in front of the muscle, and extended well down the neck and towards the outer margin of the muscle. This had all the characters of an arterio-venous injury, and tended to increase slowly in size in all directions during the few days preceding operation. It appeared to be directly in connexion with the left common carotid artery, pressure on which completely stopped the expansile pulsation of the swelling and considerably reduced its size. Beyond the presence of the swelling and slight interference with the movements of the neck, the patient was not in any way bothered.

Operation.

At the operation, on February 26th, a free incision was made from the upper limits of the swelling along the anterior border of the sterno-mastoid nearly down to the sterno-clavicular articulation. After reflecting the skin, dissection disclosed that the main mass of the pulsating tumour occupied the substance of the left sterno-mastoid muscle, and was exceedingly thin-walled in its upper and outer part. By retracting the muscle backwards it was found that the common carotid formed a kind of neck to the sac, and the internal jugular vein passed straight into it. While attempting to clear the neck of the sac from below it gave way and some haemorrhage occurred, which was immediately controlled by pressure on the artery lower down. The common carotid and the internal jugular vein were then ligatured well below the lower limits of the sac, and the difficult part of the operation appeared to be over. Then the sac burst, and we saw that there was a considerable length of the internal jugular vein split inside it. Haemorrhage was free, and only partially stopped by pressure; and it was not till, after considerable tedious dissection, we had ligatured the internal jugular vein and a large posterior tributary and the external carotid artery above that all bleeding ceased. The sac was then opened up and cleared, and it was seen to be confined to the body of the muscle. Beyond the limits of the muscle there was no definite sac, and it is remarkable that a more extensive and diffuse extravasation of blood had not taken place. The operation took nearly two hours, and the patient, who had been given intravenous saline injection on the table, had a fairly good pulse, and under appropriate treatment soon rallied.

After-History.

But he lay in a dull, heavy, stupid condition for twenty-four hours. After that he gradually seemed to understand what was said to him, but was quite unable to put any of his thoughts into words for several days. His pupils were equal, as were his radial pulses; he swallowed well and, though quite aphasic, was able to make certain grunting noises, which reassured us as to the integrity of his recurrent laryngeal nerve. For two or three days there was a definite paresis of the right side, and the grip of the right hand was appreciably weaker than the left. Rectal and bladder functions were not interfered with in any way after the first twenty-four hours.

The peripheral symptoms very soon passed off, and in ten days' time no difference in the power of the limbs could be distinguished. The mental dulness rapidly improved, but recovery from the aphasia was a much more gradual process. About fourteen days after operation, much to his delight and to our satisfaction, he greeted me by articulating quite distinctly, after one or two false starts, "My name is Mustapha" (in Arabic), and two days later he wished me "Good-morning," without any preliminary bungling. Very gradually his re-education has proceeded, until now, four weeks after operation, he is sufficiently recovered to be able to tell us where he lives and to ask to be discharged.

His wound healed perfectly, and there is only a small hard lump in the muscle. In the left side of the neck no dilatation of veins can be seen, and there is no pulsation to be felt either along the course of the carotid or over the facial or temporal arteries. The radial pulses are the same on both sides.

Every day his aphasia is less, and it is very interesting to note his distress and annoyance at not being able to put his ideas into words, especially now that his mental condition is so thoroughly re-established and his ideas flow so much more readily. So rapid has been his progress in the last few days that I am hopeful he will eventually completely recover without any physical or mental defect.

NOTES ON A FATAL CASE OF ACUTE NEPHRITIS OCCURRING IN THE EXPEDITIONARY FORCE.

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My reason for publishing these notes on this single and rather incomplete case of nephritis is that I think it of peculiar interest; it gives me at any rate food for thought, and may help to elucidate the problems of this disease. Also, although I am, I admit, out of touch with the literature, I understand that very few complete *post-mortem* examinations on early cases of nephritis occurring in our forces in the field have been made.

