this figure and that of the blood under examination is shown above or below this line, according to the numerical excess or deficiency in the average of germs phagocytied by each leucocyte.

It is too early to be able to say whether the temporary benefit which followed the inoculation is likely to be permanent; but the point which I wish to bring out is that the patient's phagocytic power, as gauged by the control blood, has varied in direct proportion to the amelioration of his symptoms.

Prior to the date of inoculation I made several examinations of his blood, and found its phagocytic power always lower than the control, but these have not been entered in the chart, as no exact numerical count was made on these occasions. For six days after inoculation it will be noted that his phagocytic power remained considerably lower than the control; during this time he was still having fresh crops of small boils and pimples on his neck. From the seventh to the tenth day he was free from boils, for the first time for many weeks, and a corresponding rise is seen in his curve. From the eleventh to the thirteenth day a few fresh pimples appeared, which gave him less annoyance than those he usually suffered from, and a similar outcrop occurred towards the end of the period of observation. On both these occasions a drop will be noted in his curve below the level of the control blood.

Without insisting on the uniformity or importance of the phagocytic estimation in this case, which has only been selected as an example of the method, the results appear sufficiently striking to warrant the hope that it may be of service in the investigation of immunity when combined with an estimation of the bactericidal power of the blood fluids.

Since the above note was written I have had the opportunity of examining the phagocytic reaction in another case which was treated eight months ago by Dr. Wright with injections of staphylococcus vaccine for boils from which the patient had suffered for eight years, and in whose case a permanent cure appears to have been effected. The phagocytic power of the polynuclears was found to be markedly greater than that of the control blood when tested with the strain of staphylococcus, which was originally isolated from one of his boils, and from which the vaccine used in his case was prepared, while the reaction was slightly higher than the control when tested with another variety of staphylococcus.

REFERENCES.

¹ *Lancet*, December 1st, 1900, March 2nd, 1901, and June 1st, 1901.
² *BRITISH MEDICAL JOURNAL*, September 21st, 1901. ³ *Lancet*, June 1st, 1901. ⁴ Proceedings of the Pathological Society, *Lancet*, March 23rd, 1901.

EPIDEMIC CATARRHAL JAUNDICE.

By ELIOT CURWEN, M.B., B.C.CANTAB.,
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In view of the present epidemic outbreak of jaundice in Derbyshire, so well described by Drs. Peck and Kershaw, the following brief notes of the first cases in similar epidemics in Pekin and Tientsin during the winters of 1898-9 and 1899-1900 may not be without interest. I have notes of only 44 of the cases seen during the first two months of the epidemic, which, beginning towards the end of October, 1898, lasted well into the spring of the next year; the following winter the epidemic returned, and presented the same features as in the previous year.

The first case seen was on October 22nd, and for some months after this few days passed without one, two, or three patients coming to the hospital for treatment. Their ages varied from 17 to 60, and seven times as many men as women applied for treatment. The attack from which one of my students suffered was typical of the series.

On October 27th and 28th he told me he felt weak and lethargic. On the evening of the second day he returned to his home feeling sleepy, ate two pears, had pain in the abdomen, and felt sick. At night he was very hot, complained of severe pain in his temples, and his conjunctivæ were, he said, suffused. During the next two days his temperature kept up between 102° and 102.4°. He felt very weak and ill, and the pain in his temples continued. The administration of calomel (gr. ii.) resulted in five dark green motions. On November 1st his temperature was down to 96° in the morning, and the conjunctivæ were slightly yellow for the first time; he was still very weak, and had a pulse of 60; there was no tenderness over the liver, or any abdominal discomfort; the tongue was slightly

furred. The colour of the conjunctivæ became more marked by November 4th, when he began to complain of itching of the skin; from this date his symptoms gradually improved; ten days later, however, the conjunctivæ were still of a pale lemon colour.

Most of the patients complained of great weakness and of some shortness of breath; there was generally anorexia, a feeling of abdominal distension, and nausea, and constipation with pale motions was the rule; in 4 cases definite abdominal pain was complained of, but from only 3 did I hear of actual vomiting; diarrhoea occasionally occurred. Another prominent symptom, noted in two-thirds of the cases, was pain in the temples, and this in some cases was described as very severe; it was often accompanied by drowsiness, and in 2 cases by vertigo. More than two-thirds of the patients told me that for the first few days of illness, varying from one to fourteen, they suffered from some degree of fever with shivering, and that as a rule the icteric tint of the conjunctivæ was first noticed the day, or the day after, the fever disappeared. In 5 cases the colour of the conjunctivæ was noted as deep, but in the majority of cases it was a pale straw or lemon colour, and not dark enough to be noticed in the yellow skins of the Chinese; the length of time it lasted was very variable. Only 4 patients complained of pruritus. In 2 cases the liver was noted as slightly enlarged and tender; no albumen was found in any urine examined, but Martin's and Gmelin's reactions were obtained in each case. The pulse was rapid—from 80 to 128—in each patient, with the exception of the case narrated above. One man, aged 60, whose fever had lasted ten days, told me that about the time his conjunctivæ became yellow he began to suffer from pains in the muscles of the right arm and in his two ulnar bones; the muscles and bones were still tender on deep pressure when I first saw him five days later.

The affection cannot be very infectious, for I never saw two patients from the same house, and one of my servants who was attacked did not communicate the disease to the other men who slept on the same brick bed; only one patient told me that he had neighbours suffering from the complaint. Some of my patients were severely ill for some days, and, on account of their high fever, weakness, and headache, were thought by their friends to be suffering from typhus fever; others, on the other hand, were affected so slightly that the febrile symptoms, if they existed, passed unnoticed, and they did not know there was anything the matter with them till I handed them a looking glass and told them to look at their eyes.

