On examination, a firm elastic mass could be felt below the lower edge of the right lobe of the liver in the gall-bladder area, which moved with respiration. Its outlines were rounded, and its lower extremity could be plainly outlined just above the level of the umbilicus. Its size was approximately that of a small cocoa nut. Its upper outline was lost beneath the liver. The abdominal veins were visible, and were, if anything, more marked on the left side. The size of the tumour appeared to her to have varied from time to time since it had been pointed out to her.

On December 3rd, 1900, a vertical incision was made through the right rectus muscle, over the tumour in the usual line. On opening the peritoneum a bluish grey tumour was seen, surrounded by omentum, which was loaded with fat, but was not adherent in any way to the mass. The tumour was easily turned out of the abdominal cavity, and was seen to be the greatly enlarged gall bladder. It was evidently transformed into a multilocular mass, the outer wall showing the outlines of the various cavities. To its lower extremity intestine was firmly adherent, and the adhesions contained large veins. As separation of these seemed undesirably risky, the lower extremity of the mass was stitched all round to the parietal peritoneum, after paracentesis of one or two of the larger cavities failed to bring about any notable diminution in its size. The main mass was then cut away, and the pedicle, which had a diameter of over an inch, left fixed in the abdominal wall. Some bleeding points in its circumference required a few ligatures. Gauze was packed around and over this.

Four days later she was somewhat jaundiced, and she was annoyed by a slight, but persistent cough. Bile had been escaping by a crescentic opening near the lower edge of the pedicle, but not to any great extent. There was for a time some abdominal distention, which was rapidly relieved by turpentine enemata. The stools varied in colour, sometimes light, sometimes evidently containing a normal amount of bile. The urine contained bile, but also in varying amounts.

As the wound had healed around the stump, but this remained practically unchanged, on January 28th, 1901, Paquelin's cautery was applied to its surface until it was mummified. After this it pretty rapidly contracted, but when she left the hospital on February 6th a biliary fistula still remained. This, however, closed entirely during the next two months.

In January, 1902, she married a second time, having been perfectly well in the meantime, and unfortunately became infected with the gonococcus almost immediately. In September she died with acute peritonitis following double salpingitis. I had the opportunity of inspecting the area of operation *post mortem*. The stump still lay in the abdominal wall, but was entirely covered by epithelium. Its continuation inwards was a normal cystic duct. Some small cystic cavities containing clear fluid were still discernible in the fragment of stump remaining; there were no secondary deposits, and no sign of new growth about the part still in existence. There were no gall stones.

The tumour on examination showed a large number of cystic cavities, of all sizes, generally polygonal in shape, owing to counter-pressure, containing clear mucoid fluid, but no calculi. The septa between them were composed of a central thin layer of connective tissue, lined on either side by mucous membrane. No sarcomatous or carcinomatous elements could be found.

It is far more usual to find a number of calculi in those cases in which they exist than single unfaceted specimens. It is, therefore, somewhat curious that I should have had 3 cases of single gall stones in a list of 35 upon which I have operated. One of these is of additional interest because of its probable causation.

CASE VI.

In April, 1903, I was asked by my friend Dr. Willis, of Gorton, to see with him a maiden lady, aged 48, who gave the following history.

When she was 4 years of age she was taken by her parents to New Zealand, where she lived for eighteen months; then she returned to England and has lived in this country ever since.

When 16 years of age she had a severe attack of pain in the right hypochondrium at the same point as she felt it in all succeeding attacks. This attack did not last long.

When 20 years of age she had a severe illness, which lasted about eight months, during four of which she was confined to bed. At this time she was deeply jaundiced, but has never been so since. This illness culminated in the suppuration and bursting through the epigastrium of a hydatid cyst.

