coccus toxin, the method we have used for preparation of the medium is given in detail.

Two pounds of chopped veal is placed in one liter of distilled water and left in the ice box over night. The infusion is then strained through cheese cloth, boiled, filtered and the reaction adjusted to pH 7.4. After autoclaving, the infusion is inoculated with *B. coli* and incubated for 18 hours. The broth is then sterilized in the Arnold, filtered, the volume made up to one liter, and 80 grams of "proteose peptone" (Digestive Ferments Co.) and 10 grams of NaCl are added. The whole is boiled in a double boiler for 40 minutes, filtered, the volume brought to one liter and the pH adjusted to 7.4, one liter of phosphate buffer [N/15 ($Na_2H\ PO_4$ and $KH_2\ PO_4$)] pH 7.4 is added, the media is distributed in Ehlermeyer flasks and autoclaved.

On this media in the presence of 8 to 10 per cent CO_2, the strongest toxin is obtained after 6 to 8 days' growth of the organism.

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The occurrence of scarlet fever without a rash during epidemic.

FRANKLIN A. STEVENS

[*From the Department of Medicine of the College of Physicians and Surgeons of Columbia University and the Presbyterian Hospital, New York.*]

During the winter of 1924-25 an epidemic of scarlet fever and acute streptococcus pharyngitis occurred among the nurses at Presbyterian Hospital, New York City. Early in the epidemic the entire nursing staff was tested for susceptibility to scarlet fever by means of intracutaneous toxin injections. Following this series of skin tests cultures were obtained from all throats showing an angina. Practically all of the cases of pharyngitis and tonsilitis showed hemolytic streptococci. The strains of hemolytic streptococci recovered from these cases were tested for agglutination with scarlatinal immune sera and with sera pre-

pared with two of the throat strains proven not to be scarlatinal strains. Filtrates were prepared from cultures of all these strains and tested for the presence of toxin by means of cutaneous reactions in Dick positive individuals. The presence of toxin was determined by heat lability (100 degrees C. for 2 hours), and by neutralization with scarlatinal antitoxic sera.

Twenty-three strains of hemolytic streptococci were studied. Six strains, which were obtained from cases of clinical scarlet fever, produced a heat labile toxin which was neutralized by anti-scarlatinal sera. Five of the strains were agglutinated by two scarlatinal immune sera, and the sixth, although it did not agglutinate, absorbed the agglutinin from these sera for other scarlatinal strains. This strain was apparently physically incapable of agglutination, yet was similar antigenically to the other scarlatinal strains. These strains (six) include all the strains from clinical scarlet fever.

The remaining seventeen strains were obtained from cases of pharyngitis and tonsilitis in which no rash occurred. Five of these strains produced toxin which was heat labile and was neutralized by scarlatinal antitoxin. As far as biologic characteristics are concerned these strains are scarlatinal strains, since they produced toxin and likewise were found to agglutinate and absorb agglutinin with the two scarlatinal immune sera. Four of the individuals from whom the strains were obtained were Dick negative previous to their throat infection. The fifth had not been tested.

The majority of the remaining twelve strains (9) agglutinated in immune sera prepared with non-scarlatinal strains. There were apparently two groups of strains represented by these sera as well as some additional groups, but the reactions were not strongly positive with these sera so we can only conclude that the strains were not antigenically similar. Apparently the strains from these cases were not bound as closely together as were the strains from the cases of scarlatina. One strain produced a heat labile toxin which could not be neutralized.

We conclude that throat infections with scarlatinal streptococci may occur without a rash. In this epidemic they occurred in individuals who showed negative Dick reactions and were presumably immune. In general, toxin production and agglutination of scarlatinal strains were parallel. Considering the agglutination reactions with all of the sera and all of the strains we conclude

also, that no antigenic likenesses exist between strains of streptococci from acute throat infections similar to that observed between scarlatinal strains.

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Unusual instances of infection with streptococcus scarlatinae.

FRANKLIN A. STEVENS.

[*From the Department of Medicine of the College of Physicians and Surgeons of Columbia University and the Presbyterian Hospital, New York.*]

In the preceding study we noted that infections of the throat with scarlatinal streptococci may occur during an epidemic of scarlet fever, and that these infections are not accompanied by a rash. There is also clinical and epidemiological evidence that these cases may be responsible for cases of clinical scarlet fever among contacts. Williams has recently found this streptococcus in osteomyelitis, endocarditis and in chronically inflamed tonsils, so that we may be assured that *Streptococcus scarlatinæ* is a rather widely distributed organism and is not confined in its distribution to the cases diagnosed as clinical scarlet fever.

We know little concerning the clinical manifestations of *Streptococcus scarlatinæ* in conditions other than the usual angina with the scarlatina-form rash because the methods of identification are so recent that few observations have accumulated. Some of the infections are associated with a rash as in wound scarlet fever, yet we know that the infection may take place without cutaneous manifestations. The original strain for the toxin made by Dick and Dick was obtained from the infected finger of a nurse caring for a scarlet fever patient. In this instance no rash was reported.

The study of such atypical infections is important from the epidermiologic standpoint. Unfortunately, there is but little opportunity to study such cases in infectious hospitals because they are seldom recognized unless they occur among known scarlatinal contacts, or unless they are uncovered in an attempt to account