a small quantity of turbid whitish fluid, acid in reaction, escaped, but no blood. A large flat sponge was put into this wound, and two fingers were passed through it up above the umbilicus and cut down upon in the middle line as upon a director, the object being to save every minute possible, and this second opening was enlarged upwards with scissors. The stomach was found to be congested, and flakes of lymph were beginning to form on its anterior surface. Towards the cardiac end the lymph seemed to be thicker, so the stomach was pulled over towards the right and carefully examined, when the perforation was found quite easily. It was high up on the anterior surface, well to the left of the middle line. It was quite circular, three-sixteenth of an inch in diameter very cleanly cut, with sharp edges which were slightly blackened, reminding one very forcibly of the entrance wound of a Mauser bullet. Round the perforation there was great induration and thickening of the stomach wall, but there was no haemorrhage. An oval piece of the stomach wall was excised with a scalpel. It included all the coats, and was about an inch long and half an inch wide, with the perforation in its centre. The haemorrhage was trivial. The resulting wound was closed with a single row of fine silk Lembert sutures, particular care being taken to pass sutures well beyond each end of the wound. The whole peritoneal cavity was then most carefully washed out with warm boracic lotion, great care being taken to flush out the pelvis and the fossae near both kidneys. A small amount of food was thus washed out of the peritoneum. The upper wound was completely closed with silkworm gut sutures, and the greater part of the lower one, but a Keith tube was put down into Douglas's pouch at its lower angle, a provisional suture being put in at that spot, to be tied when the tube was removed. The wounds were dressed with cyanide gauze and iodoform. Before leaving the operating table an enema of one pint of hot saline solution with an ounce of brandy was given. The operation lasted 75 minutes, and the pulse was better at the end than at the beginning. The excised piece showed a deep indurated conical ulcer with the perforation a little to one side of its apex.

Progress.—The patient was put to bed surrounded with hot bottles, and was ordered six-hourly enemata of half a pint of beef-tea with 1 oz. of brandy, and nothing by the mouth except tiny sips of water. A ½ gr. of morphine hypodermically was ordered if restless or in pain. She soon rallied, and only complained of thirst, which was relieved by an enema of a pint of warm water. The lower wound was dressed every six hours at first, and at each dressing any fluid in the Keith tube was sucked up with a syringe. At the first dressing the quantity was 2 oz., but this rapidly diminished, and the frequency of dressing was reduced accordingly. The Keith tube was removed on the fourth day (July 11th) and a smaller rubber tube substituted for it, which was finally removed on the seventh day (July 14th). On this date the stitches of the upper wound were irritating and were all removed, the lower ones being left a few days longer. The bowels were opened by a soap and water enema on July 10th, and after that acted of themselves. Food was first given by the mouth on July 11th, 3 oz. of Benger's food weak and cool being given, and as it caused no pain or discomfort whatever it was continued every four hours alternately with 2 oz. of somatose solution. On July 14th some peptonized milk with barley water was allowed, and food was given at three-hour intervals. On July 13th Brand's essence was added. On July 14th she was advanced to jelly and eustard, and on July 18th to plain biscuits and lightly-cooked eggs. On July 21st, the fourteenth day from the operation, she was allowed boiled sole, on the 28th chicken, on August 1st a chop, and on August 6th anything she fancied in reason. After the operation her temperature was erratic, never very high, but always above normal for quite a month. Usually it was 100° or 100.5° at 8 p.m., but on several occasions it was up to 101.5°. There was nothing in the condition of the wounds to account for this, and there was no pain, and repeated examination of the chest and abdomen failed for a long time to find any cause for it, though I suspected empyema or subdiaphragmatic abscess. At last about August 9th she began to cough and brought up a little thick purulent expectoration, and on August 15th a small patch of dullness with scattered coarse crepitations was discovered low down in the left mid-axillary line. By this time the temperature had dropped, and in a few days the dullness and crepitations entirely disappeared. She got up for the first time on August 19th and left the hospital on September 2nd. At no time after the operation did she have pain or even discomfort after food.

RESULT.

