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-*