Driver L., A.S.C., attached to a field ambulance, was admitted into the clearing station on February 17th, 1916. On January 30th his urine was examined, in course of a routine

examination of his unit by Captain McLeod of a mobile laboratory, and found to be normal. On February 7th he reported sick, and two days after he was admitted into the field ambulance with follicular tonsillitis. The temperature never exceeded 101° F. In two days the fever subsided and the throat cleared up. At this stage a moderate amount of albuminuria was present, but no oedema. For the next three days the temperature kept down, and the only thing which prevented his being returned to duty was the presence of complete anorexia and some vomiting.

On the fifth day in hospital his face was noticed to be puffy, and there was a large amount of albumin in the urine. The next evening the temperature ran up to 102.6° F., and in the morning he was evacuated to the casualty clearing station. He arrived with the convoy at about 2 p.m., and I examined him at 3 p.m. He looked very pale and ill and complained of headache and weakness. There was no marked oedema of the face, though there was some swelling of the ankles and a well-marked lumbar pad. The temperature was 100.5° F., and the tongue furred, brown, and extremely dry. The pulse was 100 and of fair quality. Except that the heart sounds were rapid and slightly feeble, and that there were rhonchi at the bases, the examination of the chest was negative. The abdomen was somewhat tumid, and there were signs of moderate ascites. The liver dullness was higher than normal behind, and the edge was felt in front below the costal margin. The spleen was not felt. Examination of the central nervous system elicited nothing unusual. The fundus was not examined. His general condition, though serious, did not lead me to suppose then that he would die. The urine was fairly profuse, pale, and clear. On boiling it was solid with albumin. On spinning the sediment showed hyaline casts, often with polymorphonuclears attached and free polymorphs. There were no epithelial casts, and nothing suggestive of the presence of epithelial cells.

I saw him again in the evening and found him no worse. Towards the morning he had diarrhoea and abdominal pain, and at 8.30 a.m. suddenly collapsed. I saw him shortly after, and found him cold and pulseless, but quite conscious and complaining of abdominal pain. He died an hour later, perfectly conscious to the last; in fact, he dictated a letter home some ten minutes before he died.

Necropsy.

I performed a *post-mortem* examination two hours after death, with Captain McLeod and Dr. Amecielle of the French medical service.

There was no abnormal pericardial fluid. The heart was small and the walls of the left ventricle distinctly thickened. The valves and aorta were normal. There was oedema of the bases of the lungs. The peritoneal cavity contained about 2 pints of opalescent fluid. The intestines were normal.

The liver was somewhat enlarged and fatty. The spleen was enlarged slightly, flabby; no necrotic patches. The kidneys were large, firm, and congested. There was much swelling of the cortex and pyramids, the latter standing out very clearly. The capsule stripped easily. The bladder was normal, as was the pancreas. The suprarenals showed some medullary hypertrophy and medullary haemorrhages. The meninges were slightly injected and the brain a little oedematous.

Blood taken from the vena cava contained 1.80 grams of urea per litre. A blood film taken just before death showed a high polymorphonuclear leucocytosis, estimated at about 40,000.

Bacteriological Data.—The peritoneal fluid yielded on spinning a sediment of leucocytes, mostly polymorphonuclear, amongst which were very numerous streptococcal chains of medium length. No organisms were found in smear preparations of the kidney or spleen pulp, but polymorphonuclear leucocytes were very numerous in the smears from the spleen. A growth of streptococci was obtained both from the kidney and the spleen. The organism had moderate haemolytic power; 5 c.cm. of a thirty-six-hour culture in horse serum bouillon, when injected subcutaneously into a rabbit, did not cause the death of the animal. A control injection into a similar rabbit of a streptococcus taken originally from a wounded French soldier in subculture for five or six months killed the animal. The only other organism isolated from the viscera was a coliform bacillus, which, as it grew from only one of several preparations of the kidney, can, I think, be disregarded as a *post-mortem* examination.

Microscopic sections of the organs showed that in the kidney the main brunt of the attack had fallen on the interstitial tissue. The glomeruli had escaped entirely and most of the tubules. In the interstitial tissue were several areas of acute congestion. In these were foci of polymorphonuclear and large mononuclear renocytes, forming microscopical abscesses. The tubules running through these areas showed some degeneration, but no desquamation of the cells. More over diapedesis of polymorphonuclear cells was seen taking place through the basement membrane, and some were seen in the lumen of the tubules. Globules of fat were seen in many of the liver cells. There were a few areas of cellular necrosis in the cortex of the suprarenal. The medulla showed haemorrhages and collections of polymorphonuclear leucocytes, and there was distinct medullary hyperplasia. Lungs showed some foci of congestion and oedema. The spleen was congested with an undue amount of leucocytes. The heart and pancreas were normal.