At the date of writing (July 4th, 1903) she can eat and digest any ordinary food without trouble, she has put on weight, and is in better health than she has been for many years, and greatly appreciates the fact that she has no longer to study her diet in any way.

REMARKS.

The interesting point of this case is the entire disappearance of all gastric symptoms after operation. This I believe to be due to the complete excision of the ulcer. Jacobson¹ rather discourages excision on the grounds that "much extra time will be consumed, there may be a good deal of haemorrhage, and the perforation converted into a large gap requiring numerous sutures to close it." These objections may often be sound, but in a case like the present, where the patient was seen soon after perforation took place, where the perforation was easily found, where the stomach was empty, and the ulcer was small, excision certainly seemed to be the right thing. Simple suture of the perforation cannot have any

curative effect upon the ulcer, and though the patient might recover more rapidly from the operation itself the gastric pain and discomfort would probably remain unrelieved, and the risk of haemorrhage would be as great as before.

REFERENCE.

¹ *Operations of Surgery*, 4th edition, vol. ii, p. 205.

SLEEPING SICKNESS AND TRYPANOSOMIASIS IN A EUROPEAN: DEATH: PRELIMINARY NOTE.

By SIR PATRICK MANSON, K.C.M.G., M.D., F.R.S.,
Lecturer, London School of Tropical Medicine, etc.

DR. DANIELS, late Superintendent of the London School of Tropical Medicine, and I recorded a case of trypanosoma fever in the *BRITISH MEDICAL JOURNAL* of May 30th of this year. At that date the patient had left hospital for some time, but I occasionally heard of her progress. About two months ago suspicious drowsiness was reported. This gradually became more pronounced and she died with marked symptoms of sleeping sickness on November 26th, two years and three months after the presumed date of infection. At the necropsy and subsequent histological examination of the brain unequivocal evidence of sleeping sickness was obtained.

Considering that this is the first well-authenticated case of sleeping sickness in a European, that it affords almost conclusive evidence that the trypanosoma is, to say the least, an important factor in the etiology of this terrible disease, and that the subject is deservedly exciting much interest, I feel justified in offering this preliminary note for publication. In due course further clinical particulars, together with a detailed histological examination of the brain and other tissues by Dr. Mott, F.R.S., and by Dr. Low, of the London School of Tropical Medicine, will be forthcoming.

The leading facts are briefly as follows:

About August 14th, 1901, the patient, the wife of a missionary stationed at Monsembe, Upper Congo, was bitten on the left leg by some animal, presumably a tsetse fly (*Glossina palpalis*). The bite inflamed, and a fortnight later she had the first of a long series of recurring attacks of fever. About the same time a peculiar patchy and ringed erythematous eruption appeared on the skin; and the spleen and liver enlarged. She returned to England in December, 1901. In May, 1902, she had an attack of phlebitis in the left leg. On October 3rd, 1902, I saw her for the first time and diagnosed trypanosomiasis, a diagnosis subsequently confirmed by Dr. Daniels, who after long search found trypanosomes in her blood. She was in the tropical wards of the London School of Tropical Medicine for about five months. A number of futile attempts were made by arsenic, methylene blue, horse serum, and other means to destroy the trypanosomes. She left the hospital on March 27th.

On leaving hospital she went to the seaside and improved considerably. When again I saw her on May 5th, 1903, she seemed to be in fairly good health, bright, and able to walk about, taking an active part in conversation, free from fever, and apparently much improved, although the erythema and parasites persisted. She then went to Cromer; the weather there was cold and raw, and, she thought, had a prejudicial effect on her health. The attacks of fever became more frequent and severe, and her general condition rapidly deteriorated. She returned to her home at Bristol feeling weak and ill. About the middle of October, although able to get out of bed and walk from one room to another, her friends noticed for the first time a tendency to drowsiness. On this being reported, Dr. Low and I went to Bristol to see her. She was in bed, extremely weak, very much wasted, with a rapid pulse, which she had had all along, and some fever; the ringed erythema was still distinct. The trypanosomes were somewhat more numerous in the blood. She was emotional, but could converse for a short time intelligently, only when conversation stopped she would close her eyes and appeared to sleep. Her husband informed us that this soporose condition was somewhat less marked at the time of our visit than a few days before. The knee-jerks and superficial reflexes were present, the sphincters were under control, the pupils reacted normally; there was no tremor, but there was a distinct twitching at the left angle of the mouth. Dr. Low, who had seen a great deal of sleeping sickness in Uganda, would not commit himself to a diagnosis, though we both regarded the case as exceedingly suspicious.

