whether the abscess is due to primary caries of the mastoid or to tuberculous inflammation of the middle-ear. But, reasoning by analogy, it seems at all events probable that mastoid abscesses of this class have a common origin—viz., in caries of the mastoid process; that the patch of caries occurs nearly always along the line of the masto-squamosal suture and that whether the mastoid antrum is opened up or not depends upon the exact position in the line of the suture at which the caries occurs.

Birmingham.

OBSCURE CEREBRAL MANIFESTATIONS OF TUBERCULOSIS.

NOTES ON THREE CASES.

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THERE are few diseases which do not appreciably upset the intricate mechanism of the nervous system, but it is surprising in how few the disturbance is betrayed by prominent symptoms or remains a permanent one. It is not perhaps so surprising to observe in how many different forms the disturbance is manifested. The following notes on two cases in which the cerebral symptoms were the most urgent and in which post mortem lesions of tuberculosis were apparent will serve to show, however, the difficulty of tracing these disorders to their proper source, while Case 3 may serve to accentuate the diversity that occurs. Tuberculosis, even when confined to one organ, may appear in the most divergent forms, and when diffused the unity of the disease may not always be determined. It is similar in its secondary effects on the nervous system. So often has the unreasoning hopefulness accompanied the progress of the lung affection that the "spes phthisica" has come to be regarded as characteristic. In phthisical insanity, again, the variableness of mind, the unsocialness, or irritability with suspicion, even at times monomania, have all appeared and been regarded as symptomatic. Indeed, the insanity has been stated to begin usually before any marked symptoms of pulmonary tuberculosis appear,¹ and this may account for

¹ Clouston: Mental Diseases.

the assertion that it has not been shown that more than an average number of phthisical patients become insane (Hector Mackenzie) and may be very suggestive in the consideration of Case 1. Thus much for the lungs.

Disease of the adrenal glands is usually accompanied by nervous symptoms, the chief mental ones being apathy and depression,² but the mind remains clear unless coma or delirium supervenes.³ Other symptoms, such as prostration after exertion and disinclination for exercise, may be common to pulmonary tuberculosis and Addison's disease, where both exist in the same individual. In the following case the existence of suprarenal disease was undetected during the life of the patient, the severity of the symptoms of the lung affection completely overshadowing any sign which might have suggested an additional focus of disease.

CASE 1.—The patient, a male, was first admitted on Oct. 8th, 1898. His family and personal history was negative as to pulmonary tuberculosis and alcohol, but a maternal uncle had died in an asylum. He said that he had some complaint which prevented him from working and thought it was due to someone "illwishing" him. He had attempted suicide. Physical examination revealed no evidence of disease save that the first sound of the heart was slightly rough in character and the pulse was 90. He was dejected and depressed but answered all questions readily and rationally, had a good memory, and realised his position. He was discharged recovered on June 6th, 1899. He was readmitted on Jan. 30th, 1902. His certificate again showed that he asserted himself to be suffering from an internal and incurable disease and had threatened to do away with himself. The pulse on this occasion was found to be 89 and of high tension; the heart was as before, but no trace of organic disease could be discovered. He exhibited good health and his melancholic symptoms seemed to be disappearing until the beginning of 1905 when he became very distressed, saying that he could not live. Examination of his chest in January showed the presence of consolidation in the apex of the left lung, and in February there were all the physical signs of a cavity. He began to lose weight, became anæmic and emaciated, had gastric irritability, and an uneven temperature. His pulse became feeble and he had copious night sweats and died on June 16th, 1905. He was never observed to cough during the whole course of his illness. His age at death was 52 years.

