atropine sulphate gr. $\frac{1}{15}$. He became quiet in about a quarter of an hour and slept peacefully and continuously until 7 p.m. He then awoke conscious and comparatively himself. He had a good night and awoke next morning feeling "very comfortable."

He has since then had no more bad symptoms, and although he has still a fair quantity of albumen in his urine he is in other respects very well. Oedema, flatulence, and vomiting all quickly disappeared, the cardiac impulse came in to well inside his nipple line, and he now expresses himself as feeling better than for many months. He remembered faintly my finding him on the floor of his bedroom at 1 a.m., and then remembered nothing else until he awoke at 7 p.m. on the same day, and wondered why his elbows were bandaged. His case was, I imagine, an acute attack on the top of a chronic parenchymatous nephritis.

Although the venesection undoubtedly did him good, and possibly, too, the inhalation of oxygen, which was given continuously for about ten hours except when his restlessness was too great to admit of it, I have no doubt in my mind that it was the morphine which saved his life.

TWO CASES OF PEPTIC ULCER OF THE OESOPHAGUS.

By C. GORDON WATSON, F.R.C.S.,

ASSISTANT SURGEON TO ST. BARTHOLOMEW'S HOSPITAL; SURGEON TO THE METROPOLITAN HOSPITAL.

My attention has been called recently to the account of an interesting case of perforative peptic ulcer of the oesophagus, published by Dr. James Miller in the BRITISH MEDICAL JOURNAL of January 20th, 1912. The two following cases came under my notice at St. Bartholomew's Hospital, and should be recorded on account of the rarity of the condition. The ulcer in the first case perforated into the left pleura; in the second case no perforation occurred. In the first case a diagnosis of perforated gastric ulcer was made, and in the second a diagnosis of gastric ulcer. In both cases acute abdominal symptoms occurred; laparotomy was performed, and nothing abnormal discovered. Both cases ended fatally, and occurred in males over 40.

CASE I.—*Acute Perforation of an Oesophageal Ulcer into the Left Pleural Cavity.*

A. S., male, aged 47, was admitted to St. Bartholomew's Hospital under my care at 11.45 p.m., May 22nd, 1910.

History.—Sudden epigastric pain at 7.30 p.m. (whilst lying down). Last meal 3.30 p.m. (mutton and potatoes). No history of jaundice, indigestion, or urinary troubles.

Condition on Admission.—Face pale and pinched. Temperature 95°; pulse 100, regular, weak; respirations 36; abdomen distended, not moving on respiration. No localized swelling. On palpation, abdomen very hard and very tender in the right hypochondrium. Liver dullness two inches below costal margin. Nothing abnormal discovered in heart and lungs.

Exploratory Laparotomy, May 23rd, 12.15 a.m.—No free gas or fluid. Intestines red and injected. Several small omental haemorrhages. Stomach, duodenum, liver, gall bladder, bile ducts, pancreas, spleen, appendix and kidneys examined and found natural. Wound closed. The stomach was washed out on the table, but only a small portion of the fluid was returned on siphonage. No vomiting occurred after this. The patient became very restless, respirations increased in rapidity, and he died at 8.10 a.m.

Autopsy.—The right pleura contained about a pint of blood-stained fluid. The right lung was collapsed, but was not otherwise abnormal. The left pleura contained four pints of thick turbid brown fluid with an odour of gastric contents. The fluid contained recognisable pieces of meat and vegetable. The left lung was collapsed. In the lower lobe near the middle line there was a small gangrenous cavity which communicated by an aperture about $\frac{1}{2}$ in. in diameter with the left pleural cavity, and by a similar sized opening with the oesophagus immediately above the cardiac orifice of the stomach. The opening in the oesophagus had a discoloured shelving edge, and was not clean cut. It showed no evidence of any pre-existing chronic ulcer or of new growth. No foreign body could be found in the neighbourhood of the perforation. The rest of the oesophagus was natural. The stomach was natural, and contained a considerable quantity of solid food. Liver fatty. Remaining organs natural. No peritonitis.

