(3) That there may be an utter absence of leucocytosis with marked perforation and peritonitis; in fact, that the cells may be lower than normal.

(4) That with typical signs of perforation and a definite leucocytosis there may be no such complication present, and an operation may be performed unnecessarily.

(5) That a marked degree of leucocytosis may occur in complications other than perforation, for example, bronchitis, cholecystitis, etc.

(6) That with pain and tenderness in the abdomen, coming on suddenly during an attack of typhoid fever (and in the absence of evidences of cholecystitis or other definite complication), and a distinct leucocytosis, even without other signs of perforation, an exploratory operation is justified, even advisable, thereby obviating the dangers of a fatal issue from too great a delay. The exploratory operation in Cases IV and V, done unnecessarily, resulted in no bad consequences, and the patients made a satisfactory recovery.

In conclusion I would like to record my indebtedness to Dr. C. F. Martin for assistance in carrying out my little investigation, and to the other clinicians of the hospitals for kind permission to use their material.

Clinical Department.

CONTUSION OF THE ABDOMEN; NO EXTERNAL WOUND; RUPTURE OF THE DESCENDING COLON; FECAL ABSCESS; DRAINAGE; SUTURE OF RUPTURED GUT; RECOVERY.

BY CHARLES L. SCUDDER, M.D., BOSTON.

J. H. M., thirty-six years old, was caught between a station platform and a moving railroad car. He was crushed. A few hours later he entered the accident room of the Massachusetts General Hospital, service of Dr. A. T. Cabot, in a condition of moderate shock. The temperature was 100°. Over the left iliac crest was an hematoma slightly tender and fluctuating. The urine was smoky in color. The white blood count was 10,200.

The first night he vomited three times, the vomitus consisting of the stomach contents. The next morning the abdomen was slightly distended. The third day all food by mouth was stopped on account of nausea. On the fifth day he had two fecal dejections. There was no more vomiting. On the sixth day the temperature was 101°. The abdomen was less distended. On the left side in the loin the hematoma was tender and fluctuating and the skin over the area was red. Under ether the swelling was opened and a large fecal abscess was evacuated. The cavity toward the abdominal side was bounded by the small intestine, descending colon and omentum. The muscles of the abdominal wall had been torn from their attachments to the iliac crest. At the extreme depth of the abscess was discovered an opening into the descending colon which admitted the tip of the

forefinger. The abscess, so well separated from the peritoneal cavity, was thoroughly evacuated and its wall cleansed with sterile water. Gauze drainage was used. After the sloughing portions of the wound had granulated, some eight weeks after the accident, an unsuccessful attempt was made to close the fistula. Ten days later a second attempt at closure succeeded. The edges of the fistulous opening were refreshed, all tension in the part concerned was removed and the gut was closed with silk sutures.

The particular interest in this case lies in the fact that the rupture of the bowel was not attended by such symptoms as would ordinarily lead one to suppose that a serious injury had been received.

A suppurating hematoma was evident. The rupture in the gut was concealed.

The very important sign that was misinterpreted was that of vomiting. This symptom, instead of being explained by the patient's statement that he had eaten something indigestible before he was injured, should have suggested a lesion of the bowel, or at least serious involvement of the peritoneum.

Vomiting once of stomach contents after an abdominal contusion is of no especial importance. Continuous, unexpected vomiting without any apparent reason is significant of an intestinal lesion. This is true in spite of the absence of other signs of involvement of the peritoneum.

During convalescence this patient had a pneumonia and a phlebitis of the left thigh and leg.

Medical Progress.

RECENT PROGRESS IN NEUROLOGY.

BY PHILIP COOMBS KNAPP, A.M., M.D., BOSTON.

THE INFECTIOUS AND TOXIC ORIGIN OF NERVOUS DISEASES.

At the tenth congress of the Società Freniatria Italiana in Naples last October, one of the subjects for formal discussion¹ was the intoxications and infections in the pathogenesis of mental diseases and neuropathies. This discussion was opened by D'Abundo, who maintained that infections and intoxications are the most frequent sensible and active factor in the pathogenesis of nervous diseases in general, in every period of life both intra-uterine and extra-uterine. The toxic infectious heredity (as syphilis or alcoholism) facilitates in the descendants the evolution of toxic-infectious neuropathies with a classical anatomico-pathological basis. Infections and intoxications in the progenitors, or in the mother during gestation, very often produce in the fetus very marked retardation of the processes of myelinization of the different systems of nervous connections. Some degenerative forms of neuropathy are often to be regarded as due to defective cerebral or spinal organization, the result of toxic pathological processes in the embryo which have been healed.

¹ Rivista Sperimentale di Freniatria, xxvi, 919, December, 1900.