shell having torn most of the extensor tendons and laid open the ankle-joint.

Earlier in the war it was the custom to make careful inquiries as to whether or not a wounded patient had received a prophylactic injection of antitetanic serum. The administration of these injections has become so much the invariable practice at the front that in the base hospitals one has ceased to ask the question.

Thus it happened that it was not until the occurrence in this case of subsequent events that one was led to investigate the question. The patient did not remember having received an injection; but it is possible that it was given while under an anaesthetic at the time of his first operation in France, or, on the other hand, it may have been that, wounded as he was far behind the firing line, he missed receiving treatment at a field ambulance or at a clearing station.

He came under my care at the 4th London General Hospital on November 16th, 1915, five days after he was wounded. For some days the wounds, which were extremely septic, were treated by peroxide irrigation and boric fomentations.

Six days later—on November 22nd—some trismus and a definite risus sardonius were noted. He was at once given 4,000 U.S.A. units of antitetanic serum, and 1 c.cm. of 1 in 20 carbolic acid was injected subcutaneously every four hours. He received a further 1,500 units of serum the same evening.

Had it been possible to localize the focus of infection as being in the wound of the foot, it would have been an easy matter to amputate immediately, especially as the foot was so severely injured that the prognosis was not hopeful. But in the presence of other septic wounds, any one of which might be equally the source of infection, it seemed to me wiser to secure efficient drainage everywhere by means of opening and irrigating thoroughly the wounds of the thigh and buttocks, and by an astragalectomy on the foot.

After the initial symptoms the march of events was rapid. The muscles of the abdomen and back were strongly contracted, and convulsions occurred frequently. On the evening of November 23rd he was given 1,500 units, as well as a quarter of a grain of morphine and 2 drachms of chloral. The pain in the back and leg was severe. On November 24th he had a further 1,500 units of serum, chloral, and bromide every six hours and morphine in the evening. The same course of treatment was pursued on November 25th. On November 26th and 27th the patient remained in a semi-comatose state from the effects of the chloral and bromide. Whenever he was allowed to come round at all the spasms became intense; on November 28th they were less severe, and on November 30th he was treated less energetically with hypnotics. The mental condition now began to show a marked change. He became extremely restless and passed into a state of low, muttering delirium, with visions and hallucinations—a condition closely resembling delirium tremens. From time to time he attempted to get out of bed, and had to be forcibly restrained. This lasted for five days, during which time he had incontinence of urine and faeces. Chloral and bromide, which had previously quieted him, had now no effect. After four four-hourly doses of half a drachm of paraldehyde, however, he settled down into a quiet sleep.

On December 7th incontinence had ceased and he had regained a more normal mental state. Paraldehyde was continued every four hours until December 8th, as well as occasional injections of morphine and atropine and draughts of chloral. On December 9th the mental condition appeared normal, but on December 10th there was a bad relapse with more incontinence. This lasted acutely for three days, but it was not until December 20th that the mental state again became normal and he was able to recognize those around him.

With the exception of the single shell explosion noted above, the man had never been under fire. This fact, as well as the delay in onset of the mental symptoms, tends to show that they were not due to shell shock. On the other hand, I am not familiar with the occurrence of such a form of delirium as being a symptom of tetanus itself. This is also the experience of those of my colleagues with whom I have consulted. I have thought it possible that the carbolic acid injections may be not unassociated with the condition.

Injections of 1 c.cm. of 1 in 20 carbolic acid were given at four-hourly intervals from November 22nd till December 4th. At no time, however, were there any other symptoms of carbolic acid poisoning.

The patient's general health is now (February) perfect, and there is no trace of mental instability. The movements at the ankle are quite good, and with his remaining extensors he is able to carry out a fair degree of dorsiflexion.

THE Secretary of the United States Treasury, Mr. W. G. McAdoo, has transmitted to the House of Representatives, on behalf of the Public Health Service, a recommendation for an emergency appropriation of £20,000 for the promotion of rural sanitation with the object of checking the spread of pellagra and typhoid fever.

A CASE OF ACUTE GANGRENOUS CHOLECYSTITIS WITH SPREADING PERITONITIS OCCURRING IN THE EPIDEMIC OF JAUNDICE, GALLIPOLI, 1915.

