to some of these different serological types, but actually retain the names that have been applied to them, such as *Spirochaeta* (*Leptospira*) *autumnalis* (p. 312), *hebdomadis* (p. 313), and *pyrogenes* (p. 314). Moreover, I conclude with the statement (p. 314) " that Fletcher isolated six serologically distinct groups of spirochaetes from patients in Malaya, all showing very similar clinical symptoms.—I am, etc.,

London, N.W.3, Aug. 15th. EDWARD HINDLE.

We have submitted the above letter to our reviewer, who replies as follows:

In his excellent article on the leptospiroses Dr. Hindle makes it perfectly clear that the pathogenic leptospira comprise a variety of serological types and that, in fact, by their serological reactions alone can they be distinguished from one another. The remarks to which Dr. Hindle takes exception were made in view of the work of Taylor and Goyle on epidemic jaundice in the Andaman Islands. The condition investigated by them and referred to as Weil's disease possessed all the clinical manifestations and post-mortem appearances of that disease. Further, its epidemiology was that of Weil's disease, in that the cases occurred principally in the wet season—the south-west monsoon—and were confined exclusively to a certain class of individuals—namely, adult males whose occupation entailed wet working conditions. The twenty-eight strains of leptospira isolated by Taylor and Goyle in the Andamans were compared serologically with a representative collection of leptospira from different parts of the world, including *S. icterohaemorrhagiae*. Not one of the twenty-eight strains corresponded serologically with *S. icterohaemorrhagiae* ; twenty-four of them differed apparently from any known strain, although forming a homogeneous serological group themselves ; the remaining four were serologically related to *S. autumnalis* (Akiyami Type A). These workers further claimed that, from their examination of the various strains of leptospira by cross-agglutination, *S. icterohaemorrhagiae* could be divided into two groups, which they designated A and B. A point overlooked when writing the review was that Dr. Hindle's article had probably gone to press before Goyle and Taylor's work appeared, in which case the criticism of his article was not valid, and an apology is due to him. He might, of course, contend that, since the leptospira isolated by Taylor and Goyle were not serologically identical with *S. icterohaemorrhagiae*, the condition they were investigating was not Weil's disease. It is with considerable hesitation that one questions the opinion of Dr. Hindle on this subject, of which he is an acknowledged authority, but it does seem that narrowing the definition of Weil's disease in this way would be almost comparable to confining the term lobar pneumonia to those pneumonias caused by the Type I pneumococcus.

CONVULSIONS DURING ETHER ANAESTHESIA

SIR,—The following case occurred at Victoria Hospital, Burnley, on August 5th.

A schoolboy, aged 11, was operated upon by Mr. Callam for a gangrenous pelvic appendix, with abscess. The condition began about sixty hours before admission. He had on admission a temperature of 103° F., a pulse rate of 120, rigidity of the lower abdomen, and marked toxaemia.

Before the operation he was given 1/150 grain of atropine sulphate subcutaneously. Anaesthesia was induced by C.E. mixture, followed by open ether, and continued with ether and oxygen by Shipway's apparatus. The anaesthesia was uneventful till the operation had been in progress for about forty-five minutes, when convulsions began just as the operator was beginning to close the wound. The convulsions were first noticed in the lower extremities, and became generalized. The pupils were widely dilated and the conjunctiva was insensitive. There was no clonus of the lower jaw, the tongue being rhythmically protruded and withdrawn. There was no cyanosis. Chloroform and oxygen were then given. The convulsions diminished in severity and ceased in about seven minutes—by the time the operation was concluded. On return to bed he recovered normally from the anaesthetic and there was no recurrence of the convulsions. There was no history of epilepsy or of any previous fit, though the boy was of a highly strung, nervous type.

The ether used was Duncan's " anaesthetic ether," which has been in general use in this hospital for years. The same stock bottle had been used just previously, and was used in the next case satisfactorily. Analysis revealed neither ethyl sulphate, ether peroxide, acetaldehyde, nor other impurity. The absence of impurity in the actual sample used, and the fact that the same brand of ether has been in general use daily for years without a previous case occurring, seems to disprove the theory that the condition is due to impurities in the ether.

One is inclined to think that toxaemia and instability of the nervous system are the most probable causes of the condition in this case, and seem to be the commonest factors in previously reported cases.

I have to thank Mr. Lawrie for his analysis of the ether.—I am, etc.,

JAMES HAWORTH,
Senior Anaesthetist.

Victoria Hospital, Burnley, Aug. 17th.

SIR,—The cause of ether convulsions appears to remain a mystery. During my small experience I have made careful observations which may assist in providing a clue.

Toxaemic patients seem most prone to convulsions, and then only if oxygen is given with the ether. Convulsions apart from oxygen are probably clonus. Toxaemic cases commonly met with are appendices in the gangrenous stage, osteomyelitis, salpingitis, infected compound fractures, etc.

An explanation of convulsions on a physiological basis is beyond my powers ; however, the sequence of events leading to convulsions is easy to follow. The induction of a toxaemic case is of the easiest, and deep anaesthesia is quickly attained. Deep anaesthesia, in spite of a perfect airway, causes cyanosis, and it is at this point that mistakes are made and convulsions occur. Oxygen is promptly given and the signs of convulsions soon become apparent.

Convulsions can be avoided by inducing toxaemic cases with caution. Provided the airway is clear, meet any cyanosis by reducing the dose of ether and by removing the mask if necessary. If oxygen seems essential, give the smallest quantity and remove as soon as the colour is normal. In the treatment of convulsions remove the oxygen at once. If cyanosed, remove the mask as well, and replace when the colour is normal. The convulsions will cease in a time approximately equal to their duration. —I am, etc.,

C. J. BASHALL, M.R.C.S., L.R.C.P.

Marlborough, Aug. 18th.

CARCINOMA OF THE OESOPHAGUS

SIR,—On reading the article by Mr. Woodman in the *Journal* of August 15th on the treatment of carcinoma of the oesophagus, I was struck by his omission of simple dilatation from the methods he discussed. Leaving radical extirpation out of the question of the treatment of this condition, we are faced with palliative measures consisting of radium, *x* rays, diathermy, intubation, gastrostomy, and dilatation. Since one of them is to be chosen, it seems to me that the best method is one