it appears to me no more ridiculous than many of the clinical pictures which one sees from time to time ascribed to syphilis on no other grounds than a positive Wassermann reaction in the blood.

Dr. Elliott says in his article that Mrs. Z. (the daughter of the original case) had no clinical signs of the disease, yet she was submitted to a full course of anti-syphilitic treatment. This does not appear to have altered her serum reaction. Her offspring also had no clinical signs of the disease, but had a positive reaction which was rendered negative by treatment.

In 1928, in a paper in the *Practitioner*, I drew attention to the two types of case represented by these two reactions: one which is serologically "fast," and the other which I called the "pendulum" type because the serum reaction can be made to alternate between positive and negative by treatment. It appears to me that there is a danger of over-emphasizing the value of a Wassermann reaction in late cases, and especially in congenital cases, where I have seen a positive reaction in a patient of 34 become negative *without* treatment.

I feel that Dr. Elliott's interesting paper really referred to a case of third-generation seropositive Wassermann reaction. The change in the blood proteins which is the basis of the Wassermann reaction may quite well be such as to be more or less fixed for that individual, and I see no reason against the further possibility that such a protein pattern may be directly transmissible through the placental membrane. This appears more likely than that a living spirochaete can exist through three generations, and in the last one invade the ovum.

The whole question of the transmissibility of syphilis is the most fascinating problem of this protean disease, and if each observer would go to the trouble of recording cases as carefully as Dr. Elliott has done some of the existing dubiety might be cleared up.—I am, etc.,

Sheffield, June 13th. E. FRETSON SKINNER.

Complete Transposition of the Viscera

SIR,—I am anxious to obtain information about this condition, which I believe to be inherited as a Mendelian recessive, and shall be much obliged if anyone who sees a case will send me the following data. Are the parents blood relations or not? If they are blood relations, are they first cousins, and, if not first cousins, what is their exact relationship? I should also like to know the sex of the patient, the number and sex of the normal brothers and sisters, whether the parents are normal, and whether there is a history of a previous case in the family. —I am, etc.,

E. A. COCKAYNE.

91A, Harley Street, London, W.1, June 11th.

Percentage of Alcohol in the Blood

SIR,—Dr. H. Kenneth V. Soltau (*Journal*, June 13th, p. 1231) states: "I believe that the more the problem is investigated the more will it be found that behaviour differs because of the variation of the blood-alcohol content." From this he would appear to agree with me that, so far, it has not been proved that incapacity in alcoholism varies with the alcoholic content of the blood. If this is so, I fail to see how a blood test can be considered conclusive, as he has suggested.

There is a further objection to the use of blood tests in the case of alcoholic drivers. It has been stated that a percentage of alcohol in the blood of 0.1, or over, should be accepted as conclusive evidence of unfitness, but it also has been stated that some people with lower percentages, even as low as 0.088, have been found to be "under the influence, etc." Are those cases with a blood-alcoholic content of less than 0.1 per cent. who are clinically unfit to drive a car not to be dealt with? That some persons with a blood-alcohol content of 0.08 per cent. have been found to be "under the influence, etc.," and others with the same percentage have not been so found is indication of the uselessness of the test for the purposes of the law.—I am, etc.,

London, N.W.8, June 13th. WM. M. FAIRLIE.

Familial Haemoptysis of Unusual Aetiology

SIR,—Rarely one meets with instances of haemoptysis the origin or aetiology of which is quite obscure. In such cases tuberculosis, mitral stenosis, malignancy, purpura haemorrhagica (Rivière, Lusitanus, Werlhof), haemophilia, thrombasthenia (Glanzmann), or constitutional thrombopathy (Jürgens and Willebrand), leukaemia, hepatic cirrhosis, etc., are readily excluded.

The occurrence recently of attacks of haemoptysis in two patients—members of a family previously reported by me as instances of "heredofamilial angiomatosis with recurring haemorrhages (Goldstein)" or Rendu-Osler-Weber's disease [*Arch. Int. Med.*, xlviii, Part I, 836; *Trans. Amer. Therap. Soc.*, 1932, 47; *La Riforma Medica*, lii, 256; *British Medical Journal*, 1936, i, 721 (family tree with eighteen affected individuals); *Arch. Derm. and Syph.*, xxvi, 282]—leads me to make these few comments on familial haemoptysis.

Similar cases of haemoptysis have been previously noted by R. J. Graves (*System of Clinical Medicine*, p. 268, 1843, Dublin, Fannin and Co.), Charles Elam (*A Physician's Problems*, p. 28, 1869, London, Macmillan and Co.), Antonio Lombardi (*Il Tommasi*, Naples, iv, 202), P. Ste. Marie (*La Revue Médicale du Canada* (Montreal), viii, 34), E. Libman and R. Ottenberg (*Journ. Amer. Med. Assoc.*, lxxxi, 2030), A. Arrak (1923, Dorpat), H. Mantchik (*Schweiz. med. Woch.*, lii, 949), and V. Cordier, P. Lagèze, and P. L. Mounier-Kuhn (*Lyon Méd.*, clvi, 51).

K. Ullmann of Vienna mentions (in reporting his first case in 1899) haemoptysis as occurring in his case, a woman with angiomatosis who was first seen in Hebra's clinic in 1892, and who died in 1899 from an attack of pneumonia. She had telangiectases on the face, and on the tracheal and bronchial mucous membrane, which bled. Similar instances have been noted by T. FitzHugh, jun. (Philadelphia), Aubertin and Lévy (Paris), H. I. Goldstein (Camden), and others.

Kofler's (*Wien. klin. Woch.*, xxi, 570) patient had telangiectases on the face, lips, nose, tongue, palate, posterior wall of pharynx, and the arytenoid cartilage. Kofler states that haemorrhages from the lesions on the posterior wall of the pharynx and in the larynx might be confused with true haemoptysis.

These instances of familial haemoptysis are probably similar in aetiology to the cases of familial haematuria ("renal haemophilia") reported by H. Senator (1891, 1896), Atlee (1901), Guthrie (1902), Pearson (1904), Apert (1907), Aitken (1909), Kendall and Hurst (1912), Hurst (1923), Barford (1926), and W. E. Foggie (1928), and much like the gastric haemorrhages noted by W. H. White (1912, *Gastrostaxis*), K. Ullmann (1896, 1900), L. N. Boston (*Amer. Journ. Med. Sci.*, clxxx, 798), M. S. Ersner (1930), Guttmann, Laval, and Schlumberger (1932, Paris), Ed. Benhamou (*Les Gastrorrhagies*, December 16th, 1933), Leo Kessel (New York, *Journ. Amer. Med. Assoc.*, xcvii, 1058), and others.—I am, etc.,

HYMAN I. GOLDSTEIN, M.D.

Camden, New Jersey, U.S.A., May 30th.