BY

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*With Observations on the Pathological Findings in
their Relation to the "Epidemic" Jaundice
of the Dardanelles.*

BY

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Cases of ascending infection of the bile ducts, liver, and gall bladder became extremely common during October, 1915, amongst our troops on the Gallipoli peninsula. The cases, as we met with them in the early days of their illness, may be grouped clinically into three categories:

1. Jaundice, associated with a variable degree of pyrexia, and usually enlarged and tender liver.
2. Acute cholecystitis without jaundice.
3. A combination of the first two types, with jaundice accompanied by acute cholecystitis.

According to our observation, the three classes occur with a relative frequency that corresponds with the order given above, though the patients with cholecystitis may well develop jaundice at a later stage. The following case is reported in order to draw attention to the danger of an acute gangrenous inflammation of the gall bladder—a condition which is in any event of extreme gravity, and, apart from prompt surgical intervention, is almost inevitably fatal from general peritonitis.

Case.

Pte. S., R.A.M.C.(T.), aged 22, reported to one of us (F. B. S.) on sick parade on October 5th, 1915, complaining of the following symptoms: (1) Pain in the epigastrium that had been constant, slight, and aching in character for four or five days; (2) a cough that was worse at night, aggravating the pain; (3) general malaise, anorexia, and slight headache for the same period. There was no history of vomiting, though there had been occasional nausea and retching after food. There was nothing irregular in the frequency and character of the urine or faeces, though he had suffered from diarrhoea a few weeks before. He had been inoculated against typhoid (two doses) about twelve months before.

On examination the temperature was normal. There were no abnormal physical signs in the chest. The tongue was not quite clean, and there was indefinite epigastric tenderness. He was treated as a case of simple indigestion.

On the following day the symptoms were the same, and the temperature still normal. On the morning of October 7th he complained of pain across the lower chest on respiration. The temperature was 99.2°. The chest was quite clear. The epigastric tenderness was more marked but not definitely localized. The tongue was coated yellow, and there was a left follicular tonsillitis. That evening the man complained that his pain was worse, and he was admitted to the dressing station tent with a temperature of 102° F. During the night the pain grew worse, and the patient vomited several times.

On the morning of October 8th he was evidently very ill indeed. The temperature was 102.8°, pulse 120, and he was flushed and showed a marked "abdominal facies." On

examination of the abdomen respiration was shallow, jerky, painful, and limited to the thorax, the abdomen being almost immobile. On palpation there was general abdominal tenderness and rigidity, but all down the right side it was most marked, and in the right hypochondrium the tenderness was peculiarly exquisite. Percussion did not reveal any shifting dullness in the flanks. The liver dullness was more limited than usual.

Perforation of an inflamed gall bladder with spreading peritonitis was diagnosed, and the patient was removed to the main dressing station of the 1st East Lancashire Field Ambulance for immediate operation.

Operation.—Captain J. M. Postlethwaite anaesthetized with chloroform, and Captain Haskins assisted. Subcutaneous saline was given into the left axilla during the operation. A vertical incision through the right rectus was made over the gall bladder. A considerable quantity of turbid, foul-smelling, bile-stained fluid was found in the peritoneum in the right kidney pouch, extending down into the right iliac fossa and pelvis. The gall bladder, which proved to be unusually difficult of access, because so far back under the liver, and adherent, was examined after the peritoneal cavity had been packed off with gauze. It was distended and deeply congested. It showed no gross perforation, but on its inferior aspect, showing up in marked contrast against the surrounding purple, was a green gangrenous area rather larger than a shilling, with two or three smaller discrete patches of gangrene round it. No gall stones were present. The liver was normal in colour and consistency, and was not enlarged. Cholecystectomy was hardly feasible owing to the anatomical peculiarity of the case and the lack of appropriate instruments, so a drainage tube was inserted through the gangrenous patch near the fundus and sutured in. The gall bladder contained somewhat turbid, foul-smelling bile under considerable tension. A gauze drain was inserted alongside of the tube in the gall bladder, and the peritoneal cavity swabbed dry. The abdomen was closed with through-and-through silkworm-gut sutures.