The necropsy revealed a large cavity in the left lung, which was full of pus and over two inches in diameter. Chronic pleuritic adhesions were present and the smaller bronchial tubes were choked with purulent material, most of the lung being in a state of consolidation. The weight of the organ was 60½ ounces. The heart was small, the muscle being soft, but the valves were competent. The spleen was soft and friable and the kidneys were apparently normal. The suprarenal gland of the right side was firmly attached to the kidney; that of the left was separate from its kidney and surrounded by a separate envelopment. Both suprarenals were enlarged, each weighing two and a quarter ounces, and in parts were fibrous and cartilaginous in feeling to the finger and gritty to the knife, other parts being studded with cysts which exuded a colloid material and cavities full of thick caseous pus, the left—i.e., on the same side as the affected lung—showing an abscess one inch in diameter. The liver was waxy. The brain was cedematous with distended ventricles. There was no anasarca, no pigmentation of the skin, no leucoderma, or deepening of the natural pigmentation in the usual sites. The hair showed no bronzing. There was a patchy, purple discolouration of the mucous membrane of the upper lip and of the gum of the upper jaw at its junction with the teeth, but no deposit of pigment on the tongue, the palate, or the lower lip.

There are several features of interest that rather tempt speculation as to the connexion between the mental state and the physical conditions. Was the lung affection previous to the suprarenal or *vice versa*? Was the mental disorder secondary to the suprarenal? In the order of appearance by outward symptoms the melancholia was succeeded by the pulmonary tuberculosis and the latter had cut short the life of the patient and presumably the outward exhibition of the disease of the adrenal bodies. But these glands could hardly have been more diseased than they were and the patient's lungs were healthy till within a year of his death.

His melancholia was a recurrence of a previous attack and was certainly previous to the pulmonary tuberculosis. Heredity is important in phthisical insanity and in cases of the latter an inherited tendency to insanity can usually be traced (Clouston). Is it that though the insanity appears before the pulmonary tuberculosis there is already a focus of the disease in the patient, though undiscoverable? Or can the depression of Addison's disease in a predisposed patient go on to insanity? One case with melancholia has been observed by Dr. Rutherford Macphail. While again drawing attention to the patient's main "delusion" I leave the discussion to abler judges.

CASE 2.—The patient, a male, aged 50 years, was admitted on March 31st, 1904. He was confused and semi-stuporose. He had been subject to epileptic fits for "some months." The pupils were contracted. The gait was shuffling and Romberg's sign was present. There was slight albuminuria. The breath sounds were normal. He remained dull and apathetic and suffered from nocturnal epileptic fits, crying out before them and falling or rolling over to the left. He was irritable occasionally. He was able to do outdoor work, his fits averaging 16 per month. In October, 1905, he had a succession of fits which left him feeble and stuporose. Bromide of sodium in 30-grain doses reduced the fits considerably but he died after several severe fits on Dec. 16th, 1905. He vomited once on the 15th.

Post mortem caseous nodules were found in both apices and in the bronchial glands. Miliary tubercles were found in the liver. No other organ with the exception of the brain was found to be affected to the naked eye. In the latter a curious condition presented itself. On running the hand over the cortex hard patches were felt where the pia mater was firmly attached. In the grey matter under, the cortex was firm and tough and fibrous in feeling. These scar-like nodules were less than pea-sized and in the cerebrum of each side but not in the frontal lobes, six such spots being on the right side and four on the left. In addition, in the cortex of the right occipital lobe a cyst with tough walls and containing caseous granules was found. The cyst was a quarter of an inch in diameter. None of these cortical nodules was encapsuled or easy to separate from the surrounding grey matter. In both lateral ventricles the ependyma was granular and small calcareous nodules of the size of a hemp-seed were found. In the left lobe of the cerebellum the white matter was firm and difficult to tear and the grey matter was almost non-existent. The white matter was contracted and gritty on section. Here one finds very suggestive signs. Slow-growing tumours are not rarely found in the brains of epileptic demented (Mott) and these during life may give rise to symptoms very like idiopathic epilepsy (Oppenheim, Mott). Small fibrous areas are occasionally found post mortem (Shennan) as if a tuberculous lesion had become quiescent. And tumours of the cerebellum (Mott) and of the parieto-occipital region (Risien Russell) may grow to a considerable size without evidencing focal symptoms. In cerebellar atrophy (Russell) epileptic convulsions are not rare and if a tuberculous seat in the cerebral convolutions contract and dry up it may cause epileptic attacks (Cotterill). That the last was the cause of the epilepsy and that affection of the cerebellum accounted for the fall to the left and the disturbance of gait is quite possible.

