more so to vie with each other in the common cause of humanity. Then, perhaps, they may come to acknowledge the truth of the saying of Cicero in his oration *Pro Ligario*, "Neque enim ullâ aliâ re homines propius ad Deos accedunt, quam salutem hominibus dando".

EPIDEMIC CEREBRO-SPINAL MENINGITIS: REPORT OF *POST MORTEM* EXAMINATION IN THREE CASES OF THIS DISEASE.

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THE two *post mortem* examinations which I here record represent the sole occasions on which I have had an opportunity of making a dissection after death from epidemic cerebro-spinal meningitis; they will, therefore, be conveniently detailed apart from the clinical history of other cases which have lately fallen under my care at the Birmingham General Hospital or in private practice, leaving an opportunity of inserting hereafter the clinical details of these cases.

The two examinations which follow afford confirmation of the truth of Mr. Simon's observations on this subject, that "for practical purposes, the state of the covering membranes of the nervous centres may be regarded as a mere index of changes more or less distinctive which these centres, in their own intimate composition, have at the same time undergone; and hence it is that the essential phenomena of the disease during life consist in disturbances, more or less grave, of the functions of those all-important organs" (Reynolds's *System of Medicine*, 2nd ed., p. 513).

CASE I occurred in the person of a girl, aged 14, who was admitted on June 22nd, on the eighteenth day of the disease, and died on July 16th. I shall merely record here that the origin of the disease was acute, and dated from June 5th; it was referred to the child having fallen into some water and remained in her wet clothes. Certainly, she felt ill the same evening, and shivered and vomited the following day; the vomiting ceased in three days. The headache was the prominent symptom; it was seated in the occipital and frontal regions. The intellect was clear from the second or third day. The symptoms assumed somewhat of an intermittent type, but, during the last ten days of life, settled into a state of apathy, with much depression of vital power, ending in torpor and death by asthenia. The urine was very phosphatic. The temperature manifested a tendency to morning remissions and evening exacerbations, very decided in two quarter periods, very imperfectly marked in the other two quarters. The maximum temperature never exceeded 102 deg.

POST MORTEM EXAMINATION.—No external marks on the body; a loose fibrinous riband in the longitudinal sinus; fluid blood in this and in the lateral sinuses; very marked injection of the minute vessels over the entire surface of the brain; but the arachnoid and pia mater retained their normal transparency, and could be stripped off the brain with ordinary facility. To this statement there are the following exceptions.—Just at the anterior extremity of the superior vermiciform process of the cerebellum was a collection of not very recent yellow lymph in the pia mater, over a space not half an inch square; a similar deposit at the base of the cerebrum covered the chiasma, and a few broad streaks existed in the membrane at each Sylvian fissure, running for a short distance along the vessels. The lateral ventricles had plainly been subjected to distension; at the present time, they contained a moderate quantity of opaque highly albuminous fluid, in which Dr. Saundby found blood-corpuscles and small round granular nuclear cells. Their ependyma was sufficiently thickened to float in water as a consistent semi-transparent membrane. The surface of all the nervous masses in the ventricles showed minute branching vessels filled with blood, and the surface-tissue was rather soft. The blood-points throughout the white tissue of the brain were large and numerous, and on the surface of the convolutions the perivascular spaces were very distinct. The grey substance throughout the cerebrum was deeply tinged with pink; this pink colour, very apparent in the convolutions, was especially striking in the large ganglia. The ganglion-cells in the corpora striata were well formed. Dr. Saundby examined some of the minute vessels of the grey matter of the brain, and reported them simply loaded with blood. In all other respects, the brain-substance was healthy. The spinal cord presented nothing abnormal, excepting injection of the pia mater, which, though considerable, was less marked than in the brain. There was the same strongly marked pink colour of the grey matter as already noticed on the cerebrum. The tissue of the cord was of

normal tenacity; the rest of the nerves were healthy. The muscles of the back presented a granular appearance in the sarcolemma. There were fibrinous clots in the heart. The spleen was small but healthy. Nothing else was presented worthy of note. The blood was not unusually fluid.