The actual cause of death in this case was, I think, septicaemia in a patient whose resistance was lowered by a certain amount of uraemia. The slight uraemic symptoms, namely, headache and anorexia, the mode of death

and terminal peritonitis all point to this. The case, however, presents many problems of interest. It started with tonsillitis probably of streptococcal origin, and at that time there was no albuminuria. The question arises, then, Did the tonsillitis pass on to a septicaemia, and cause a streptococcal interstitial nephritis, or did the toxin circulating in the blood from the focus in the throat cause the kidney trouble, leading to a general lowering of resistance, which allowed the streptococcus to get a footing in the general circulation? In other words, was this nephritis of pyaemic or toxic origin?

Personally I am inclined to the former view. I admit that no organism was demonstrable in the sections, and the fact of its growing from the kidney pulp can be explained by a general blood infection. I support my case entirely on the microscopical abscesses in the interstitial tissue of the kidney, and on the clinical course of the case.

My thanks are due to Captain McLeod, R.A.M.C., who did the bacteriological work, and to Dr. Amecielle, of the French medical service, who got the tissues sectioned for me, and gave me his valuable opinion on them.

INJURY TO THE INTERNAL EAR PRESENTING SOME UNUSUAL FEATURES.

BY

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SOLDAT D. was admitted on December 29th, 1915, with a bullet wound of the head.

Entrance wound left frontal eminence. No exit wound. Skiagrams (1) on admission, showed no bullet, but only splintered bone below the wound; (2) on February 2nd, showed no bullet, but a rarefied area, where at operation the bone spicules had been removed.

Treatment.—On admission the patient was anaesthetized and trephined. Fragments of bone were removed from the frontal lobe of the brain and the wound dressed with gauze. There was nothing to suggest a fractured base. There were no signs of focal injury nor of intracranial pressure. As regards his head wound, the patient made an uninterrupted recovery.

Evidence of cerebral irritation (sulkiness, mischievousness, resentment of any one approaching him) persisted for some days, and disappeared with the sudden onset, on January 8th, of escape from the left ear of fluid, which flowed slowly but steadily, was quite clear, and reduced Fehling's solution. With the flow of cerebro-spinal fluid all signs of cerebral irritation ceased. The flow persisted until January 24th.

Otoscopy.—The right ear was normal. In the left ear, half an inch from the external meatus, a sessile swelling of the bony roof bulged downwards, occluding three-quarters of the meatal lumen and preventing inspection of the membrane. It was covered with intact skin, sensitive and stony hard to the probe, and resembled a meatal exostosis. It was impossible to be certain that it was the result of the injury. This appearance persisted unchanged as long as the patient was in hospital, so that the membrana tympani was never seen.

Hearing was at first good, and equal on both sides to watch, speech, and whisper; no tuning-fork was then available. The hearing on the right remained normal, while on the left it became progressively worse. On January 26th he heard speech left 18', whisper 4'; on February 6th, speech 2', whisper 1'. On February 24th I obtained a tuning-fork (D.V. 512); Weber to right; absolute bone-conduction on left—15 secs. On and after March 5th, for speech, watch, and tuning-fork, both air and bone conduction were nil on the left side.

Equilibrium.—As soon as the patient was well enough to be lifted into a sitting position he fell over on to his left side. In a few days he could sit up without support, and walk unsteadily by January 15th. After January 26th he could walk straight with his eyes open; but with eyes shut staggered to the left. He next developed a crab-like gait (with eyes shut), diverging constantly to the left, and crossing the right foot over the left as he walked, to avoid falling to the left. On and after March 5th his gait was normal, with eyes opened or closed.