CASE 3.—A male, aged 25 years, was admitted on March 14th, 1905, suffering from acute delirious mania. There was a family history of pulmonary tuberculosis and the patient had been under treatment for the same disease in a sanatorium and been discharged cured. It was impossible to examine him properly but a cavity was found in the left lung. He remained violent and delirious till his death on March 17th. Post-mortem examination confirmed the diagnosis of a cavity, most of the remaining part of the lung being consolidated.

Here, on the basis that a toxæmia is the cause of this form of insanity, one must presume that mixed infection had occurred. Many are the causes assigned to this disease, quite as many, in fact, as to all diseases not known to be due to a specific organism, and pulmonary tuberculosis is in this array.⁴ And whatever the cause may have been in this case, it is at least certain that the delirious mania occurred during the course of the disease.

In reviewing the records of these three cases I am aware

² Stockman: Text-book of Medicine, edited by Gibson.

³ Rolleston: Encyclopædia Medica.

⁴ Blandford: Insanity and its Treatment.

of the objections that occur to classifying them as manifestations of the same disorder. They have only one feature in common—that tuberculous lesions existed and were found post mortem. In Case 2 only were those exhibited in the brain and there the symptoms in reference to it were physical rather than psychical. Phthisical insanity is a distinct disease and embraces none of these cases, which in relation to mental symptoms were classified as melancholia, epileptic insanity, and delirious mania respectively. But it is easy to push the *post hoc, ergo propter hoc*, too far, and it is a known fact that more insane than sane patients die from tuberculosis. Nevertheless, Case 3 had pulmonary tuberculosis before he became insane; Case 2 had tuberculous lesions before he developed the epilepsy which brought the mental symptoms in its train; and Case 1 very probably had Addison's disease while in a state of sanity. They are all illustrative of the benefit derived from post-mortem examination, even where the diagnosis seems perfectly clear. Without such procedure Cases 1 and 2 would have been put down as examples of acute pneumonic phthisis and of ordinary major epilepsy. They each present interesting and debatable features and apart from their suggestiveness they seem, from their intrinsic interest alone, worthy of record.

Exminster.

A SUCCESSFUL CASE OF EPIPLOPEXY FOR CIRRHOSIS OF THE LIVER WITH ASCITES.

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At the present time, when surgical procedures for the relief of some of the symptoms of hepatic cirrhosis are being so much discussed, the following case is interesting and its publication may be of value as an item among the many observations necessary to a thorough appreciation of the value of such operations, their scope, and their limitations.

The patient, a man, aged 40 years, was successfully treated by Dr. F. V. Adams in the later months of 1904 for nasal obstruction resulting from enlargement of the turbinates. One day in January, 1905, while feeling perfectly well and walking in the street, he was seized with sickness and vomited blood, after which he fainted for a second or so and fell on his knees. He went home to bed and by the next day seemed all right again and was able to go out. After this he remained to all appearance perfectly healthy until July 10th, 1905, when he was on holiday in Ireland. After a cup of tea at breakfast that morning he felt sick, vomited blood, and fainted slightly, exactly as on the previous occasion. He was kept in bed for a week this time and was fed by the rectum. He has had no other attack of this kind and he can suggest no cause for either of the two attacks. His previous health had always been excellent, apart from slight bilious attacks which he attributed to confinement and want of exercise. He had no cough. He could take any kind of food, his bowels were regular, and he did not suffer from piles. He lost about 10 pounds in weight during the week of rectal feeding but appeared to regain this afterwards. According to his own statement, which is corroborated by his friends, he was always moderate in the use of alcohol. About a bottle of beer at supper was the extent of his indulgence. He never took beer in the day-time and he took no spirits at any time. After the first hæmorrhage, which has been described, the stools were black for a time but there were no stomach symptoms. After the second attack the stools were again black for a time, but, in addition, he began to suffer from abdominal pain when he had been in bed for a few days; and, after a few more days, when he began to take food by the mouth he became troubled with distension. The pain, though constantly present, was never very severe but on the whole it tended to get worse as time went on. It did not seem to be related to the taking of food but it was aggravated by pressure

and it kept him from lying on either side. At the same time his general health remained good.

