The similarity of the infections in the 3 subjects makes the results of the study most interesting. The cholesterol values were already subnormal before the specific serum was administered. The response to the single dose of serum was a drop in the temperature in each instance to normal within 12 to 24 hours, and one day later when the second blood sample was collected the total cholesterol and esters had already returned to the normal range. In case 2 the rise was not rapid and in this child the roentgenogram revealed a slight increase in the extent of the pneumonic process in the lung. However, after the fourth day of convalescence, all cholesterol values were normal and the roentgenograms showed complete resolution of the pneumonia.

The total fatty acids and the phospholipids were slightly depressed before the administration of serum. Immediately following the fall in temperature, there was a rapid increase in the total fatty acids while the phospholipids rose more slowly. The iodine numbers of the total fatty acids and of the phospholipid fatty acids underwent very little change for the prompt response to serum therapy apparently prevented any marked drop. The intensity of the fat metabolism during pneumonia and the presence of bacterial toxins may be responsible for the fall in the plasma lipids to abnormal levels. The early use of a specific serum tends to control this altered lipid metabolism which is present in acute infections such as pneumonia.

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Lymph Flow from the Heart-Lung Preparation During Pulmonary Edema.

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The absence of demonstrable lymphatics beyond the alveolar ducts[1] suggests a factor in the rapid development frequently characterizing pulmonary edema. In the following experiments this anatomical fact was put to a functional test.

In 4 dogs under nembutal anesthesia the thoracic duct was cannulated one centimeter from the point of its entrance into the left subclavian vein. Following this, a Starling-Knowleton heart-lung preparation was made, and the thoracic duct tied off above the dia-

[1] Maximow, A. A., and Bloom, W., *A Textbook of Histology*, W. B. Saunders Co., 2nd Ed., 1934.

phragm. The cannula in the duct was connected with a saline-filled rubber tube terminating in a glass dropper, the tip of which was adjusted to lie on a plane about 2 cm below the lowest level of the thoracic duct in the chest. The effluent was received into a graduated tube and its volume measured over 15-minute periods. Care was taken to keep the cannula free from clots. In 2 experiments chlorazol fast pink was administered (100 mg/kilo), rendering the lymph noncoagulable, and obviating the danger of intravascular clotting.

Flow measurements were made until the pulmonary edema that develops spontaneously in the heart-lung preparation had assumed massive proportions. No increase in flow was noted as the edema developed, but rather a tendency to decrease as fluid was lost from the blood stream into the lungs. Addition of saline or fresh blood to the reservoir restored the flow temporarily.

Any lymph collected from the thoracic duct in the preparation employed in these experiments can come only from the left side of the heart and the left lung. Moreover, since the bronchial vessels are

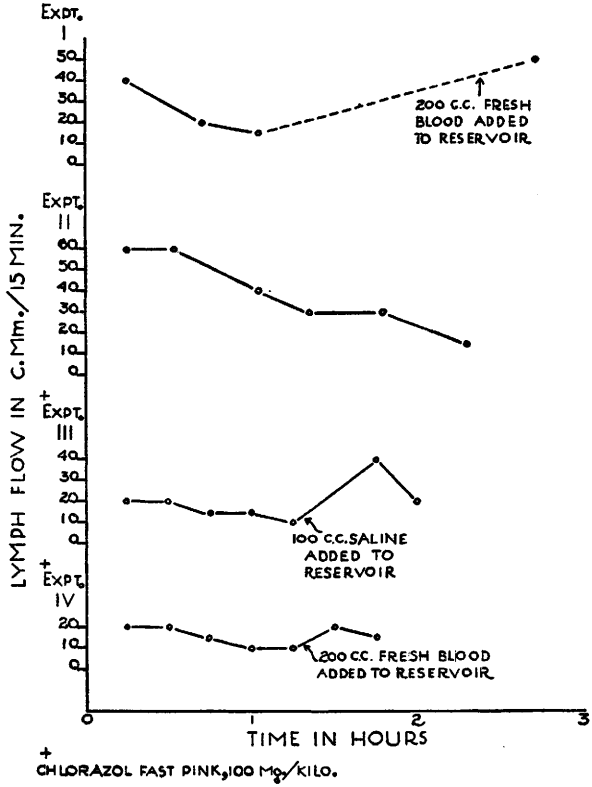

FIG. 1.

not being perfused, any pulmonary contribution must be made by the respiratory areas of the lung. But in view of the fact that flow was not augmented in spite of the development of massive pulmonary edema, it seems unlikely that alveolar tissue is provided with a mechanism for lymph drainage capable of conducting an appreciable flow.

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In vitro Conversion of Prontosil-Soluble to Sulfanilamide by Various Types of Microörganisms.*

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Fuller[1] has shown that both prontosil and prontosil-soluble are partly converted to sulfanilamide in the body. He also demonstrated *in vitro* that stannous chloride and sodium hydrosulphite could reduce prontosil-soluble to form sulfanilamide and amino-acetylamino-naphthol-disulphonic acid. When he gave prontosil-soluble to normal mice, only one-fourth appeared in the urine as sulfanilamide, whereas in mice infected with hemolytic streptococci and given prontosil-soluble, nearly one-half was excreted as sulfanilamide. Bliss and Long[2] succeeded in completely reducing prontosil-soluble *in vitro* with cysteine hydrochloride, and the resultant product proved to be actively bacteriostatic for hemolytic streptococci. They also observed that hemolytic streptococci and a strain of anhemolytic streptococcus were capable of decolorizing prontosil-soluble.[3]

Our purpose is to show that various types of bacteria are capable of converting prontosil-soluble to sulfanilamide. Under standard conditions, the facility with which this change takes place varies with different microörganisms. While such compounds as stannous chloride actually reduce prontosil-soluble to form free sulfanilamide, the mechanism is not as clearly defined whereby the conversion is

* Aided by a grant from the Graduate School of the University of Minnesota.

1 Fuller, A. T., *Lancet*, 1937, **1**, 194.

2 Bliss, E. A., and Long, P. H., *Johns Hopkins Hosp. Bull.*, 1937, **60**, 149.

3 Long, P. H., and Bliss, E. A., *Clinical Use of Sulfanilamide and Sulfapyridine and Allied Compounds*, 1939, 89.