CASE II.—*Oesophageal Ulcer without Perforation.*

A man, aged 52, was admitted to St. Bartholomew's Hospital under the care of Dr. Herringham on May 8th, 1909.

History.—Quite well until five months ago, when he began to suffer from sickness after food without pain. Vomiting con-

tinued almost daily until admission, and the vomit was occasionally "coffee ground." For the last two months epigastric pain had been constant.

Condition on Admission.—Large, well built, stout, anaemic man. Temperature 99°; pulse 98; respirations 20. Brassy cough. Arteries not thick. Tongue furred. Chest: Nil abnormal. Abdomen: Tenderness to the left of the ensiform cartilage. Urine: Natural. Blood: Red corpuscles, 3,104,000; haemoglobin, 24 per cent.; colour index, 0.4; white corpuscles, 10,000. A skiagram of the chest showed a normal area of cardiac dullness and a normal aortic shadow.

May 8-27. Vomit contained altered blood and no free HCl. Temperature was irregular.

May 15. Pain constant. Vomiting frequent.

May 22. No food given by mouth. Rectal salines.

May 26. Eggs and milk by the mouth.

June 2. No pain or tenderness.

June 4. Vomited altered blood.

June 6. Pain returned. Frequent vomiting.

June 15. Transferred to surgical ward (under the care of Mr. Lockwood).

June 20. Operation. Incision in middle line above the umbilicus. Nothing abnormal found. Wound closed. Temperature and pulse rose after operation and patient died on June 22nd.

Autopsy.—No peritonitis. All organs very pale. Stomach natural. Duodenum contained a pouch the size of half a chestnut. Oesophagus: There was a chronic punched out ulcer the size of half a crown involving the lower end of the oesophagus close up to the cardiac orifice. There was no perforation into the pleural cavity, but the base of the ulcer was extremely thin. Microscopically the ulcer showed chronic inflammation and no evidence of malignant disease.

EIGHTIETH ANNUAL MEETING

OF THE

British Medical Association.

Held in Liverpool on July 19th, 20th, 22nd, 23rd, 24th, 25th, and 26th.

PROCEEDINGS OF SECTIONS.

SECTION OF TROPICAL MEDICINE.

Professor J. L. Todd, McGill University, Montreal, President.

PRESIDENT'S INTRODUCTORY REMARKS.

THE PRESIDENT limited his introductory remarks to expressing a few words of welcome to those present, and then called upon Drs. J. W. W. Stephens and H. B. Fantham to read the first paper.

PAPERS DEALING WITH TRYPANOSOMIASIS.

I.—J. W. W. STEPHENS, M.D. Cantab., and H. B. FANTHAM, D.Sc. Lond., B.A. Cantab.

TRYPANOSOMA RHODESIENSE (STEPHENS AND FANTHAM).

A SECOND SPECIES OF AFRICAN TRYPANOSOME PRODUCING SLEEPING SICKNESS IN MAN.

Prefatory Note.

EARLY in 1910 a case of sleeping sickness in a European from Northern Rhodesia was being treated in the Royal Southern Hospital, Liverpool. The case was studied from various points of view by Sir Ronald Ross and Dr. D. Thomson.

In March of the same year I was demonstrating the trypanosomes in the blood of a rat sent as *T. gambiense* to a class of students, when I noticed certain peculiarities in the structure of the trypanosomes. These peculiarities were so striking that I remember well telling my students to label their slides *T. gambiense* (?) as I felt certain that some mistake had been made. On inquiry I found that the rat had been inoculated with the blood of the patient in hospital, though at the time I was ignorant of this. I now proceeded to see if the forms I had observed were to be found in the ordinary laboratory strain of *T. gambiense*, but after examining the latter over and over again I was unable to find them. In a letter dated