cases the distribution is bilaterally symmetrical. The tumours are deep-seated; they are hard, non-fluctuating, very painful, with their long axes either in or parallel to the long axes of the muscles affected. They do not correspond to lymphatic glands. Each tumour is about the size of an English hen's egg. They are at first hard and firm, and are easily visible and palpable; later on, if the patient survives, they become slightly soft, but never flabby. Usually, in mild cases, the abscesses point after some weeks, and burst if unopened. As a rule, the fewer the nodules the better the prognosis.

The natives often incise the nodules to relieve the symptoms; if white fluid exudes, the case is labelled fatal; if the fluid is yellow, the patient will recover. Sometimes there is no fluid, and then the Ju-ju man gives the patient a delphic reply. Some of the natives carry the scars of former "bungpaga" abscesses.

The patient is alert and sleepless on account of the great pain in the affected muscles. Sedatives have to be used in large doses before any relief is obtained; salicylates are of no avail.

If the patient survives the acute stage he stands a good chance of recovery. The deaths usually occur within the first week, the third and sixth days seeming to be the most critical. Even during convalescence (for two months or more) the patient exhibits an occasional febrile reaction

Mortality.

"When it is called Bungpaga it is very fatal, nearly all die; but when we call it Bungyura most of the people recover." This is the typically oracular answer one gets from the natives. Some of the people consider the disease to be as fatal as sleeping sickness.

General Remarks.

From the investigations of Drs. Corson, Ryan, and myself we are inclined to believe that this is a special disease. The Hausas say that they have seen it occur among the other tribes, but not among themselves. Perhaps they wish strangers to be impressed with the efficacy of the *dawa* offerings. One could not discover whether the fortunate poor contracted the disease after eating these oblations.

The natives also readily recognize such infections as actinomycosis, anthrax, bubonic plague, guinea-worm, and malaria, syphilis, etc. Their knowledge of disease seems to be exact, but they are so superstitious and suspicious that they make one wade through a quagmire of falsehood and general information to get to the tiny islets of truth bearing on one's quest.

An epidemic of this disease broke out recently among the labourers employed on the Yeji-Mcongo road. The officer in charge of the construction work said that by far the largest number of fatalities were due to this disease. He had made arrangements for the provisioning of his workmen; large quantities of maize, millet, guinea-corn, etc., were bought and stored in mud huts so as to meet requirements. Some of this grain may have been infected, and may have given rise to this outbreak. Preventive measures should be adopted in good time so as to prevent an outbreak among troops who live on such food. Millet is often eaten in the raw state. Our men should be warned of their danger and should be ordered to cook their grain.

Investigations into the cause and spread of this disease are being taken in hand in West Africa, but it would help matters considerably if all cases of this disease were reported on.

AN interesting experiment in school hygiene at Winchester, Massachusetts, is described in the *Boston Medical and Surgical Journal*. Two seventh-grade classes, consisting of children of the same kind, were compared after one class had been for four months taught in a room so altered that a full supply of fresh air could be admitted. The temperature was kept at about 45° F., and the children were clothed in blanket coats and bloomers, with lined canvas boots. At the end of the period the children were compared with those of another seventh-grade class in the same building who worked in an ordinary room. It was found that whereas the average gain in weight of the open-air class was 3.61, that of the other class was 1.69. In height the open-air class gained 0.49, the other 0.24. In the haemoglobin test the open-air class gained 4.55, while the other lost 3.07 per cent.

AN OBSERVATION IN PERNICIOUS ANAEMIA.

BY

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SOME years ago, after careful examination of the literature, I published a paper* embodying an original idea, and ventured to express the view that, although original, I doubted whether it was novel. A few weeks later, whilst buried in journals in search of data on quite a different subject, I found that my idea had been anticipated some years previously, and on looking up that paper I found yet an earlier reference to a journal published in 1886 embodying the treatment suggested.

The vastness of the literature must be my excuse for absence of any references to experiments of a similar nature to the one I am about to describe.

In March, 1915, I had under my care a very severe case of pernicious anaemia, also one of well-marked erythraemia (polycythaemia).

The consensus of opinion as to the process leading to anaemia in pernicious anaemia is that an excessive destruction of red cells is occurring. The cause of this destruction has been a subject of considerable work, more speculation, and no satisfaction. It would ill become me to add to the speculation.

Erythraemia must be due to one or a combination of the following: abnormally rapid development of red cells, an abnormally slow destruction of red cells, an insufficient quantity of plasma.