From that time the patient's condition gradually got worse; drowsiness increased, conversation became confined to monosyllables, occasionally food was retained in the mouth for a considerable time before it was swallowed, the sphincters were no longer controlled, spasm of one arm occurred, bed-sores formed, and she died comatose on November 26th.

Dr. Neild, University College, Bristol, kindly made a necropsy the same day. Macroscopic signs of chronic meningo-encephalitis— injection of vessels, and milkiness of the pia arachnoid—were found. Subsequently microscopical examination of brain sections by Drs. Mott and Low (whose preparations I have examined) revealed the extensive perivascular small mononuclear infiltration so characteristic of sleeping sickness.

MEMORANDA:

MEDICAL, SURGICAL, OBSTETRICAL, THERAPEUTICAL, PATHOLOGICAL, ETC.

RED LIGHT TREATMENT OF SMALL-POX.

[Communicated by the DIRECTOR-GENERAL OF THE MEDICAL DEPARTMENT OF THE ROYAL NAVY.]

I READ with interest in the BRITISH MEDICAL JOURNAL of June 6th the communication of Professor Niels R. Finsen on the red light treatment of small-pox. In July, 1897, I had an opportunity of trying this treatment—or, as Professor Finsen prefers to call it, "preventive measure"—and the results surpassed one's highest expectations. A bluejacket was received on board a gunboat I was then serving in from the flagship on the China station at a port in Korea for passage to hospital at Nagasaki. His temperature was 103°, rapid pulse, lurred tongue, headache, and the patient very ill. He was in the second week of the disease, but suppuration was slight. There was an abundant eruption over the face, scalp, backs of the hands, and a less abundant eruption over the trunk and limbs. He was placed under the forecabin in a swing cot, and enclosed with a canvas screen. The only light was supplied by two scuttles, which were covered with thick "red bunting" (used for making flags). He was treated in the usual way with liquid diet and tepid sponging. In two days the temperature had fallen to 99°, and there was a very marked effect on the eruption, and his general condition much improved. He made a rapid and uninterrupted recovery, although the light preventive measure was not carried out after he was placed in hospital at Nagasaki. There is nothing new in this communication, but is one more positive proof of the efficacy of this light treatment, and it may help others in a similar position to try it.

MONTAGUE L. B. RODD,
Surgeon, R.N.

HYPERPYREXIA IN MEASLES.

I HAVE in a recent epidemic of measles in this district (Falls-worth) had about 50 cases, of which 4 died, and these all 4 died of hyperpyrexia. They all had lung complications. Two had moderately severe bronchitis, 1 very severe bronchitis, and 1 moderate broncho-pneumonia. The latter was seen by Dr. Ernest S. Reynolds, who verified the diagnoses and temperature. The cases and ages were:

M. J. D.	...	...	19 months	...	F.
J. N.	...	...	3 years	...	M.
E. A. M.	...	...	10 months	...	F.
J. H. W.	...	...	33 years	...	M.

The history of the first three cases was similar. In them I was called in to a case of apparently normal measles, with varying degrees of bronchitis, the two female children having commenced convulsions, and J. N. having "fainted." The rash in all three was just beginning to fade, and the temperature in them when I first saw them was in all the cases about 106° F. Cold sponging, ice to the head, etc., and, in the cases where a bath could be got, a cold bath with added ice was used, but the temperature (taken in the rectum) rose in the bath, and death ensued in all three, in periods ranging from four to seven hours.