Evidence as to Cerebellar or Meningeal Lesion.—There was never any asthenia, dysidiadokokinesis, giddiness, vomiting, or nausea. There was no indication for lumbar puncture or examination of optic discs.

Nystagmus.—No galvanic apparatus or rotation-chair was available. Repeated observations were made, with the head in various positions and water at different temperatures; and the ears were irrigated both separately and simultaneously. The mean results obtained were as follows:

Spontaneous Nystagmus.—On deviation to right: Fine, pure horizontal, never of more than first degree. This became progressively less marked, and no nystagmus was obtainable after February 17th. On deviation to left: No nystagmus.

Caloric Response.—Head backwards 60 degrees; water at 90° F. run in for two minutes. Right: During the persistence of spontaneous nystagmus on deviation this was always made

coarser and raised to third degree. After spontaneous nystagmus had ceased (February 17th), irrigation elicited a similar but less violent normal response. Left: On deviation to this side there was never any response to irrigation. But, for as long as the spontaneous nystagmus to the right persisted, this could always be inhibited for periods varying from two to fifteen minutes by warm irrigation of the left ear.

To test this inhibitory effect of stimulating the left labyrinth on nystagmus to the right, the two ears were simultaneously irrigated several times after February 17th, when spontaneous nystagmus to the right had ceased. But at no time did the irrigation of the left ear have any effect on the nature or duration of the nystagmus obtained by simultaneous irrigation of the right ear.

On his discharge to the base, after ten weeks in hospital, he had on the right side normal caloric labyrinthine response and normal hearing. On the left side, total loss of labyrinthine response and complete deafness. Gait and general health normal.

The signs of cerebral irritation during the early days disappeared suddenly and permanently with the appearance of cerebro-spinal otorrhoea, which again ended spontaneously sixteen days later. Although there had been no classical signs of compression, it seems reasonable to suppose that the irritative stage might have merged into such, and that the cerebro-spinal flow was nature's decompression. The gradual loss of spontaneous nystagmus to the sound side and of a tendency to fall to the injured side, were merely evidence of recovery from partial or complete unilateral loss of labyrinthine function. Since stimulation of the left labyrinth always inhibited any spontaneous nystagmus to the right, but later on failed to inhibit caloric nystagmus to the right, and since also there was a progressive loss of hearing, there was obviously a progressive lesion of cochlear and vestibular apparatus.

The results of caloric tests on the left ear seem puzzling. Whether the lesion was in any part of the left external semicircular canal or in the nervous mechanism proximal to this, it was striking that stimulation should inhibit nystagmus to the right, but never succeed in eliciting nystagmus to the left.

The bullet was never removed from the skull, nor seen in a skiagram. It may never have entered, although the shape of the wound suggested penetration rather than glancing. If it is still in the patient's head, that would not seem to account for the findings here reported.

THE TREATMENT OF DIABETES.

BY

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THE cause of diabetes is looked upon as the result of the faulty action of certain glands, which action appears to result from, or be aggravated by, absorption of toxic products produced in the alimentary tract.

This statement appears to be borne out by the remarkable improvement which takes place in diabetics when the treatment is directed to controlling toxic production in the bowel. The beneficial effects of removing alimentary toxæmia is fully explained in an article on starvation and purgation by Dr. Guelpa of Paris.¹

There is a variation each day in the amount of sugar in the urine of diabetics which takes place independently of treatment. In my opinion this is due to the condition of the bowel, as I have invariably found the sugar content of the urine high when the bowel was loaded; after it was cleared by aperients a reduction in the amount of sugar was apparent.

As the treatment of diabetes by means of a starch-free diet is very irksome to the patient, and often unsatisfactory, I have for several years treated my diabetic patients by fasting, and cleansing of the alimentary tract, at the same time allowing a diet from which starch was not entirely excluded.

As early as three days after commencing treatment, I have invariably found alleviation of the symptoms, the thirst much less, the craving for food not nearly so pronounced, the pruritus, if present, markedly relieved, and the patient all round much better than on a rigid starch-free diet and opium derivatives.

Mode of Treatment.

I thoroughly inspect the mouth, have any defective teeth removed, and enjoin the necessity of thoroughly cleansing the teeth and washing out the mouth frequently.