For the last five or six years she had had intermittent attacks of paroxysmal pain. These attacks began as a dull ache over the hepatic region or with a feeling of dyspepsia; this might become gradually more acute, or the intense pain might begin suddenly. At times there was a dull pushing sensation in the gall-bladder area, at times it appeared to shoot, so that it made her jump. There accompanied all this an aching pain across the back below the scapulae, which was worst on the right side. The last attack of pain had been on April 10th, lasting for several hours, but not very intense. There was then an interval of a few hours, after which it returned again, and was only relieved by hypodermic morphine. The attacks had occurred every week or fortnight, but during the last few weeks before examination, they had been as often as two or three in a week.

Since morphine had been used, some vomiting had been noticed, but she believed that the drug was losing its effect, both as relieving pain, and as causing rejection of food.

The bowels had always been somewhat costive, but usually acted every day or every alternate day. The stools had never been white.

On April 19th, 1903, the abdomen was opened in the usual line. A

long Riedel's lobe was found hanging downwards from the right hepatic lobe, marked outside at the level of the costal arch by an obliquely transverse line of fibrous tissue, which however did not penetrate through its whole tissue. Behind this, a small contracted gall bladder which was adherent to stomach, colon, and duodenum contained one unfaceted calculus; its surface dark brown, and covered with small irregular eminences, about the size of a hazel-nut. No other stone could be felt. The adhesions were separated, and a drainage tube placed in the gall bladder, which contained, besides the calculus, a small quantity of muco-pus. The tube was removed on the third day. Bile continued to flow by the wound for about two months, but ultimately the wound entirely healed. Dr. Willis writes, March, 1904: Our patient has made a perfect recovery.

In this case, the sequence of events strongly suggests that the nucleus of the stone has probably been formed by one or more hydatid hooklets.

A CASE OF LEAD ENCEPHALOPATHY.

By W. ROSSELL JUDD, M.R.C.S.ENG., L.R.C.P.LOND.,
Ashton-under-Lyne.

At 11 p.m. on February 16th of this year I was called to see a young man, W. R., aged 23, and was told that he had been in a fit since 9 p.m.. He left home in the evening for the purpose of attending a cricket meeting, and was apparently in good health when at the above-mentioned time he was seized with a fit.

Previous History.—He was a plumber by trade, and up to four years ago enjoyed good health, when he had a similar fit, from which he recovered, and up to the last illness again enjoyed good health. He was very abstemious, never had colic, and was never troubled with constipation. On inquiry from relatives no history of neurosis could be elicited in his antecedents or in any living members of his family.

State on Examination.—On my arrival at his residence I found the patient in a state of violent epileptiform convulsions, almost amounting to acute delirium. On examination I found him to be well developed but slightly anaemic. The teeth were in a fairly good condition; there was no blue line on the gums; the tongue was slightly furred, and the breath not offensive. The pupils were contracted and did not react to light. The knee-jerk was present, and the plantar reflexes were exaggerated. Temperature was subnormal. The pulse was frequent during the height of the convulsions and almost imperceptible during the quiescent stage. The tension was poor.

Progress.—An enema was given, with the object of thoroughly emptying the bowel. Sinapisms were applied to the neck, precordium, and calves. The convulsions showing no signs of subsiding, I gave a rectal injection of chloral and potassium bromide. An hour after this no change having taken place, I gave a hypodermic injection of hyoscine hydrobromate ($\frac{1}{16}$ gr.). This quieted the patient, and he remained in this quiescent condition until the following morning. I saw him at 8 a.m. on the 17th, and found him in a semi-comatose condition. The same evening he again became very convulsed and passed urine involuntarily. The hyoscine was repeated with renewed quiescence. This alternate condition of semi-coma and convulsions went on until the morning of the 19th, when on my arrival I found him severely convulsed. I decided to abstract a little blood, with the object of relieving any cerebral congestion; not having a wet-cupping apparatus at hand I applied leeches to the cervical region, and by this means abstracted about 6 drachms of blood.

Result.—The patient, however, gradually sank into a state of coma and died the same day, so that from the beginning of his illness on the 16th to his death on the 19th he never regained consciousness.

REMARKS.

Fortunately for diagnostic purposes the patient passed some urine prior to his leaving home on the evening of the seizure. On analysis the urine presented the following features: Specific gravity 1020, fair quantity of albumen, no sugar, no casts, presence of lead. The presence of lead was detected by Von Jack's test much advocated by Abram and Marsden, of Liverpool.¹ A deposit of lead was noticed upon the magnesium within an hour. I allowed the magnesium to remain in the urine for twelve hours, and after carefully washing and drying, on the application of a crystal of iodine and warming, a yellow discolorization took place due to the formation of iodide of lead. The presence of lead was further verified by confirmatory tests. I may add that to the busy practitioner the test is one of great simplicity, provided that reliance can be placed upon the agents used—that is, to be free from any lead contamination.

The case is of interest from the fact that although this was the first case under my personal care, two other cases seen during my hospital career had a fatal termination, so that one must agree with Dr. Oliver² that of all the lesions which plumbism gives rise to, lead encephalopathy is the most grave. The question arises, what is the actual cause of the convulsions?

Here we have a young man apparently in good health, without any preliminary signs or symptoms of lead poisoning, suddenly struck down by the most severe of convulsions, which terminate in his death. Has lead a special predilection for the nervous system in certain individuals? If so, are we to regard the convulsions as due to the direct irritation of the lead upon the nervous system, or may we look upon the convulsions, as in the case of uraemia, due to auto-intoxication from the retention of harmful substances due to defective renal metabolism?

REFERENCES.

Lancet, January 16th, 1897. * Clifford Allbutt's *System of Medicine* vol. ii, p. 988.

TRYPANOSOMIASIS AND MORBUS DORMITIVA.

By ALEX. MAXWELL ADAMS M.B., C.M. EDIN.,
Late of the Gambia Protectorate.

IN the *BRITISH MEDICAL JOURNAL*, March 28th, 1903, there appeared an article of mine entitled Trypanosomiasis and its Cause. In this I asked the question, "Has the trypanosome any relation to sleeping sickness?" pointing out that in Europeans the parasite is found in the peripheral circulation, and suggesting that the African lethargy is an identical disease, and also that the parasite might be found in the brain centres blocking the capillaries, thus causing local anaemia and corresponding somnolence.

This very important contention afterwards received confirmation at the hands of Castellani and Bruce, who first published their results in May and June, 1903, and in the report of the Royal Society, issued later on, Professor Castellani had first observed a trypanosome in the cerebro-spinal fluid about the end of the year 1902, but his opinion was that the disease was due to a streptococcus which he had isolated. My paper, written in December, 1902, could, therefore, not be influenced by this as yet unpublished observation. I was led to my conclusions by the following clinical considerations:

1. In the trypanosome fever and in sleeping sickness we have the same puffiness of the face and lower eyelids (I was the first I fancy to point out the fugitive nature of the oedema and temporary unilateral distribution).

2. Irritability and apathy in both instances.

3. The same alteration of voice—a characteristic huskiness. I do not here allude to the changes in the speech of the negro where the sounds are unintelligible through paralysis of the tongue, but to a primal change in voice *timbre* noticed early in the disease, and in all probability due to a local oedema of the larynx. I have not seen this change remarked upon by other observers. The symptoms in the two races seemed to me to differ in degree rather than in kind. The chief difference appeared to be that the European, though apathetic, seemed to have less capacity for sleep than usual, and that the African suffered but slightly from the initial fever.

An important point now arises, Colonel Bruce attributes the infection to a species of tsetse fly, and no doubt in Uganda this holds correct. It is understood that no horse can exist in a country infested with this pest; and it is very interesting to note that in the Gambia (with the exception of Kombo) we have the coexistence of horses and sleeping sickness, the horses in good health, and the sickness quite typical. Therefore, with this fly presumably absent, what is the origin of the disease in the Gambia? In my previous article I traced the origin to a rat-bite. However, people are not often so bitten, and cases of the disease in this colony, though somewhat scarce, are not uncommon. Hence I conclude that if the disease were caused by insect bites cases would be numerous, and a state of affairs would exist similar to what we have on the Congo, where we hear of as many as 4,000 cases in one district alone; whereas in the Gambia it is to be doubted if more than a few cases could be collected at one time, facts which point to a different method of dissemination.

