be reconciled with the statements of Felix and his co-workers on the important role of *Vi* antibody, and its antigenic counterpart, in typhoid immunity and infection; in fact, this antibody forms the principal constituent of Felix's therapeutic anti-typhoid serum. Further experiments were, therefore, undertaken which revealed that when a convalescent serum was injected into human beings, as well as laboratory animals, it led to the production of an appreciable amount of *Vi* antibody and that its therapeutic efficacy was directly proportional to the amount of antibody so produced. These observations could only be explained by assuming that the convalescent serum either stimulated the antibody-forming apparatus in some unknown manner or that it contained an antigen responsible for *Vi* antibody formation. It has now been ascertained that the latter explanation is correct and the typho-precipitin reaction reported here is based on the presence of a *Vi* precipitinogen in the sera of typhoid patients both during infection and during convalescence. The qualitative and quantitative determination of this antigen has been found to be of definite significance in the diagnosis, the prognosis and the treatment of typhoid fever.

Five capillary drops of a *pure Vi* serum (titre 250–500) were measured in each one of a row of precipitin tubes with a capillary pipette (56 drops to 1 c.c.). To these were added equally large five drops of a serially diluted serum under test for its *Vi* precipitinogen content. The mixture, after being well shaken, was incubated at 37° C. for one hour. It was then left in the refrigerator overnight and the reading taken after an interval of 20–24 hours. The development of a deposit and not an opalescence was the criterion of a positive test.

No *Vi* precipitating antigen was found to be present in the sera of typhoid patients during the early stage of disease, especially during stadium incrementi. It could, however, be detected towards the end of stadium fastigium and continued to be present in considerable quantities for 2–3 weeks after the temperature had stayed normal. A large number of normal sera examined did not show its presence nor could it be detected in infections other than typhoid fever. A previous history of *T.A.B.* inoculation did not affect the diagnostic value of the test. Any evidence of flocculation was proof positive that the patient was either suffering or had just suffered from typhoid fever, especially in cases where the isolation of the infecting organism had been unsuccessful and the results of other serological reactions only suggestive of typhoid infection.

From the prognostic point of view the presence of this antigen strongly indicated that the patient was getting the better of infection. In cases complicated with toxæmia, hyper or prolonged pyrexia, abdominal or meningeal involvement and those who relapsed, the *Vi* precipitinogen was either found to be absent or present only in traces at irregular intervals. A fair idea could thus be obtained as to where serological interference with convalescent serum was necessary in cases of typhoid fever.

In the absence of any method by which the potency of a therapeutic convalescent typhoid serum could be assessed, the application of the typho-precipitin reaction gave valuable information as to the suitability or otherwise of the serum of a particular convalescent, the period of convalescence at which the most potent serum was available and its dosage in different types of complications.

Since these experiments proved that active rather than passive immunization resulted from this form of serum therapy, convalescent serum was tried as a prophylactic antityphoid measure. No negative phase was observed, the *Vi* antibody forming within a period of 48 hours. There was no rise of temperature and no localized or generalized after-effects. In view of the general experience that *T.A.B.* vaccine inoculation gives rise to very little *Vi* antibody formation, these results can be considered to be encouraging from the immunological point of view.

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Demonstration of the *Rh*-Factor in the Blood of a 48-mm. Embryo

Landsteiner and Wiener[1] described a new antigenic factor, *Rh*, present in 85 per cent of human erythrocytes. This factor they detected by means of agglutination reactions with a suitably absorbed serum obtained by immunizing rabbits and later guinea pigs with *Macacus rhesus* cells.

Human sera containing anti-*Rh* agglutinins and mainly obtained from mothers giving birth to children affected by hæmolytic disease may also be used as typing reagents.

These latter are more suitable since in certain cases they are very potent and obtainable in relatively large quantities. Typing of adult cells with such sera gives clear-cut agglutination reactions.

It was found that the blood of a 48-mm. embryo approximately eleven weeks old could readily be typed (see accompanying table). The agglutination reaction between this blood and potent anti-*A* immune rabbit serum* was very weak, and this relative difference between the strength of the reaction with this serum and human anti-*Rh* sera may indicate that the factor *Rh* develops relatively early in fœtal life.

TITRATIONS OF FŒTAL CELLS WITH HUMAN ANTI-*Rh* SERA AT 37° C.

	Serum				
Cells	1	2	3	4	Anti-*A*
Fœtal	1 : 16	1 : 32	1 : 1	Neg.	1 : 1
Adult	1 : 512	1 : 512	1 : 8	1 : 16	1 : 128

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* I wish to thank Dr. Langley for preparing the rabbit serum.

[1] Landsteiner, K., and Wiener, A. S., *Proc. Soc. Exp. Biol. and Med.*, 43, 223 (1940).

Infection of Laboratory Animals with Johne's Disease and Leprosy

ACCORDING to Francis[1], "until recently it was generally accepted that laboratory animals had not been infected with *Myco. leprœ* or with *Myco. johnei*". As regards the latter organism, in 1913 five out of twenty-three rabbits, six out of forty mice and three out of twelve rats were successfully infected[2], the