

## 11132 P

**Influence of Specific Serum Therapy on Plasma Lipids in Pneumonia.\***

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The author has thoroughly studied the changes which occur in the plasma lipids during acute infections in children. The investigations have included pneumonia.<sup>1, 2</sup> The patients did not receive any specific treatment during the febrile period of the disease. It has been observed that there is a definite lowering of the values for total cholesterol, total fatty acids, and phospholipids during the height of the infection as compared with the levels during convalescence. The drop in the total cholesterol is due to a marked fall in the ester cholesterol content of the blood. The low total fatty acid values are accompanied by a definite reduction in the iodine absorption values resulting in a low iodine number for the serum fatty acids. The decrease in the phospholipid content of the serum is accompanied by a rise in the iodine absorption values for the phospholipid fatty acids. This rise is, however, quickly followed by a marked fall so that toward the end of the height of the illness very low iodine numbers are obtained for the fatty acids of the phospholipids.

The lipids have been closely followed during the convalescent period. It has been noted that the cholesterol values rise first to normal and in some instances go above the normal range for a short period of time. The total fatty acids increase slowly during the first phase and more rapidly to normal during the last part of convalescence. The phospholipids with the low iodine number of their fatty acids rise very slowly and do not reach the normal range until the patient has fully recovered and there is no residual infection.

These observations in untreated patients during the natural course of the pneumonia prompted an investigation of the influence of serum therapy on the lipid changes. Three children were carefully selected for the preliminary study which is presented in this paper. The subjects were 6, 7, and 8 years of age, all ill with lobar pneumonia due to the pneumococcus, Type I. They received the same dose of pneumococcus Type I serum—namely 40,000 units. It

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<sup>1</sup> Stoesser, A. V., and McQuarrie, Irvine, *Am. J. Dis. Child.*, 1935, **49**, 658.

<sup>2</sup> Stoesser, A. V., *Am. J. Dis. Child.*, 1938, **56**, 1215.

was administered intravenously soon after admission of the child to the hospital. During the period of observation little medication was given to the patients. The administration of fluid or blood subcutaneously, intramuscularly, or intravenously was not permitted. The first sample of blood was obtained just before the serum was given. At this time each child had only been ill from 24 to 48 hours and the temperature ranged between 40° to 40.5°C. The second sample was collected about 24 hours after the temperature had fallen to normal. The third and fourth blood samples were drawn on the fourth and seventh days of the afebrile convalescent period respectively. Each time the blood sample was collected, the patient was thoroughly examined and a roentgenogram of the lungs was obtained. Bloor's methods<sup>3, 4, 5</sup> were used to determine the total, ester and free cholesterol values. The microgravimetric technic of Wilson and Hansen<sup>6, 7</sup> was employed in studying the other serum lipids. The Rosenmund-Kuhnhehn method as described by Yasuda<sup>8</sup> was used to determine the iodine absorption of the serum fatty acids.

The results are summarized in Table I.

TABLE I.  
Plasma Lipids of Pneumonia Before and After Administration of Specific Serum.

| Case No. | Total cholesterol   |     |     |     | Cholesterol esters |     |     |     | Free cholesterol |     |     |     |                |     |     |     |
|----------|---------------------|-----|-----|-----|--------------------|-----|-----|-----|------------------|-----|-----|-----|----------------|-----|-----|-----|
|          | Mg per 100 cc serum |     |     |     |                    |     |     |     |                  |     |     |     |                |     |     |     |
|          | A                   | B   | C   | D   | A                  | B   | C   | D   | A                | B   | C   | D   |                |     |     |     |
| 1. P.H.  | 122                 | 225 | 234 | 221 | 70                 | 146 | 152 | 162 | 52               | 79  | 82  | 59  |                |     |     |     |
| 2. R.S.  | 117                 | 157 | 240 | 219 | 72                 | 89  | 158 | 152 | 45               | 68  | 82  | 67  |                |     |     |     |
| 3. D.C.  | 130                 | 242 | 243 | 250 | 75                 | 156 | 183 | 195 | 55               | 86  | 60  | 55  |                |     |     |     |
| Avg      | 123                 | 208 | 239 | 230 | 72                 | 130 | 164 | 169 | 50               | 77  | 74  | 60  |                |     |     |     |
|          | Total fatty acids   |     |     |     | Iodine number      |     |     |     | Phospholipids    |     |     |     | Iodine number* |     |     |     |
|          | Mg per 100 cc serum |     |     |     |                    |     |     |     |                  |     |     |     |                |     |     |     |
|          | A                   | B   | C   | D   | A                  | B   | C   | D   | A                | B   | C   | D   | A              | B   | C   | D   |
| 1. P.H.  | 289                 | 386 | 452 | 440 | 96                 | 101 | 107 | 114 | 113              | 146 | 152 | 150 | 134            | 114 | 112 | 121 |
| 2. R.S.  | 296                 | 546 | 401 | 456 | 98                 | 90  | 101 | 93  | 99               | 126 | 116 | 126 | 109            | 113 | 107 | 103 |
| 3. D.C.  | 297                 | 423 | 463 | 378 | 91                 | 98  | 96  | 100 | 87               | 115 | 110 | 173 | 107            | 113 | 123 | 115 |
| Avg      | 294                 | 451 | 438 | 424 | 95                 | 96  | 104 | 102 | 99               | 129 | 126 | 149 | 116            | 113 | 114 | 113 |

\* Iodine number of the phospholipid fatty acids.

A—Blood sample collected before serum administered.

B—Blood sample collected 24 hours after temperature normal.

C—Blood sample collected on 4th day of convalescence.

D—Blood sample collected on 7th day of convalescence.

<sup>3</sup> Bloor, W. R., *J. Biol. Chem.*, 1916, **24**, 227.

<sup>4</sup> Bloor, W. R., and Knudson, Arthur, *J. Biol. Chem.*, 1916, **27**, 107.

<sup>5</sup> Bloor, W. R., personal communication to the author.

<sup>6</sup> Wilson, W. R., and Hansen, A. E., *J. Biol. Chem.*, 1936, **112**, 457.

<sup>7</sup> Hansen, A. E., *PROC. SOC. EXP. BIOL. AND MED.*, 1939, **40**, 376.

<sup>8</sup> Yasuda, M., *J. Biol. Chem.*, 1931-32, **94**, 401.

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<sup>1</sup> 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-Knowlton heart-lung preparation was made, and the thoracic duct tied off above the dia-

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<sup>1</sup> Maximow, A. A., and Bloom, W., *A Textbook of Histology*, W. B. Saunders Co., 2nd Ed., 1934.