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.

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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.