The last case, J. H. W., I had attended through a preceding bronchitis, and when the rash came out diarrhoea and broncho-pneumonia commenced. I saw him on the evening of October 28th (the evening before death), when he was delirious, with a temperature of 104° F. The rash was beginning to fade. A bath was given. He fell asleep, and slept quietly until 4 a.m., awaking talkative, and much better; the temperature was then 101° F. I was again sent

for at 7.30 a.m., as he had "fainted." I found him pale, with moist skin and stertorous breathing, and complete insensibility, with a temperature of 106° F. I telephoned for Dr. E. S. Reynolds, and meanwhile repeated the bath. Dr. Reynolds, at 9.30 a.m., October 29th, found the temperature up to 110° F. This was confirmed by three thermometers. The child died at 10 a.m., with a temperature of 111° F.

I understand from Dr. Reynolds that hyperpyrexia in measles is unknown, and in no authorities he and I have consulted do we find any mention of it.

I have since casually heard of three other cases of sudden deaths during measles, with apparently similar symptoms. Convulsions accompanied the hyperpyrexia in the two female babies. The treatment adopted was cold to the body, tepid baths with ice added, and ice to the head. No effect was seen on the temperature.

Dr. Reynolds considers these four cases unique, and that in the case he saw the hyperpyrexia was doubtless the cause of death. It is by his request and advice I put them on record.
Falls-worth, Manchester. E. BARNES, M.B.Aberd.

CASE OF ACTINOMYCOSIS OF THE LOWER JAW.

IN the beginning of March, 1901, a delicate, anaemic girl suffered from a hard swelling on the lower jaw, connected with a decayed, hollow right molar tooth. The swelling had gradually become larger, and at intervals shooting pain occurred in it. This was different to the continuous active pain associated with the usual development of an alveolar abscess. External application had no appreciable effect, and eventually, as pus was forming, the molar tooth was extracted, and matter escaped. In spite of this, the abscess continued to advance to the surface, and opened in two places. A granulomatous condition speedily developed round the openings, and granules of actinomycosis were discovered in the discharge. They were small in size, yellowish, and did not exhibit many of the "clubbed" elements. Potassium iodide in increasing doses was given till the amount taken per diem soon reached 90 gr. Similar effects in the discharge and granules were observed as detailed in a case reported in the BRITISH MEDICAL JOURNAL in June, 1896. Several days after the potassium iodide was administered, the granulomatous condition entirely disappeared, the wounds becoming quite flat. On April 16th Mr. Godlee, who had seen the patient and corroborated the diagnosis, operated by incising the abscess and scraping away as much deposit as possible, and also thoroughly cleaning the bone, about three-quarters of an inch of which was denuded of periosteum. Pure carbolic acid was poured into the wound, and allowed to remain for a few minutes, and finally the bone and abscess cavity were well rubbed with a swab soaked in pure carbolic acid. The wound was packed with iodoform gauze, which was removed in three days. Healing gradually took place by granulation, leaving a scar of a keloid type about an eighth of an inch wide; but a certain amount of induration remained about the margin of the abscess cavity. The opening of the abscess into the mouth had closed, although this was the channel of least resistance.

Another abscess a little behind and inferior to the first one, formed, and on May 16th was treated by Dr. Dickson and myself in the same manner and with a similar result. Induration round the margin again remained.

A third abscess still lower in the neck, in close contiguity to the previous one, speedily followed, and was opened on May 24th; but as we proposed to try the effect of spirit of wine we adopted the following plan which, we think, deserves further trial and investigation.

The needle of an antitoxin syringe was plunged into the abscess cavity, and some pus was squeezed out. Then spirits of wine was injected under pressure and allowed to remain for about ten minutes. The abscess was then opened and thoroughly scraped out and packed. It cleaned out much more thoroughly than the previous ones and healed quickly, leaving a narrow linear scar without any induration at the margin. There was no further spread of the disease in this direction.

On July 4th a final smaller abscess communicating with the jaw-bone was opened over the site of the first abscess. It had worked its way straight through the middle of the keloid scar. It was treated with spirits of wine and thoroughly scraped. It healed well, and there has been no further sign of the disease.

The potassium iodide was continued in large doses for about three months as a precautionary measure, and