vascular complication, while the relative low white blood cell count was an evidence of lowering of the body resistance at the time of the spread of the infectious process to the brain.

The rapid onset of meningitis in Case 2 cannot pass without notice. Perhaps the sequence of events is as follows:—

The thrombosis is primarily caused by pneumococcal toxin because of its special toxicity towards the vascular endothelium of the brain. As a result of the deficient blood supply the resistance of the part is lowered, together with that of brain of which it is a component part. The pneumococci circulating in the blood thus get an opportunity to attack the brain, culminating ultimately in the clinical picture of meningitis as in Case 2. If death occurred after cerebral thrombosis and before the onset of meningitis from some other cause, e.g., effects of the pneumococcal toxin on the heart muscle, actual meningitis may not develop though the symptoms of meningeal irritation may be present as in Case 1.

The hypothesis of vascular thrombosis previous to the attack of pneumococci would also explain the selection by remote organs in pneumococcal complications of pneumonia, which is now accepted to be a septicæmia from the beginning of the disease. So that other similar complications in pneumonia, like arthritis, endocarditis, etc., can also be explained on a similar hypothetical sequence of thrombosis leading to lowered resistance and consequent attack by the pneumococci circulating in the blood. More cases will be required to confirm the hypothesis which is suggested by the two cases quoted above.

The interest of the cases lies in the following facts:—

1. Thrombosis and hæmorrhage in the brain may occur in pneumonia without any obvious clinical symptoms.

2. Pneumococcal toxin appears to have a special toxicity towards the vascular endothelium of the brain.

3. In pneumonia vascular thrombosis and hæmorrhage in the brain may occur as a complication without meningitis as in Case 1, and perhaps precede meningitis when it complicates pneumonia as in Case 2.

4. It would appear that the susceptibility of the vascular endothelium of a particular organ for thrombosis under the influence of pneumococcal toxin may determine the occurrence of pneumoccal complication in that organ in pneumococcal septicæmia.

My sincere thanks are due to Dr. R. Row, Professor of Pathology, for the kind permission he has given me to report these cases, and to Professor Row and Dr. J. L. Saldanha for their help and guidance in writing this paper.

A CASE OF LATENT TYPHOID FEVER.

By CAPTAIN A. ROY CHOUDHURY, M.B. (Cal.), B.SC.,
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I WAS called in, on the 22nd March at 3 p.m., to see a patient having the history of passing a large black coloured stool about 15 minutes before, and of repeated vomiting.

The patient was a thinly built girl, aged about 13 years, looking extremely pale, and breathing quickly. She was fully conscious and complained of pain in the abdomen in general and in the umbilical region in particular. Her pulse was soft, thready and running at the rate of 130 per minute. Temperature 97° (oral). Tongue—slightly coated and dry. Extremities somewhat cold. Abdomen—stiff and tender. She would hardly allow me to touch a spot about an inch above and to the right side of the umbilicus. Spleen—just palpable; liver—not enlarged. On enquiry I found that the girl had not felt well and had no desire for food for about a week preceding the attack of hæmorrhage. There was no history of fever. She was on her usual diet up to the time I saw her.

There was no history, or any symptoms pointing to previous gastric or duodenal trouble. The stool was preserved for my inspection. It was a large tarry stool without any fæcal matter. Once she vomited in my presence. The vomited matter was composed of undigested food particles and mucus. An injection of 2 c.c. of 10 per cent. calcium chloride solution was given intravenously and an ice bag was ordered to be placed on abdomen. She was advised to suck small pieces of ice. An injection of morphia was given in the night at 10 p.m. The next day her blood was taken for the Widal reaction which was positive—1 in 100. The urine was also examined next day; there was no albumin. She had a motion after 48 hours as a result of a glycerin enema. The stool was small in amount and tarry. Subsequently she made an uneventful recovery.

Points of interest are (1) one of the dangerous complications appeared as the first symptoms in this case. (2) Difficulty in diagnosing the condition owing to the remarkable absence of symptoms and signs previous to the onset of the complication. (3) Effectiveness of intravenous injection of calcium chloride as a measure against internal hæmorrhage.

ELIMINATION OF URIC ACID VIA THE SKIN.

By J. L. SALDANHA, M.B., B.S. (*Bombay*).

A MALE, aged 60, cleric by profession, sought advice for troublesome and incessant pruritus of three years' standing, limited to dark-brown irregular patches of various sizes on his trunk and extremities. There were shining crystalline incrustations over these patches. The patient had been continually under the treatment of several qualified practitioners without finding any appreciable relief. He was unable to secure more than three hours' sleep at nights