ENTERIC FEVER IN THE INOCULATED.

By MAJOR C. BIRT, R.A.M.C.,
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THE mortality of 947 uninoculated individuals who have suffered from enteric fever in Harrismith, South Africa, from September, 1900, to September, 1901, has been 14.25 per cent. There were 135 deaths among them.

During the same period 263 persons who had been inoculated with typhoid vaccine, for the most part 6 to 18 months previously, contracted this disease. Eighteen of these cases proved fatal. This is equivalent to a death-rate of 6.8 per cent.

The tabulated results of an analysis of 317 charts of the not inoculated and of 151 charts of the inoculated are given below.

	Uninoculated.	Inoculated.
Mortality	14.25 per cent.	6.8 per cent.
Average duration of pyrexia	28 days	15 days
Average maximum temperature recorded...	103.7°	102.9°
Percentage of cases in which relapses occurred	24.0 per cent.	6.0 per cent.
Percentage of cases with constipation	19.0 "	33.0 "
In cases with diarrhoea, average maximum number of evacuations per diem	4.4 "	2.6 "

The unmodified fever has been of severe type. *Post-mortem* examinations have been made in 65 of those who have succumbed. Death was due to toxæmia in 40 per cent., to pneumonia in 26 per cent., to perforation in 14 per cent., to exhaustion, etc., in 20 per cent.

In all the fatal inoculated cases an interval of eight or more months had elapsed between vaccination and the onset of the illness. In eight necropsies the cause of death was found to be toxæmia in 4, pneumonia in 3, perforation in 1.

It should be remembered that immunity against enteric is not readily established in some. I have before me the charts of a man who had not been inoculated, but who suffered from a 13-day attack of enteric fever in October, 1900. He was fit for duty early in November. At the end of February, 1901, he was again seized with a virulent form of the fever, and sank 26 days later from exhaustion. He suffered from delirium and diarrhoea throughout. After death, the intestinal ulcers were few in number and of no great depth or area.

TORTUOSITY OF BOTH INTERNAL CAROTID ARTERIES.

By
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WITH reference to the case published in the *BRITISH MEDICAL JOURNAL* of November 23rd, 1901, by Dr. Edington under the above heading, we forward the following notes of another case at present under our notice:

During the course of ordinary dissection in Guy's Hospital Medical School of the body of a female, aged 94, who is certified as having died from chronic nephritis, the internal carotid artery of either side was found to have a marked S-shaped bend in its course upwards. On the right side the common carotid artery bifurcated at the upper border of the

Diagram 1. Actual size.

thyroid cartilage, the commencement of the internal carotid showing a fusiform dilatation. Passing upwards in the carotid sheath to a point 5.5 cm. above its origin, the internal carotid suddenly turned forwards and inwards and then directly downwards for 2.25 cm., when it again turned forwards, inwards, and upwards to the carotid canal in petrous portion of the temporal bone. The second bend on the right side was distinctly sacculated, and the arterial wall was here very thin. The portion of the artery passing from the bend to the carotid canal was larger than normal.

On the left side there was an almost exactly similar bend in the internal carotid commencing 6 cm. above the bifurcation of the common carotid artery, with a maximum measurement between the convexities of each U-shaped piece of 2.5 cm. Just before the flexure on this side the artery was dilated and thin-walled (Diagram 2). The left external carotid of this side was also slightly tortuous. The flexures of each side lay beneath the external carotids, their posterior and inner surfaces lying on the rectus capitis anticus muscle, and, by examination by the finger in the pharynx, could be easily felt immediately behind the posterior pillar of the fauces. The arteries of the body generally are atheromatous, some presenting calcareous plaques in their wall, a few of which were present in the common carotid of each side. The external iliac arteries were tortuous, being curved internally so as to pass down on either side into the true pelvis; the left external iliac artery measured 14 cm.

Diagram 2.—The flexure in the left internal carotid. Actual size.

It seems probable that the tortuosity in this case is due to sacculation of the vessel wall consequent upon the arteritis and increased arterial pressure of the chronic nephritis, the increased length of the artery thus caused giving place to tortuosity, but the tortuosity is peculiar in its marked symmetry.

MEMORANDA: MEDICAL, SURGICAL, OBSTETRICAL, THERAPEUTICAL, PATHOLOGICAL, Etc.

CASE OF APOPLEXY DURING LABOUR.

ON November 15th, 1901, at 3 P.M., I was called to attend Mrs. J., a strong healthy primipara, aged 35. The pains were coming on regularly, the os was just beginning to dilate, and the parts were soft, relaxed, and moist. By 6 P.M. the os was fully dilated, the membranes ruptured, and the head, presenting in the first position, was making satisfactory progress in the pelvis. The pains were distinctly propulsive, but the straining was by no means excessive. She was in excellent spirits, although considerably annoyed with flatulence in the stomach and twinges of headache, which she said were not severe. Suddenly she exclaimed that her head was very bad, and that she could not get breath. She did not reply to my questions, and on going to the bedside I found her perfectly unconscious, in fact in a state of profound coma. The face was flushed, the pupils widely dilated, and the limbs limp and impassive; but the respiration, the pulse, and the temperature were still nearly normal. The uterine contractions went on, but there was no action of the accessory muscles, and the head receded away up from the brim. No time was lost in applying the forceps and effecting delivery, but this brought no abatement of the untoward symptoms. The respiration became stertorous and frequently obstructed, the pulse rapid, irregular, and weak, the temperature fluctuated from subnormal to 105°, and she died twelve hours after the seizure. There was no appearance of oedema, twitchings, convulsions, or other characteristics of eclampsia. No *post-*