Progress.—Rectal salines were given every four hours for the first twenty-four hours, and the patient was kept propped up in the Fowler position. Bile drained away freely, but two days later the patient was perceptibly jaundiced, though it soon faded. He improved at first. The abdomen became soft and mobile, the bowels were freely moved, and it was evident that there was no further peritonitis, though there was some suppuration in the abdominal wall. But the temperature remained high, and a typical clinical picture of severe typhoid fever gradually developed. For about ten days the temperature varied between 103° and 105°, with a pulse of 120 to 130. There were frequent loose motions, he was greatly prostrated, and the condition was complicated by an area of pneumonic consolidation at the base of the left lung, with a small serous effusion in the pleural cavity. Towards the end of the third week of his illness, however, the temperature began to drop and the pulse to improve, and at the time of writing (October 29th), though very emaciated, and still too weak for removal to a hospital ship, we hope that he is fairly on the way to ultimate recovery. The main credit for this is due to his nursing orderlies, and particularly to Lance-Corporal Gregory, R.A.M.C.(T.), who has shown the most unremitting care and devotion in a very difficult case.*

Pathological Investigation.

A specimen of fluid from the gall bladder and subsequently several specimens of faeces and one of blood, for examination by cultural methods, were submitted to Lieutenant William Campbell, M.B., Ch.B., R.A.M.C., at the Pathological Laboratory, Cape Helles, and we are greatly indebted to him for the following reports:

Specimen of Fluid from Gall Bladder (October 16th).

1. I have been unable to find any entamoebae.
2. *Micrococcus tetragenus* is present.
3. *Staphylococcus pyogenes albus* is present.
4. The only other organism isolated is an organism giving the cultural reactions of a paratyphoid bacillus and agglutinating up to titre with the agglutinating serum of *B. paratyphosus* B (1-4,000 Lister Institute paratyphoid B serum). Isolation of this organism was easy, and it is likely that it was present in greater numbers than either *M. tetragenus* or *S. pyogenes albus*, owing to the number of colonies of this organism (paratyphoid B) which came up on plate cultivation. *B. coli communis* was not isolated, and the above represent the total findings.

* NOTE.—Since these notes were written (October 29th, 1915) we have been informed that the patient died in hospital in Alexandria.

October 24th. The blood culture, taken from the above case (October 21st) has remained sterile.

Three specimens of faeces (No. 1, No. 2, and No. 3) were sent to the laboratory on October 19th, 20th, and 21st respectively.

Specimen of Faeces No. 1 (October 19th).

Report.—Thin, offensive stool, showing no blood or mucus to the naked eye. Microscopically, no entamoebae found. Active *Trichomonas hominis* crowds all the fields examined. No blood cells were found. Specimens plated on MacConkey's and on Endo's medium on October 19th. Likely colonies picked off after culture and examined further.

Result (October 24th): *B. paratyphosus* B isolated.

Specimen of Faeces No. 2 (October 20th).

Report.—Naked-eye appearances: Pea-soup type of stool; no blood and no mucus seen. Microscopically, active *Trichomonas hominis* present in large numbers; entamoebae present in small numbers. No blood cells found. Specimen plated on MacConkey's medium, October 20th.*

Specimen of Faeces No. 3 (October 21st).

Report.—Naked-eye appearances: As in Specimen No. 2. Microscopically, active *Trichomonas hominis* present in large numbers; entamoebae (cysts) present in small numbers; no blood seen. Specimens plated on MacConkey's medium and on Endo's medium, October 21st.*

* Since *B. paratyphosus* B has been isolated from the specimen of faeces No. 1, and as no other organisms of the typhoid or of the dysentery group were isolated from it, I do not think it necessary to carry the examination of specimens No. 2 and No. 3 further.

