

These results show that antigen persists in the human being after the serum sickness has subsided. This fact is in harmony with animal experiments. They show that anaphylactic antibody is formed as a feature of serum sickness.

The coëxistence of these factors in guinea-pigs similarly treated, I have demonstrated in previous experiments. Neither factor had as yet been demonstrated in human serum sickness. The facts indicate that the disease is due to the interaction of these factors, in accordance with an hypothesis suggested by Pirquet.

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Cellular processes in the latent period.

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Guinea-pigs were passively sensitized by the injection of the serum of a rabbit highly immunized to horse serum. Before the latent period had expired, *i. e.*, during the first 12 hours, the guinea-pig was killed, and the horns of the uteri were suspended for graphic tracing. One horn was immediately tested by means of horse serum. If it gave no response, the preparation was at once thoroughly washed, and the experiment continued. Both horns were kept in Locke's fluid for several hours. At the end of this time, both were again tested against horse serum. Both regularly responded with contractions, but that yielded by the previously tested horn was much less vigorous than by the other. The latter fact shows that the preliminary test by horse serum had partially desensitized the antibodies.

The following conclusions are drawn:

1. The cells absorb antibody from the blood during the first stage of the latent period. These antibodies can unite with the antigen, and the cells can thus be desensitized, but that this reaction produces no cellular contraction in the sensitized muscle cells.

2. The cells "activate" the absorbed antibody during the

second stage of the latent period, in such wise that the reaction with antigen produces a cellular stimulus, with muscular contraction in case of the uterus. It is for this reason, that the combination of antigen and antibody in the blood never produces an anaphylactic response.

The "activation" by the cells also greatly increases the avidity of cellular antibody for antigen, as has been shown in previous papers. Exactly the same features differentiate cellular from circulating antibodies after active sensitization.

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A test for antithrombin in the blood.

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The method of testing for antithrombin in the blood is at present very difficult, as it requires the preparation of a pure fibrinogen containing no prothrombin, that is to say, which does not clot upon the addition of calcium, and also the preparation of a pure thrombin which can be made from fibrin. As a result the method is hardly adaptable to general clinical use.

For some time I have employed a method which seems to meet this difficulty. For this purpose about 9 c.c. of blood are aspirated and put into 1 c.c. of 1 per cent. sodium oxalate. The blood is centrifugalized and the plasma siphoned off in the usual way. The plasma is then recalcified by adding 2, 3, 4 and 5 drops respectively of a $\frac{1}{2}$ per cent. calcium chloride solution. In this way we ascertain the general coagulability of the plasma which is the composite of a number of factors,—prothrombin, fibrinogen and antithrombin, and we determine the optimal amount of calcium for this particular plasma. If we heat some of this plasma to 60° C., the prothrombin, as is well known, is destroyed and the fibrinogen is coagulated. After filtering off this coagulum, we have a plasma which contains antithrombin. The strength of this antithrombin may be ascertained for clinical purposes as follows: