for her age. Although she could not speak, like the lesional aphasics, she could hum tunes and tried to sing with words. The third case was a boy (G. B.) aged 2 years and 10 months, who has never spoken. He makes efforts to speak like a child stammering very badly. He is a well proportioned child for his age, but his mental condition is difficult to estimate. His mother considers him quite intelligent. He is right-handed, but comes of a left-handed stock—that is to say, his mother is right-handed but her brother and sister and her mother also are left-handed. In these cases suggestion of lack of cerebral correlation again arises. I had one case of a left-handed boy who only began to stammer when an attempt was made to make him a right-handed writer.

A further outcome of this inquiry into the histories of the non-rheumatic choreas has indicated another causal factor which must not be overlooked. The condition occurs in both sexes, but the majority of the cases recorded were girls at or about the period of puberty presenting, as so frequently happens, certain constitutional disturbances manifested by the complexion becoming bluish or heliotrope, the hands and feet cold and blue, the heart large and palpitating, the liver somewhat small, and the thyroid enlarging. Here we have an unestablished balance of the internal secretions and a tendency to nervous upset. Chorea is not uncommonly related to this condition, and the cases constitute a class by themselves—the climacteric choreas. As in the stock-brained cases, the disorder is often precipitated by emotional causes. In the one it results from a chemical or metabolic inco-ordination, in the other from an anatomical one.

I am indebted to Dr. Simonne Thompson and to my resident physician, Miss Norah Walker, M.B., Ch.B., for having taken much trouble in tracing the stock-brained histories of every case of chorea, whether rheumatic or otherwise. Since leaving the Royal Liverpool Children's Hospital at Heswall, Miss Walker has carried out an independent investigation and has collected a considerable number of cases which confirm the above findings.

ACUTE APPENDICITIS IN A CHILD COMMENCING WITH DIARRHOEA AND CONVULSIONS.

BY

BERNARD MYERS, C.M.G., M.D., M.R.C.P.,
PHYSICIAN TO IN-PATIENTS, ROYAL WATERLOO HOSPITAL.

THE case here recorded presents many points of interest. In the first place it had to be determined whether the child was suffering from meningitis with abdominal symptoms or from abdominal symptoms with meningismus, or possibly from two separate diseases. The one thing that seemed essential was to open the abdomen quickly and deal with the appendix. Presumably the very severe diarrhoea was an attempt on the part of the body to get rid of some of the toxin. The convulsive attacks also were apparently due to the same cause, for the operation seemed to produce an almost magical effect upon the appearance and condition of the child. Personally I have never seen an attack of appendicitis commencing with severe diarrhoea and convulsions, and I think that the case may be of interest to other clinicians; doubtless some surgeons have seen similar cases.

Maud A., aged 6, was admitted into one of my cots in the Royal Waterloo Hospital for operation on enlarged tonsils and adenoids. Four days after admission the child had a sudden very severe and continuous diarrhoea for fifteen minutes. The ward sister, who was with the child all the time, stated that she had never known a more severe one. For several days afterwards constipation followed. Shortly after the diarrhoea ceased general convulsions occurred; the whole body seemed to be affected, but especially the limbs and tongue. These attacks continued for four hours before they could be checked. There were twitchings of the lips, tongue, eyes, and limbs. The child at first appeared to be weaker on the right side of the body, but later the left side was noted to be weaker than the right. The pupils became dilated; the colour was first pallid, but later on the child became cyanotic. There was stated to be foaming at the mouth.

The urine examined during the attacks showed nothing abnormal. The child was given potassium bromide 10 grains and chloral hydrate $2\frac{1}{2}$ grains by the rectum soon after the convulsions began, but this had no result. Later 30 grains of the bromide and 5 grains of the chloral were injected, but ultimately chloroform

had to be administered to check the attacks. During the same night the attacks lessened and the child finally fell asleep, but later on was awakened by three or four attacks of vomiting. The vomit being of a greenish fluid nature.

The next day, when the child was examined, the tongue was very furred. The lips showed a small trauma from the application of the forceps to the tongue on the previous night. The child seemed to be in a semi-comatose condition. Vomiting continued. The bowels had not acted since the initial short attack of diarrhoea. Urine showed the presence of acetone, which possibly was due to the administration of chloroform during the attacks.

The child complained of pain in the upper part of the abdomen, and there was tenderness in that region. No tenderness was elicited by Mr. Cockell, my house-physician, at this period in either iliac fossa, but when he examined the child later in the day there seemed to be some tenderness in the right iliac fossa, which appeared to be a little more noticeable over McBurney's point than anywhere else in this area, compared with the left side. I saw the case a few hours later on the same day, and the child still appeared to be semi-comatose. The pupils were markedly dilated, the left perhaps more so than the right. Ophthalmoscopic examination was not made. Photophobia was present. No stiffness of the neck was elicited, nor discharge from the ears, nose, or throat. The tongue was dry and furred. The tonsils were distinctly enlarged, and very slightly red on the surface. There was slight stiffness of the left arm and left leg compared with the right side, and the left knee-jerk was also a little brisker than the right. On the left side there was slight ankle-clonus. Babinski's sign was not obtained. Kernig's sign was just present on the left side on this occasion only. The abdomen was retracted; tenderness was evident in the right iliac fossa in the region of McBurney's point. Resistance was noted. The note over the right iliac fossa was slightly impaired upon light percussion; there was no discharge from the vagina. Examination by the rectum disclosed nothing definite. The heart and lungs appeared to be normal.

The diagnosis of appendicitis was made, and Mr. Cairns Forsyth came down to the hospital and operated immediately. The appendix was found to be very long, distinctly distended, and acutely inflamed. It was filled with hardened faeces. A large number of shotty glands were seen in the mesentery. The child made an uninterrupted recovery. No further diarrhoea occurred, and the mental state cleared up wonderfully immediately the child came round after the operation. There were no more convulsions, and all the limbs appeared to be quite normal next day.

British Medical Association.

PROCEEDINGS OF SECTIONS AT THE ANNUAL MEETING, BRADFORD, 1924.

SECTION OF SURGERY.

Sir CUTHBERT WALLACE, K.C.M.G., C.B., F.R.C.S.,
President.

DISCUSSION ON POST-OPERATIVE PULMONARY EMBOLISM.

OPENING PAPER

BY

P. LOCKHART-MUMMERY, F.R.C.S.,

Senior Surgeon, St. Mark's Hospital for Cancer, Fistula, and other Diseases of the Rectum.

PULMONARY embolism may occur as the result of many different morbid conditions, but the most important, and that to which I shall chiefly confine my remarks, is as the after-result of an operation. Pulmonary embolism is by no means always fatal, and although no really reliable statistics on this point are available I should estimate that not more than one out of every ten cases of pulmonary embolism causes the death of the patient; in the remainder the patient recovers. There seems little doubt that most of the cases of so-called post-operative pneumonia are in reality cases of pulmonary embolism.

Pulmonary embolism may occur at any period after operation up to several weeks. The most common time is about the eighth or ninth day, but it has occurred within twenty-four hours of an operation, and in one case eight weeks later. Statistics are difficult to obtain, as the cases are comparatively infrequent and careful tables are not kept at most hospitals which will allow of the figures being