The above pathological report is of great value in that it may indicate the nature of the infection that gives rise to a considerable proportion of cases in the epidemic under consideration. Our specimen of bile was sent to Lieutenant Campbell with a request that he should look for the entamoeba of dysentery or any other organisms present, and we had no suspicion of paratyphoid before receiving his report. On clinical grounds there was a good deal of presumptive evidence for the view that the cases constituting this epidemic may be secondary to dysentery and due to the entamoeba. The great majority of patients with jaundice or cholecystitis give a history of dysentery or diarrhoea, commonly ceasing two to four weeks before the hepatic symptoms began. But few men who have been many weeks on the peninsula have not had some diarrhoea or dysentery at some time, so that too much weight cannot be given to the suggestive association of the two epidemics. We have, moreover, found a few patients with epidemic jaundice who deny having had any diarrhoea or dysentery at all during the campaign. It is plain that the etiological problem is essentially one for the laboratory, and it is only from cases requiring operation, or from *post-mortem* examinations, that the necessary material from the biliary tract can be obtained. Without claiming for a moment that this very important etiological problem can be settled by the evidence forthcoming from a single case, the presence of *B. paratyphosus* B in the gall bladder in great numbers is an arresting and suggestive fact that should be borne in mind in further investigations. If it should be established from a series of cases that the most serious cases in this epidemic are due to this organism, it may prove possible by prophylactic inoculation to save our troops from this disease.

Observations on the Pathological Findings in their Relation to the "Epidemic" Jaundice of the Dardanelles, by WILLIAM CAMPBELL, M.B., Ch.B. Edin., B.Sc.(P.H.), Lieutenant R.A.M.C.

If any medical officer serving in the zone of active operations on the Gallipoli peninsula were asked to mention those medical conditions which, from the number of cases affected by them, were of greatest importance, he would immediately answer: (1) Diarrhoeal diseases, (2) pyrexias of uncertain origin, (3) jaundice (? epidemic), and he would inevitably add the remark, "In October, 1915, the numbers of cases of jaundice had been so remarkably large that they constituted a definite epidemic." I have queried the term "epidemic" above because it is exceedingly doubtful whether these cases form a true epidemic in the sense in which I have been accustomed to accept the term.

The designations "Weil's disease" and "epidemic jaundice" are commonly used by medical officers to describe the cases of jaundice occurring, and many such cases came from the trenches to the clearing station with one or other of these two terms already marked on their tickets.

If by "epidemic" it is meant that many cases of jaundice occurred amongst the troops in a given area, then the use of the term may be defensible; but if it be

implied at the same time that the cases are in any sense infectious in character, there can be no doubt that the qualification "epidemic" should be given up unless and until the jaundice be proved on epidemiological and bacteriological grounds to be so.

While numerous cases of jaundice showed no elevation of temperature, there were very many others accompanied by definite pyrexia. This latter fact would seem to point to some organismal cause, especially as in a few cases which have been observed an antecedent pyrexia has been noticed during the two or three days immediately preceding the appearance of yellow coloration of the conjunctivae and skin.

There is at present little to support the view that the jaundice is haematogeneous in origin, and the probability that most of it is due to ascending infection of the biliary apparatus (catarrhal jaundice) is strong. This may be stated with emphasis, because, as Captains Morley and Smith have pointed out above, practically every one who has been a short time on the peninsula admits to having suffered from diarrhoea of some description; and it is an interesting fact that, with the advent of the colder weather, the decrease in the number of cases of diarrhoea from all causes marched practically *pari passu* with a corresponding increase in the number of cases of jaundice.

Regarding this, attention may be drawn to the view mentioned above, and based on clinical grounds, that there is a good deal of presumptive evidence that the cases constituting the epidemic may be secondary to dysentery, and due to the entamoeba. But, while it is true that of those cases of diarrhoea the causes of which have been ascertained in the laboratory amoebic dysentery has formed a large proportion, it must be borne in mind that far from all the diarrhoeal cases are of amoebic origin; and it is to be noted that cases of enteric fever due to the paratyphoid organisms (including a smaller number due to *B. typhosus*), the smaller number of diarrhoeal cases which have been due to the Shiga-Flexner group of bacilli, the few cases due to the trichomonas, and finally those cases whose specific cause could not be ascertained by the bacteriologist, all go to make up the complement of diarrhoeal diseases from all causes.

If, then, it be sound to take the apparent parallelism between the incidence rate of diarrhoeal diseases from all causes and that of the so-called "epidemic" jaundice as indicating some relation between the two, it is reasonable to suppose that it is unlikely that the cases of jaundice have a uniform bacteriological cause.