CASE II.—The patient was 24 years of age. He was admitted for pleurisy. He had a very cachectic appearance, and gave a confused history of some foregoing illness; but neither in his description nor in the symptoms he presented was there anything to lead to the supposition of peculiarity in his case. He was recovering from his pleurisy, when, with no apparent reason, he was attacked with purposeless vomiting and retention of urine; the vomiting continued for three days and then ceased; but the man became rapidly apathetic, then torpid, and died asthenic on the sixth day of this last illness. The retention of urine passed into involuntary evacuation. Inquiry from the friends elicited a manifest history of cerebro-spinal meningitis, and ample confirmation was afterwards obtained from Mr. Bradley, who had attended the patient. Mr. Bradley described to me the symptoms of the simple form of the disease. The attack commenced on the 7th of last February; a brief relapse occurred; but convalescence was sufficiently established to permit of Mr. Bradley taking his leave on March 23rd. Of the subsequent attack of pleurisy, Mr. Bradley knew nothing; the patient came into the hospital on May 13th and died on the 29th.

POST MORTEM EXAMINATION.—The arachnoid and pia mater had undergone remarkable thickening over a limited space at the base of the brain, presenting a tough semitransparent membranous web covering the chiasma, stretching across the interpeduncular space, enveloping the pons Varolii and medulla oblongata and enclosing the cerebral nerves at their origin. The adhesion of the membrane to the pons and medulla was so close that it was impossible to separate it without damaging the nervous tissue. It covered the fourth ventricle and thence extended down the cord. The thickening extended on the under surface of each cerebellar lobe for about an inch in breadth, thence gradually ceasing; it appeared also as a broad band running across the anterior edge of the cerebellum, above the mesencephalon. A very scanty deposit of firm yellow lymph existed in the pia mater, upon the chiasma and beneath the parietal layer of the arachnoid upon the basilar process. In all other parts, the membranes of the brain were quite healthy. The ependyma of the lateral ventricles was much thickened, and could be raised as a semitransparent membrane of considerable tenacity. The lateral ventricles were largely dilated, but no unusual quantity of fluid was found in them at the time of the *post mortem* examination. The tissue enclosing the ventricles had an unusually fibrous appearance, as though it had been subjected to stretching. No abnormal appearance existed in the brain, except that its tissue was generally softer than usual, but not in any one part particularly. The pia mater and visceral arachnoid of the cord were much thickened through the entire length, closely encircling the cord with a dense envelope, which bound down the roots of the nerves and adhered to the cord itself along the posterior surface only. The apex of one lung was broken up, and there were remains of fluid in the pleural cavity. No other morbid change existed in the body.

CASE III.—In a third *post mortem* examination on a patient of my colleague (Dr. Fletcher), who died after an illness of six weeks, the principal appearances consisted in the presence of a soft flocculent lymph over a very limited space extending from the chiasma to the posterior edge of the pons Varolii; of a small patch of the same material over the root of the right suboccipital nerve; and of two or three similar patches within the subarachnoid space of the upper dorsal region of the spinal cord. The cerebral arachnoid was more resistant than usual, and was opaque where stretching across the sulci: in the upper dorsal region the spinal arachnoid was also opaque. The ventricles were distended. The condition of the cerebral tissue closely resembled that in my first case. One kidney was excessively atrophied, thereby accounting for the presence of a considerable amount of albumen in the urine during life.

RECURRENT EPISTAXIS FROM MALARIAL INFLUENCE.

By E. M. SINCLAIR, M.D., Surgeon-Major Royal Artillery.

WITH reference to the paper by Surgeon-Major J. H. Porter of Netley, on Intermittent Hæmorrhage from Malarial Influence, read before the Royal Medical and Chirurgical Society in February last, the following case of intermittent epistaxis from the same cause may prove of interest.

G. —, aged 25, in the service two years, by trade a shoemaker, an intemperate man and not of good character, arrived at Kamptee (from England) in December 1874. In 1875, he had four admissions into hospital for accidental injuries, and one in October under the head of simple continued fever, treated by quinine.

