precipitated in the epidermis, as described subsequently, the milliampère actually decreased after the first few moments, showing increased skin resistance and a lessened number of ions entering; to maintain the milliampère at all, the handle of the shunt resistance had to be continually moved in order to raise the voltage. In the case of ions subsequently proved to be deposited in the deeper layers of the skin, an intermediate effect was obtained, the current rising first and falling afterwards.

Hydrogen and hydroxyl ions cannot themselves be stained, but it was found that hydrogen ions caused the tissues to have an increased affinity for basic stains, the whole of the tissues under the ionized patch being deeply coloured by them, and the nuclei not picked out in the usual way. On the other hand, hydroxyl ions (introduced at the positive pole) produce the reverse effect, the tissue under the ionized patch failing to take basic stains. The staining in the hydrogen ion patches was even, and no granules could be detected. The effects persisted some time, as in one case the animal was allowed to recover, and the patches excised two days afterwards, when they still exhibited the same phenomena.

Attempts to stain sulphide ions by immersing the tissue in metallic salts, such as mercuric chloride, failed. Similarly, metallic ions could not be stained by immersing the tissue in solutions of sulphuretted hydrogen or potassium sulphide.

Ferrocyanide ions can be traced through the skin and subcutaneous tissue into the muscles; they are seen in large quantity in the capillaries of the subcutaneous tissue. All the tissues are stained blue, but the stain is not quite diffuse, as some parts seem to be picked out and stained more than the rest; the hair follicles and sebaceous glands are to a large extent avoided, and apparently the absence of staining of these is because the fat which they contain is a non-conductor of electricity. The taking up of the ions by the subcutaneous capillaries gives the deep layers of the subcutaneous tissue a more intense colour than the superficial layers. Ferrocyanide ions give a very similar appearance to that obtained with ferrocyanide ions.

Copper ions were easily demonstrated by fixing the tissue with potassium ferrocyanide. They were deposited entirely in the epidermis, not even reaching the corium, and the deposit extended a short way down the hair follicles.

With ferric ions, the tissues being fixed in a solution of potassium ferrocyanide, precisely similar results were obtained. With ferrous ions, on the other hand, the appearances were almost identical with those given by ferrocyanide ions, with the exception that there was a rather more abundant deposit in the epidermis.

Calcium ions were found to be deposited in and beneath the corium, only a small quantity going through the corium, even when a large current was passed for a long time. The deposit takes the form of minute granules which stain very intensely with a basic dye.

Unfortunately the attempts to demonstrate zinc ions failed; one would expect from the macroscopic results to obtain very similar preparations to those of calcium ions, as in each case the skin presents an opaque white appearance after the ionization.

Attempts to stain mercuric ions also failed, but from the behaviour of the current they apparently penetrated the tissue readily.

These experiments are admittedly very incomplete, although they serve to demonstrate some important facts with regard to the science and practice of ionization.

I cannot conclude without tendering my best thanks to Dr. Goodbody for kind advice and assistance during these experiments, and to Dr. Bellamy for cutting the numerous sections in the last experiment.

A COMMISSION, appointed to advise the authorities in regard to the new German Criminal Code, has recommended that duelling with rapiers, provided that certain conditions are fulfilled for the protection of life, should not be made a punishable offence. The *Muench. med. Woch.*, in noticing this recommendation, remarks that duelling among students will in future lack the stimulation of being forbidden. Our contemporary therefore anticipates that this antiquated method of exercising muscles, pluck, and skill will become still less fashionable than it has been within recent years.

A CASE OF URÆMIA TREATED BY MORPHINE.

BY

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SUGGESTIONS in the textbooks in regard to the use of morphine in acute uræmia are usually so unconvincing that I thought my experience in a recent case might prove useful.

On July 23rd of this year I was called to see W. L., a warehouseman, aged 56. He was sitting by the fire, wrapped in blankets; his face was puffy and dusky in colour, and his legs very oedematous; his whole body was also oedematous, though not extremely so. There did not appear to be any effusions into the serous cavities.

He had been feeling vaguely out of sorts for some weeks and, latterly, particularly short of breath and easily tired. A week before I saw him he had consulted his club doctor, who had prescribed for him. He had seen him again three days before my visit. He gave a history of a severe attack of scarlet fever seventeen years previously; eleven years ago he was told he had Bright's disease, but the doctor whom he then consulted told him he was cured of it.

The patient was too ill to examine very thoroughly at my first visit: the pulse was about 90, and very full; the cardiac impulse was about 1 in. outside the nipple line, with a blowing systolic murmur; the second aortic sound was accentuated, I had not my sphygmomanometer with me, so I do not know what his tension was, nor did I, unfortunately, take it with me before his acute symptoms started. He was complaining of constant eructation of wind and occasional vomiting; he had headache, but no ocular symptoms. The urine was almost solid with albumen on boiling.

The patient was put to bed, and diaphoretics, aperients and warm packs ordered. Next morning he expressed himself much better. The sickness and eructation of wind and vomiting had stopped, and the oedema had already to some extent receded. In the evening his son came to tell me that the vomiting had recommenced, and that he was not so well. Shortly after midnight I was sent for, and on going to see him I found that he had got out of bed and fallen on the floor; he was somewhat incoherent and could not raise himself; being a big, heavy, man, it was not an easy matter to lift him back into bed. I was inclined to think that he had probably had a fit and fallen out of bed. His pulse was still very full, though not rapid, and as I thought that convulsions seemed imminent, I tried venesection and inhalation of amyl nitrite. The venesection was not a success, only about 5 oz. of blood being abstracted. He became quiet and seemed inclined to sleep, so I left him, but ordered him another hot pack.

At 8.30 the same morning I was again sent for; I was told that he had had five fits since 7 p.m., and could not be kept in bed. I found him unconscious and extremely restless, cyanosed, and incessantly throwing himself about from side to side of the bed. Having had so unsatisfactory an experience of venesection, I did not attempt it again, but gave him a hypodermic of morphine sulphate $\frac{1}{4}$ grain with atropine sulphate $\frac{1}{10}$ grain, and told his friends to get a cylinder of oxygen and administer it continuously. I called to see him again about an hour after the injection, and he was sleeping fairly quietly and had not had another fit.

At 2 p.m. I called again; he had just had another very severe fit—the first since the hypodermic injection. He was unconscious, deeply cyanosed, with a slow, full pulse, and heaving himself up and down and from side to side of the bed as if in a struggle for breath. He did not keep in one position for more than two or three seconds, and the oxygen could not be administered because of his extreme restlessness. The dyspnoea was extreme and the veins extremely engorged. I now immediately venesected him, and with the utmost ease withdrew at least 25 oz. of blood. This improved his colour considerably and to a certain extent his restlessness. He was still restless, however, and I was so anxious about the condition of his heart that I thought quiet must be obtained at almost any cost, so I gave him another hypodermic of morphine sulphate gr. $\frac{1}{4}$.

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.

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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