Further, it was a matter of common knowledge on the peninsula that amongst apparently healthy individuals, even in those cases who have had no actual diarrhoea, a condition of chronic laxity of the bowels, unaccompanied by pyrexia, in which two or three bulky pultaceous motions were passed daily, was practically universal. This fact indicates that a very large amount of refuse matter is being got rid of, and would therefore seem to point to the diet containing too much indigestible material. But, as the army diet is reasonably good in quantity and quality, it rather points to there being some underlying catarrhal inflammation of the gastro-intestinal tract, which again is in favour of the view that much of the jaundice is due to ascending infection of the common bile duct from the intestine. It is improbable, therefore, that all cases of "epidemic" jaundice are due to a single specific organism.

On the other hand, the occurrence of numerous cases of cholecystitis, with or without jaundice, is striking, and these cases of cholecystitis may afford a possible clue to the cause of some of the cases of "epidemic" jaundice in that they may be due to an organism whose growth is favoured by bile. Such an organism, having multiplied in the gall bladder, might very well produce a secondary descending infection of the common bile duct.

It must be remembered, however, that, as there have been many cases of amoebic (*Löschia histolytica*) dysentery, and that as numerous individuals, who have not suffered from actual dysentery, harbour the entamoeba in their intestines, many cases of cholecystitis may represent the so-called "hepatitis," which is characterized by pyrexia and by tenderness in the right hypochondrium, and recognized as being a frequent complication of amoebic dysentery. Indeed Captains Morley and Smith have referred above to the tentative suggestion that the epidemics of cholecystitis and of jaundice may be in large part secondary

to dysentery and due to the amoeba. At the same time, I am not aware that jaundice has been found in places, where amoebic dysentery is endemic, to be a particularly common sequel of infection with the entamoeba.

It will be gathered that, while the etiology of the "epidemic" jaundice of the Dardanelles is still uncertain, the problem of its elucidation gives great scope for laboratory investigations, especially to those bacteriologists who are fortunate in working at the base hospitals, where the cases can be kept for some time and who have quiet and settled conditions in which to prosecute their work. The matter can only be cleared up in the laboratory, for it must be granted that, from the clinician's point of view, the problem is not simple because he finds his cases of jaundice complicated in various ways.

For instance, the jaundice may be slight or pronounced in degree, the duration may vary from one or two days up to several weeks, and it may or may not be associated with one or more of the following conditions:

- (a) *Pyrexia of indefinite character*; sometimes transient, but frequently of the nature of a low continued fever. Many cases, however, are afebrile and some run a subnormal temperature.
- (b) *Digestive disturbances* (generally present); usually marked; evidenced in some cases by nausea and anorexia only, in others by intestinal colic, and in a few by diarrhoea.
- (c) *Cholecystitis*, with marked tenderness in the right hypochondrium; pain sometimes referred to back as well.

Captains Morley and Smith have noted above that cases of jaundice following an attack of cholecystitis, as well as other cases of acute cholecystitis without jaundice, are also met with. Further, medical officers find that in nearly all cases of jaundice the patient gives the information that he suffered from diarrhoea and colic at some time or other on the peninsula, but, owing to the prevalence of diarrhoeal diseases, this is not to be wondered at.

Broadly speaking, the cases of "epidemic" jaundice might be divided into two groups:

1. Jaundice with pyrexia.
2. Jaundice with no elevation of temperature.

It is doubtful, however, whether this classification would help us, for the problem is complicated by the fact that temperatures of irregular type and uncertain origin are by no means uncommon, and occur equally in patients with and without jaundice. Again, typhoid fever occurring in inoculated cases shows itself as an atypical and generally abortive fever. The same is true in some degree of enteric fever due to the paratyphoid organisms, because, although the immunity conferred by antityphoid inoculation has been regarded by some as being homologous, and although many paratyphoid infections occurring in patients inoculated against *B. typhosus* give a good clinical picture of enteric fever, there is strong evidence for believing that antityphoid inoculation gives a partial immunity to the paratyphoid fevers, and that, therefore, many atypical and abortive cases of these in all probability occur as well.

It follows from these facts that the diagnosis of febrile conditions has become much more difficult for the clinician, who too often finds himself left in doubt as to what value should be placed on many a temperature chart for diagnostic purposes. This is true generally, and it is true of cases of jaundice with pyrexia. The classification of cases of "epidemic" jaundice into febrile and afebrile varieties may on this account not be of so much value to him as might at first appear.