When the patient was seen on July 27th it was noted that the abdomen was greatly distended. The signs pointed to ascites with flatulent distension of the stomach. There was tenderness at the right lower costal margin, just to the right of the xiphoid, but nothing abnormal was palpated there. The liver could not be felt. Examination of the rectum gave negative results. The lymph glands in the right axilla were somewhat large but those in the other accessible parts were normal. The heart and the urine were normal. Some crackles were heard at the posterior bases but otherwise the lungs were normal. On August 4th, in St. Elizabeth's Nursing Home, the abdomen was tapped and after the ascitic fluid was evacuated the stomach was distended with carbon dioxide, with the result that no great dilatation was found. There was still tenderness in the right hypochondrium.

Operation.—On the 8th the abdomen was opened in the middle line above the umbilicus; a quantity of ascitic fluid was evacuated and there was found cirrhosis of the liver and also an adhesion of the pyloric end of the stomach to the under surface of the liver. No tumour was found in the liver, portal fissure, pancreas, hepatic or splenic flexure of the colon, or pylorus. The stomach was healthy except for some dilatation of the veins. The omentum was then spread out and sutured by means of a fine, continuous, catgut suture to the peritoneal surface of the anterior abdominal wall, first on one side of the incision and then on the other side. There were three rows of sutures on each side. In closing the abdominal wall the first layer of sutures was passed through the transversalis fascia and peritoneum and the omentum was picked up with the needle as it passed from one side of the wound to the other, so that the omentum was attached by seven rows of suture. The rest of the abdominal wound was closed in two layers. The after-history of the patient was uneventful, the wound being healed when it was dressed a week after the operation. He went home on the 23rd—the fifteenth day—and a week later he was allowed to go to the coast for change of air. After a month's holiday he resumed work and he has continued at his occupation without interruption until the present time.

At this date,¹ eight months after the operation, inspection of the abdomen shows a dilated vein, the superficial epigastric, extending from near the umbilical end of the cicatrix to the left groin. No other venous dilatations are observed and the abdominal wall is otherwise normal. No ascitic fluid can be detected in the abdomen and no abnormalities are observed on palpation or percussion.

One of us (T. K. M.) examined the patient on April 2nd and made the following note of his condition: "Mr. — called this afternoon and expressed himself as perfectly well, being indeed brighter and altogether better than he has been for the last 15 years. He has been a total abstainer since he was in St. Elizabeth's Home. The only symptom he has had since the operation was melæna. This happened once, about two months ago, and if he had not noticed the stools black he would not have known that anything had happened. He attributed it to running for a train or to a twist when he was scrubbing himself in the bath. The liver is reduced in size and there is no tenderness in the right hypochondrium. The spleen is sufficiently enlarged to be easily felt on inspiration. The heart is normal. I note the enlarged superficial epigastric vein running down to the left groin from the scar, near its lower end. There is a good current of blood in it flowing downwards."

The case is one of cirrhosis of the liver characterised by well-marked ascites, by hæmatemesis, and latterly by tenderness of the right hypochondrium, as well as by pain in that region. The condition of the liver was demonstrated by inspection. There are an absence of any serious alcoholic history and no suspicion of syphilis or of malarial or other toxic infection, and there are no tumours in the neighbourhood of the liver, spleen, pancreas, or colon. The operation was simply that of suture of the omentum to the anterior parietes, without rubbing or scraping of the surface of the liver, of the spleen, or of the parietal peritoneum; and, after a lapse of eight months, and with the solitary exception of a trivial hæmorrhage, evidenced only by melæna, the patient reported himself as quite well, and there is satisfactory evidence of a local increase in the venous circulation of the abdominal wall originating at the site of operation.

¹ The patient was shown at a meeting of the Glasgow Medico-Chirurgical Society on April 6th, 1906.