On an attempt to determine which of these takes the most prominent part, I am engaged at present.

It seemed to me that it would be of interest to note what would happen if I were to transfer some quantity of blood from the patient suffering from erythraemia to the patient who had pernicious anaemia, and at the same time inject into the erythraemic patient some blood from the case of pernicious anaemia.

Courtesy to my colleague, Dr. F. J. Smith, makes me digress for a moment: the case of pernicious anaemia under my care wished to return home, and therefore left the hospital, and I am indebted to Dr. F. J. Smith for transferring to me one of his patients.

The patients had the circumstances explained to them in detail; that there were certain risks, and that as far as I knew, no similar observation had been recorded. The two men said that they were quite willing to undergo any treatment which might in any way benefit them, and that they did not mind any risk, because for several years their lives had been spent in hospitals and were of no advantage to them or any one else.

Details of the method of transferring the blood are unnecessary. All that is essential is to state that the apparatus was washed out with sterile olive oil and drained, so that there should be no excess, and that coagulation was prevented by the addition of a minute quantity of sodium citrate. Every precaution was taken to prevent any air being injected, and a window quite close to the point of injection allowed me to be certain that not a bubble entered.

After a small quantity—that is to say, 2 oz.—of blood had been injected into the man suffering from pernicious anaemia, he went paler; he complained of nausea and faintness, and the injection was immediately stopped. He then became slightly flushed in the face, and made grimaces as if he were undergoing pain. On being asked, he stated that he had pain all over him, something like cramp, quite severe; this lasted for a minute, and then disappeared, and he was soon quite comfortable.

I was very disappointed that only so small a quantity of blood had been introduced, but nevertheless thought that if the blood contained some material which stimulated production of red cells this might lead to improvement in the condition of the patient. Examination of the blood a week later showed that the red cells had risen from 600,000 to 1,050,000, and the haemoglobin percentage from 10 to 20.

As the result was so promising it made me almost regret that it was not my custom to publish any observation on treatment within a year of having made it; this resolution

* A Contribution to the Treatment of Chronic Empyema. *BRITISH MEDICAL JOURNAL*, April 15th, 1905.

had been brought about by a statement of a former teacher, whom I hold in the greatest respect, who had once (probably humorously, although truthfully) made the remark that journals took so long to publish papers that he had often altered his view before his theories appeared in print.

The result was sufficiently satisfactory to make the patient anxious to have further treatment, and five weeks later I again transferred blood, and on this occasion some 8 oz. of blood. The symptoms produced were similar to those produced on the former occasion, but much more severe. The patient vomited for close upon half an hour, and for a time gave rise to some anxiety due to the strain thrown upon his heart. The after-effects were not satisfactory, neither the number of red cells nor the quantity of haemoglobin being increased to any large extent.

Therefore, in this case, at any rate, transference of blood proved to be useless. The patient left the London Hospital in March, 1916, in a condition very similar to that in which he entered it in March, 1915. I record the observation in order that other observers to whom a similar idea may occur should realize that there is a certain risk in transferring large quantities, and that it is quite probable no benefit will result.

Reports

ON

MEDICAL AND SURGICAL PRACTICE IN HOSPITALS AND ASYLUMS.

THE ISOLATION HOSPITAL, SOUTHAMPTON.

AN ANOMALOUS CASE OF ENTERIC FEVER.

(Reported by OSCAR HOLDEN, M.D., D.P.H., Resident Medical Officer.)

T. F., a gardener aged 25, was admitted on August 1st, 1915. He had been out of sorts for about fourteen days, but remained up and about. Walked a considerable distance on July 29th, and got up to breakfast on July 31st, but went to bed at mid-day, and was delirious the same evening. He had consulted his private doctor on July 30th because of diarrhoea. He had not been inoculated against typhoid fever.

On admission he was seriously ill. There were sordes around the teeth and lips, the tongue was dry and foul, but there were no spots or rash. The abdomen was tympanitic, rigid, and diffusely tender; on palpation it moved fairly well on respiration. The spleen was not palpable; the liver was felt just below the chest margin. There was no cough or other lung signs, but a most persistent hiccough. The heart sounds were regular and strong, with a very faint rub at the apex.

The chief points in the further progress of the case were: Violent hiccough until the end; practically complete suppression of urine for forty hours after admission, and then a copious flow with incontinence; obstinate constipation for fifty-two hours after admission, and then large, frequent, and offensive stools, also incontinent. The abdomen, at first distended, became flat, but the tenderness persisted. Unconsciousness, becoming more profound, ensued, with increasing restlessness until death.