Pyrexia occurring in these cases indicates the facts (a) of infection and (b) of reaction on the patient's part; but cases of mixed infection are not unknown, and the temperature chart may therefore indicate reaction not to one but to several organisms. Or, in cases of mixed infection, the reaction may be to one organism, while other ordinarily pathogenic organisms found in such cases may be mere saprophytes in the intestine. In respect of this it will be noted that, in the case recorded above, *B. paratyphosus* B occurred in the faeces in conjunction with *Trichomonas hominis* and *Entamoeba histolytica*.

The first specimen sent to me from this case was one of bile, and from it *B. paratyphosus* B was isolated, as well as *Micrococcus tetragenus* and *Staphylococcus pyogenes*

albus. The two organisms last named I regard as contaminations. No organism was obtained from the blood culture taken from the same case.

Without attempting to assess to the *Trichomonas* and the *Entamoeba* in this particular case their pathological value (if any) in the production of the gangrenous cholecystitis, it seems fair to regard the *B. paratyphosus* B as having been the cause of the condition. To the clinical pathologist who has had the opportunity of seeing patients with "epidemic" jaundice, the case suggests one or two points which I shall now discuss very briefly.

As is generally recognized, catarrhal jaundice commonly occurs without cholecystitis; similarly, cholecystitis is very frequently unattended by jaundice. Both facts are capable of a simple anatomical explanation. But in some cases in the Gallipoli epidemic jaundice followed a definite cholecystitis, and one hears as well of numerous cases of jaundice in which a history of troublesome spasmodic pain in the right hypochondrium, highly suggestive of an antecedent cholecystitis, was obtained. It may therefore be that the relation between the cholecystitis and the jaundice is of greater importance than has been recognized hitherto, and it is possible that cases of cholecystitis treated by operation will furnish to the bacteriologist the most hopeful material for the solution of an etiological enigma.

From this point of view the case recorded by Captains Morley and Smith may be of special value. It shows, at all events, that in a patient inoculated against typhoid fever a paratyphoid organism in the early days of infection may ascend from the intestine to the gall bladder and there settle without causing more than an initial and indefinite epigastric tenderness associated with anorexia and headache; that later its numbers and virulence may be so increased as to cause actual gangrene of the gall bladder; and that, therefore, it is not improbable that from the gall bladder, in cases of cholecystitis, a subsequent descending infection of the common bile duct with consequent jaundice may take place—an infection due to an organism which has meantime been multiplying in the bile. This may explain numerous cases of jaundice with pyrexia, especially those cases which give a history suggesting a previous cholecystitis.

It is likely, then, that a close study of those organisms whose growth is known to be favoured by bile may help to enlighten us as to the cause, or causes, of the "jaundice with pyrexia" which occurred in the Dardanelles; and, although jaundice is not a common complication of ordinary typhoid fever, it is possible that the paratyphoid infections may play a much more important part than the *B. typhosus* in the production of febrile jaundice. The possible connexion between the "carrier" problem and the occurrence of febrile jaundice suggests itself; and it would be interesting to know how many cases of "epidemic" jaundice, bacteriologically examined, have yielded a paratyphoid organism.

Be this as it may, I am for various reasons strongly of the opinion that it is highly improbable that the cases of jaundice occurring in the Gallipoli "epidemic" have, by any means, a uniform specific bacterial cause.

REMOVAL OF INTRACRANIAL FOREIGN BODY UNDER X RAYS.

BY

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THE following case of gunshot wound of the head, with a piece of shell in the brain, illustrates the value of the use of an electro-magnet or x-ray screen for removal.

A lance-corporal received a gunshot wound on September 25th, 1915, in France. The piece of shell entered the skull about the middle of the left temporal region. He had an injection of antitetanic serum four hours after the injury. When admitted to this hospital on October 9th he was conscious, complained that he could not sleep, and that he had considerable pain. The superficial wound in the side of the head had healed. The temperature was 99° and the pulse 60. Next morning he vomited.

A skiagram taken on October 11th showed some depressed fragments of the internal table round a small hole in the middle

of the squamous portion of the left temporal bone, and the shadow of a piece of shell about $\frac{1}{2}$ in. by $\frac{1}{4}$ in. was seen on the plate 1 in. behind the hole and $\frac{1}{4}$ in. beneath the surface.