In India there is a trypanosome disease in horses probably caused by the common horse fly, and I see little difference between this species (*Tabanidae*?) and a fly found in the mangrove swamps of the African river. This insect at times attacks the human species and might thus introduce the parasite.

I am more disposed, however, to trace the disease to the rat—in the Senegambia at least. I lean towards this view on account of the long-known and well-proved existence of trypanosomiasis in that animal. Sir Patrick Manson suggests the *Ornithodoros moubata* as a probable cause, but this animal is unknown in the Gambia, though, judging from

analogy, it is quite a possible vehicle of infection, especially when we bear in mind that the parasite of Texan cattle fever is carried by a tick.

Again, the *Halteridium* of birds has been shown to be one stage of a trypanosome, whose full sexual life is completed in the body of *Culex pipiens*. As long as the parasite continues its existence by indirect generations or fission, I can easily conceive of several species of blood-sucking insects conveying the fissiparous germs, and that different methods of infection are possible as in typhoid fever.

The real animal in which the complete sexual life of the parasite is reached has not yet been discovered. Till this is done, the tsetse and other flies must be looked upon as an indirect though no doubt an efficient mode of infection.

The elucidation of the complete cycle of the life-history of the trypanosome is necessary to the complete proof of its relation to sleeping sickness; and when this is done—as I have no doubt it will be—far-reaching practical results will be in sight. My former paper seems to have attracted little attention. In this I put forward my claim to be the first one who suggested that sleeping sickness was caused by a trypanosome, and, secondly (what is more difficult to prove), that the disease in the European and African is identical. Discussions in regard to priority have been rife in regard to this disease. The observations contained in my previous paper and this are, I venture to think, non-controversial, and cannot in any way be considered as minimising the splendid work accomplished by Nepveu, Dutton, Castellani, and Bruce.

AN ADDRESS ON THE PROPOSED MEDICAL ACTS AMENDMENT BILL.*

By J. P. WILLIAMS-FREEMAN, M.D. DURH.,
Chairman of the Division.

GENTLEMEN,—The Committee of the Division which was called to arrange for this meeting suggested that it might tend to expedite the long and somewhat tiresome business before us, if I should, instead of giving a presidential address on some medical or surgical subject, endeavour to analyse and summarize this proposed Bill to Amend the Medical Acts which has been drafted by the Medico-Political Committee of our Association, and which the Council has sent down for our consideration to-night. In doing so, therefore, I am responsible only for the manner and not for the matter with which I shall try your patience.

There is one central fact which we must never lose sight of in a discussion on this subject, and that is that the Medical Acts of the past and any new legislation which we may hope to get enacted in the future can only be for the benefit of the public, and not for that of the medical profession. No doubt we are quite right to try to bring before Parliament the interests of the profession, and the public would wish to see them safeguarded, but only in so far as those interests are their interests, and not for our professional benefit. They do not wish to work the willing horse to death—they are even anxious to improve the breed of horses—but as only workers for the general weal. The days of special legislation for professional and class interests are happily nearly over.

It may seem unnecessary to insist on this, but I have done so because there is always a natural tendency to forget this principle—not certainly more amongst us than in the ranks of other learned professions—but enough I think to make us often overstate our case and do harm rather than good to our best interests. I doubt, for instance, whether the excited agitation which some of our friends thought fit to indulge in in connexion with the Midwives Bill has done much to raise the profession in the estimation of the public.

The great central change in the Bill before you is the establishment of one uniform State examination held by the General Medical Council, without passing which no one can practise medicine in the United Kingdom or (subject to any local law) in any part of the empire.

The early parts of the Bill lead up to this examination, and Part VI consists of penal clauses to protect the public from practitioners who have not passed it. It is this penal portion that will perhaps excite most discussion. The public likes and always has liked quacks of all descriptions. A certain interest in the unusual, a vein of superstitious credulity in the mysterious (*omne ignotum pro magifico*) which runs

* Delivered before the Salisbury Division of the Southern Branch of the British Medical Association.