The employment of solutions of chloramine-T to remove meningococci and other pathogenic microorganisms from the nasopharynx suggested to us its trial in the treatment of experimental pneumonia. Lobar pneumonia was produced in dogs by means of intrabronchial insufflation of a broth culture of a highly virulent Pneumococcus Type I, invariably leading to a fatal termination. The dose was 2.5 cc. of an 18 to 24 hour culture per kilo of body weight. The autopsy on untreated control animals, which usually succumbed within 48 hours, showed a lobar consolidation in the stage of red hepatization of one lobe of the lung, commonly the lower right lobe. The lower left lobe was less frequently affected, and the subcardiac lobe was only occasionally involved, together with one other. The lesions were microscopically typical of experimental lobar pneumonia in the dog. The pneumococcus insufflated was recovered in pure culture from the solid lung area and the blood of the heart.

The next step was to apply the chloramine-T solution to the inoculated lung. The plan was to introduce 5 cc. per kilo of body weight of a 1:10,000 solution intrabronchially 2 hours after the culture was given, and if the animal survived until the next day or longer, to repeat the injection.

Six animals with the experimentally induced pneumonia were given the chloramine-T solution; they were controlled by three untreated dogs. The results can be briefly stated. One animal

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received four, another three, and two others two treatments, while two died after the first dose.

Instead of a therapeutic action, the effects of the treatment were rather to intensify the pathological process. The consolidated areas involved more than one lobe and were attended by much greater edema and hemorrhage than were present in the controls. The animal which survived longest (2 days) and received four injections of the chemical, showed pus in both pleural cavities. Moreover, death resulted, as a rule, more quickly in the treated than in the control animals.

This action of the chloramine-T on the infected lungs led us to a study of the effects of this chemical on lungs of normal dogs. The quantity insufflated was 5 cc. per kilo of a 1:10,000 solution of chloramine-T in normal saline solution. The animals were killed with chloroform at intervals varying from 20 hours to 30 days. One dog of the series died on the 2nd day.

After 24 hours the injected lungs showed areas of consolidation. Usually one lobe was affected, but sometimes two or three lobes were involved (Fig. 1). The lesion proved to be bronchopneumonia, attended by marked congestion and edema. The microscope showed that the alveoli contained red corpuscles, epithelial cells, and polymorphonuclear leukocytes. Very little fibrin was present. The capillaries and larger blood vessels were congested and an excess of leukocytes invaded the alveolar walls.

The 48 hour lesion was practically identical with the preceding (Fig. 2). The 72 hour lesions showed less congestion and beginning resolution. The 4 day specimens were more advanced in resolution, while the 7 day specimens were no longer solid and exhibited only decolorized areas to indicate the seat of the previous consolidation. Two dogs were killed with chloroform on the 30th day. They presented a small wedge of organizing (unresolved) pneumonia in the lower right lobe, possibly a residue from the injections. The chloramine-T pneumonia did not cause fever or any appearance of illness in the animals.

A series of experiments was also made with Dakin's hypochlorite of sodium solution (0.48 per cent) in insufflations of 5 cc. per kilo of body weight. The lung lesions produced by insufflation of this
solution were similar to those produced by the chloramine-T solution. They ran about the same course and resolution was apparent on the 3rd day.

SUMMARY AND CONCLUSIONS.

It is obvious from the experiments reported that concentrations of chloramine-T solutions, even more dilute than those which are well borne by the nasopharyngeal mucosa, are injurious to the pulmonary tissue when introduced directly into the bronchi in large volume. The pulmonary lesions produced are of the nature of an extensive bronchopneumonia which progresses during the first 2 days after injection of the chemical and then regresses, to disappear, as a rule, by the 7th day. Similar pulmonary lesions were produced by intra-bronchial insufflation of Dakin's solution of hypochlorite of sodium.

These studies taken in conjunction with the earlier ones of Kline and Meltzer, in which aleuronat, starch, egg yolk, and lecithin were insufflated into the lungs, show that pulmonary inflammation may be induced by various chemical substances—with the following differences. Aleuronat and starch set up consolidations containing much fibrin, as is the case with virulent pneumococci; egg yolk and lecithin gave lesions with little fibrin, as is the case with avirulent pneumococci. Both series of substances produced lobar pneumonia, while the pulmonary lesions induced by chloramine-T and sodium hypochlorite were of the nature of bronchopneumonia.

The consolidations of the lung produced by chemical substances differ from infectious pulmonary inflammations only in their sterility. These experimental results strongly suggest the view that the anatomical findings in pneumonia represent a part of a mechanism of defense and repair which the animal body creates in its struggle against infection and intoxication.

EXPLANATION OF PLATES.

PLATE 55.

Fig. 1. Lungs 24 hours after intrabronchial injection of 1:10,000 chloramine-T solution. Dose 5 cc. per kilo of body weight. Right lower lobe swollen, heavy, and solid. Areas of congestion in right upper, and middle lobes. Left lung well aerated.

PLATE 56.

Fig. 2. Section of consolidated lung on the 2nd day after the intrabronchial injection of 1:10,000 chloramine-T solution. Dose 5 cc. per kilo of body weight. The intraalveolar exudate consists of epithelial cells, polynuclear leukocytes, and erythrocytes. The alveolar walls are infiltrated with polynuclear cells.
FIG. 1.

(Wollstein and Meltzer: Experimental chemical pneumonia.)
Fig. 2.

(Wollstein and Meltzer: Experimental chemical pneumonia.)