In January of the present year, he became an inmate of the military jail at Secunderabad, and was, after five months' imprisonment, admitted to hospital at that station in a debilitated state, complaining of muscular pains in the trunk and limbs, anorexia, and want of sleep. The temperature was normal; bowels very sluggish; tongue rather foul; urine clear and normal, of specific gravity 1016. He was ordered a mixture of citrate of iron and quinine; and twenty-five grains of hydrate of chloral at bedtime.

On the sixth day after admission, he complained of tingling sensation in the legs, with pain in the back and abdomen, through the thin walls of which the pulsations of the abdominal aorta could be felt most distinctly on very slight pressure, simulating somewhat an aneurism. There was a *bruit* of anæmia, but no tumour or enlargement of the artery, which could be distinctly traced to the bifurcation. He was ordered beef-tea, milk with lime-water, a small quantity of brandy and water occasionally, and a mixture of chlorate of potash, hydrochloric acid, and tincture of iron; also a morphia draught at night. Little or no change occurred till the twelfth day, when he was suddenly seized, about 8 A.M., with epistaxis, and lost blood to the extent of about two ounces. This was checked for the time by the application of ice, but recurred to about the same extent at 1 P.M. Temperature 100.5. On the following two days (thirteenth and fourteenth days), he had epistaxis twice daily, though to rather less extent. Morning temperature normal; evening 99.4. He was given tincture of ergot in draught, and also added to the chlorate of potash mixture; and equal parts of tincture of perchloride of iron and water were thrown up the nares with a syringe. He improved slightly. The epistaxis was less in quantity, but recurred, at irregular hours, once in the twenty-four hours until the nineteenth day, when it came on with increased severity twice, about the same hours as at first, viz., 8 A.M. and 1 P.M. These attacks were preceded by a "shivering"; but there was no hot stage, the temperature not rising above 99.4. He was now put upon quinine, fifteen grains a day, with generous diet. On the twentieth, twenty-first, and twenty-second days, there was recurrence twice daily of the epistaxis, but it gradually became less in quantity. On the twenty-third and twenty-fourth days, there was no bleeding. On the twenty-fifth day, it occurred once, in the morning, having been preceded by a longer "shivering" than usual some hours before; but, as the man had failed to call an attendant at the time, as directed, his condition was not noted. The quinine was continued for ten days longer; but he had no return of the epistaxis or "shivering", as he described the cold stage.

The question in the above case seems to be this: Was the epistaxis in any way attributable to malarial influence? This would appear to be answered in the affirmative, from its cure and prevention by the administration of quinine.

The station of Secunderabad has a red gravelly soil, and is wonderfully free from malaria, a case of true *ague originating* in the station being a rarity; while Kamptee, whence the man came and where he was previously stationed at for one year, is just the reverse, the soil being that known as "black cotton-soil", and intermittent fevers of all sorts the prevailing diseases. Had we here, as Dr. Lauder Brunton considered to have happened in Surgeon-Major Porter's case above referred to, "the poison of *ague* remaining in the portal system, and being directed into the systemic circulation", owing to the debilitated condition the man had fallen into from the five months' confinement in jail? or was the poison all the time in the systemic circulation like that of syphilis, making itself evident in the same manner when the vital powers were debilitated and brought below par?

CASE OF ACUTE TUBERCULOSIS IN A CHILD AGED FOUR MONTHS.

By THOMAS BARLOW, M.D., B.S.,

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R. W., a boy—a triplet—aged ten weeks, was brought to my out-patients' room at Great Ormond Street on June 30th. He was an eight months' child, and, like his brother and sister who were born at the same time, he had been fed almost from birth on Robb's biscuits, baked flour, etc. His sister had succumbed, and he and his brother were in an extremely marasmic condition. General atrophy, very

severe stomatitis, diarrhoea, and sore nates were the features of his case. The diarrhoea improved, but the stomatitis was very obstinate; it was accompanied by slight superficial loss of substance just within the margin of the lower lip.

On July 28th, some lumps were noticed under the chin. They were evidently slightly enlarged glands. There was no redness and no heat of the skin over them. No enlarged glands were felt elsewhere. On August 11th, the boy looked greatly pinched; the diarrhoea, which had been before then quite controlled, recommenced. The stomatitis had ceased. His voice was weak, and he had a very occasional slight cough. The spleen was felt two finger-breadths below the thoracic margin. The diagnosis at first had been atrophy from bad feeding, but the enlargement of the spleen noticed on this day, taken along with the group of other symptoms, viz., the stomatitis, which had gone on to ulceration, the sore nates (albeit not characteristic), and the weak voice, led my colleague Dr. Lees and myself to question whether, after all, it was not a case of congenital syphilis.

The child died in two days with convulsions, and our doubt was solved in the negative at the *post mortem* examination. The glands under the chin, though only very slightly enlarged, had probably undergone rapid caseation and softening. This was the case also with the glands on each side of the trachea, although there was scarcely any enlargement of them. It was so also with the mediastinal glands and with two or three glands situated in the fissure between the upper and lower lobes of the lungs of each side. In all these glands, the central portion was softened purulent stuff; in some of them, the peripheral part was slightly firmer and opaque, greenish-yellow in colour. The pharynx was natural. The larynx showed, behind the posterior extremity of the right vocal cord, a small cavity bigger than a mustard-seed, exposing part of the arytenoid cartilage of that side. It was just such a loss of substance as one often sees in cases of old phthisis, but I failed to detect any caseous matter in it. There was no ulceration of the trachea or bronchi. The lungs were crepitant, but there were sparingly disseminated grey discrete tubercles, in a few places becoming yellow. Near the base of the left lung was a cavity of the size of a Barcelona nut, which had apparently ensued on acute softening of a group of tubercles. There was no lobular pneumonia. No tubercle was seen on the pleure. The spleen was enlarged, firm and crowded with tubercles, just becoming yellow, mostly about the size of a mustard-seed. The organ weighed one ounce. The liver, intestines, and mesenteric glands showed no sign of tubercle; but there were some distinct grey and white granulations in the cortex of one kidney.

Tuberculosis in children under twelve months old has not been regarded in England as a common disease. Dr. Duckworth has put on record the case of a child eight months old in the *Pathological Transactions* for 1875. Dr. Powell has recorded under the head of fatal hæmoptysis a case of tuberculosis in a child seven months old (*Pathological Transactions*, 1874). But my patient was just under four months old when he died, and I own that the idea of tubercle never occurred to me before the *post mortem* examination. With the exception of a rather weak voice and a very occasional cough in the last few days of his life, he had no respiratory signs; and, having at first satisfied myself that there was no congenital heart-disease to account for his atrophy, I had paid no further attention to his thoracic organs. I see from Steiner's book on *Children's Diseases* (Tait's translation) that the Prague physician has found tubercle in sucklings of two months old.

The question arises, whether some of the cases of *young* infants, which we write down as marasmus from bad feeding, may not be tubercular. But this case leads one strongly to believe that the tuberculosis may be secondary rather than primary. In this boy's family, there was no history of phthisis whatever.

The succession of events seems to have been as follows:—Bad feeding, diarrhoea and stomatitis; glandular inflammation, first of the glands under the chin, subsequently extension to glands along the trachea, in the mediastinum, and in the fissures between the lobes of the lungs, probably caseation, but, at all events, breaking down of inflammatory products; last of all, acute tuberculosis of the lungs, spleen, and kidney. I have carefully avoided the use of the word infection, but it seems impossible to doubt that the local inflammation was primary, and not necessarily specific, and that the specific products were secondary.

I wish to draw special attention to the enlargement of the spleen. Everybody is familiar with the disseminated small round caseous masses found *post mortem* in the spleen in acute tuberculosis; but the enlargement which goes along with the above condition, and which makes the spleen palpable below the thoracic margin has not received sufficient attention as a clinical feature.