At no time was his temperature above 97° F. Widal reactions were inconclusive. Lumbar puncture, thirty-six hours before death, afforded no evidences of intraspinal mischief. The urine contained abundant albumin and a few granular casts. Death appeared to be due to a profound intoxication.

Post-mortem Examination.

August 7th, 2.30 p.m. Well-developed man. Post-mortem staining marked.

Abdomen: The omentum was spread out over the small gut and was injected. No free peritoneal fluid and no adhesions. Stomach, duodenum, and jejunum normal, except for slight injection, probably due to turpentine given therapeutically. The ileum showed no enlargement of solitary follicles or Peyer's patches, except in the last foot. Here in the upper half the patches and follicles were reddened and swollen, but not ulcerated. In the 5 in. above the ileo-caecal valve were masses of small ulcers, with some larger ones. So numerous were the ulcers that the gut wall presented a ragged, rat-eaten appearance. The ileal side of the valve was partially sloughed away. The appearance of the ulcers hardly warranted the

assumption that they were typhoidal, but bacteriologically they proved to be such. The appendix and large gut were normal. The spleen was hard, not enlarged, and rather anaemic. The liver, normal. The kidneys showed parenchymatous nephritis, more severe in the right organ. **Heart:** A patch of recent fibrinous pericarditis showed over the apex. There was no valvular disease. Atheromatous patches were scattered over the ascending aorta. The brain and meninges were normal.

Comments.

It seemed that the patient had been up and about during an attack of enteric fever, ending fatally six days after taking to bed.

The complete constipation may be accounted for by blockage at the ileo-colic valve during separation of the sloughs; the subsequent diarrhoea to imperfect closure after sloughing. The suppression of urine may have been caused by the onset of a specific parenchymatous nephritis.

The continual subnormal temperature is difficult to account for in view of the extensive ulceration and sloughing of the lower end of the ileum. The spleen was of a non-febrile type. Possibly the intoxication was so great that there was a depression of the cerebral nerve centres, but the heart rhythm was unaffected and the strength unimpaired until a few hours before death. The action of the toxin was similar to that seen in some suddenly fatal cases of diphtheria.

I have to thank Dr. Lauder, M.O.H. Southampton, for permission to record this case.

Reports of Societies.

VISUAL DISTURBANCES FROM CEREBRAL LESIONS.

At a meeting of the Ophthalmological Section of the Royal Society of Medicine on March 22nd, Colonel LISTER, A.M.S., C.M.G., and Lieutenant-Colonel GORDON HOLMES, M.D., R.A.M.C., read a paper on disturbances of vision from cerebral lesions, with special reference to the cortical representation of the macula. The authors stated that in their work at the base hospitals in France during the past eighteen months they had observed a large number of cases in which the vision was affected by lesions of various portions of the optic system, and a certain number of suitable cases had been selected bearing on the cerebral localization of vision, more particularly on the representation of different regions of the retina in the cortex. As the observations were necessarily made at an early stage after the infliction of the injury, some of the effects observed may have been due to functional disturbances, rather than to local injuries of the corresponding cortical areas or their centripetal fibres; but if it could be shown that there was a constant relation between the probable site of the injury and the form of the visual defect, certain general conclusions would be justifiable. The injuries in the cases referred to included penetrating and perforating wounds of the cranium by rifle bullets, shell fragments, and shrapnel, as well as local concussion and depressed fractures.

Superior quadrantic hemianopia was strikingly rare in gunshot wounds of the occipital region. The fact that in over 2,000 cases of head injury a central scotoma was never seen when a direct injury of the occipital poles could be excluded was regarded as striking evidence that central vision was represented on either, or both, the mesial or the lateral surface of the posterior borders of the occipital lobes. The observations conformed to the general view that the visual area corresponds with, or at least includes, the area striata. No conclusive evidence had been obtained that achromatopsia, with intact vision for white, was produced by cerebral lesions which involved either the cortex or the optic radiations.

The authors formulated the following conclusions, which they do not yet regard as final:

1. The upper half of each retina is represented in the dorsal, and the lower in the ventral, part of each visual area.
2. The centre for macular or central vision lies in the posterior extremities of the visual areas, probably on the margins and the lateral surfaces of the occipital lobes.
3. That portion of each upper quadrant of the retina in the immediate neighbourhood of, and including, the