On October 13th Mr. Leslie Paton examined the eyes (pupils not dilated) and found: The right optic disc, highest point + 3 D., edges blurred, veins distended, general retina + 5, swelling 2 $\frac{1}{2}$. Left optic disc, highest point + 5 D., general retina + 1, swelling + 4. The patient had sensory aphasia, word-blindness, but no apraxia.

Operation.

A large temporal flap was made, and the opening in the skull enlarged; pieces of depressed bone were removed, the torn edges of the membranes were opened up, and about 2 oz. of thick pus under pressure escaped. The piece of shell could not be felt with the probe, and a rubber tube was put in and free drainage established.

On October 15th another skiagram showed that the piece of shell had dropped further back in the abscess cavity into the occipital lobe. The temperature at this time varied between 97° and 99°, but on October 23rd the evening temperature was 100°, on October 24th 101°, and on October 26th 102°; it then became normal and has remained so ever since. The pulse-rate varied between 64 and 100, and after October 26th was from 72 to 80. On October 23rd the eye appearances were: Right optic disc, highest point + 3.5 D.; left optic disc, highest point + 4 D.; veins still much distended.

A probe was passed down along the abscess track, and a stereoscopic skiagram taken.¹ The fragment of shell was found to be lying in the occipital lobe, one eighth of an inch in front of the tip of the probe and half an inch mesial to it. A steel probe was then passed down the track, and an electro-magnet capable of lifting 14 lb. weight was applied to the probe and held in position for a few seconds, but the bit of shell was not drawn out by this means.

It then seemed to me that it might be possible to see the fragment, and remove it under the x-ray screen, and, after consultation with Dr. Florence Stoney, the radiologist to the hospital, the attempt was made on November 9th. The steel probe was again passed in along the track, and I was able quite clearly to see the probe and the fragment. It was found that the latter had been pulled forward by the magnet, and caught in brain tissue. A crocodile forceps was then passed along the track to the depth of four inches, and after some manipulation the piece of metal was seized and removed. The patient was not under anaesthesia and felt no pain, except just as the sharp edges of the fragment came into contact with the edges of the superficial wound.

On November 13th the patient's general condition was quite good. On November 17th the wound had healed; aphasia was less marked, but he still had some slowing of mental cerebration, and at times difficulty in naming articles, with some loss of memory in naming places and for recent events. The eye symptoms were: Right optic disc still blurred; swelling subsided very much; highest point of disc + 2 D., general retina ametropic; small linear haemorrhages especially in lower part of disc; left optic disc still all blurred; subsidence not so marked in appearance as in the right eye; swelling, highest point of disc + 2 D.; some haemorrhages.

The two practical points demonstrated are: First, the use of an electro-magnet for the removal of portions of shell which are capable of being so attracted; and, secondly, the fact that bits of metal can be demonstrated inside the skull, although the x-rays have to penetrate two layers of bone; the surgeon may thus receive very material assistance in such cases by seeing the relationship of fragment to instruments during actual removal.

My thanks are due to Dr. Florence Stoney for the excellent skiagrams and help with the screen; to Dr. Leslie Paton, who kindly worked out the eye conditions; to Major Parsons, the Officer-in-Charge, and to Colonel Peterkin, the D.D.M.S., for permission to publish this case.

¹ One skiagram was reproduced in illustration of a report of this case published in the *Journal of the Royal Army Medical Corps*, January, 1916.

DR. ALLEN WALCOTT has contributed an article on beri-beri in the Amazon basin to the *Journal of the American Medical Association* for December 18th, 1915. He has been for six years in the employ of the Madeira-Mamore Railway, which runs through a dense tropical forest along the east bank of the Madeira River 1,500 miles from the mouth of the Amazon. There is a hospital at Candelaria on the line, but no other for many hundred miles in any direction. Out of 30,430 admissions in four years, 963 cases were diagnosed as beri-beri. On the strength of his own previous experience of this disease in San Francisco and Panama, Dr. Walcott concludes that beri-beri in Brazil is of the same type as is found elsewhere, and its cause is the lack of vitamins in the food. The doctors, nurses, and labourers in the medical department have been completely free from attacks of the disease for the last three years, protected by the special precautionary measures used in other lands where it prevails. These measures are described in detail by Dr. Walcott.