

CHEMICAL CHANGES IN THE BLOOD OF THE DOG IN EXPERIMENTAL PERITONITIS.

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In previous publications, we have recorded the chemical changes in the blood occurring in pyloric and intestinal obstruction (1, 2). Since distension of the intestine and paralytic ileus are often associated with general peritonitis, a study of the blood chemistry has been made for comparison. It has been suggested that the cause of death in general peritonitis may be due to an intoxication from the intestine rather than to the peritoneal infection.

Method.

Dogs were used for the experimental studies. All operations were done under ether anesthesia with aseptic technique. Peritonitis was produced by ligating the appendix with tape. The animals were kept in a warm room in metabolism cages and allowed to drink water *ad libitum*. Blood for chemical analysis was drawn from the jugular vein daily and at times twice daily if the animal appeared quite ill.

The non-protein nitrogen was determined by the method of Folin and Wu (3), the urea nitrogen by the Van Slyke and Cullen (4) modification of the Marshall method and the carbon dioxide-combining power by the method of Van Slyke (5). The chlorides were determined on the tungstic acid filtrate as suggested by Gettler (6).

OBSERVATION.

Normal dogs weighing from 8 to 13 kilos were used. In all cases the appendix was snugly ligated with tape completely blocking the blood supply. General peritonitis was produced in this way in less than 50 per cent of the animals.

In those dogs developing general peritonitis early changes in the blood chemistry were usually noted. There was a fall in the blood chlorides and a rise in the urea and non-protein nitrogen. The rise in

TABLE I.
Blood Findings in Experimental General Peritonitis.

| Dog No. | Day after operation | Blood | | | CO ₂ -combining power |
|---------|---------------------|----------------------------|---------------|------------|----------------------------------|
| | | Amount per 100 cc. | | | |
| | | Total non-protein nitrogen | Urea nitrogen | Chlorides | |
| | | <i>mg.</i> | <i>mg.</i> | <i>mg.</i> | <i>vol. per cent</i> |
| 1 | 0 | 26.8 | 16.8 | 450 | 43.8 |
| | 1 | 30.6 | 10.6 | 400 | 45.7 |
| | 2 | 91.5 | 46.8 | 330 | 28.7 |
| 2 | 0 | 25.4 | 11.2 | 490 | 28.7 |
| | 1 | 42.2 | 11.9 | 430 | 34.3 |
| | 2 | 32.6 | 21.0 | 410 | 34.3 |
| | 3 | 52.0 | 27.3 | 380 | 36.2 |
| | 4 | 31.6 | 23.1 | 370 | 45.7 |
| | 5 | 34.9 | 13.3 | 420 | 43.8 |
| | 6 | 27.3 | 14.0 | 380 | 41.9 |
| | 7 | 22.8 | 11.9 | 450 | 43.8 |
| | 8 | 37.5 | 20.3 | 450 | 49.0 |
| | 9 | 34.5 | 16.8 | 430 | 46.6 |
| | 10 | 50.8 | 26.6 | 420 | 43.8 |
| | 11 | 78.0 | 45.5 | 400 | 32.4 |
| 12 | 258.0 | 123.3 | 340 | 28.7 | |
| 3 | 0 | 27.3 | 15.4 | 560 | 38.1 |
| | 1 | 27.0 | 18.2 | 510 | 38.1 |
| | 2 | 32.3 | 20.3 | 470 | 38.1 |
| | 3 | 71.4 | 52.5 | 440 | 45.7 |
| 4 | 0 | 28.0 | 15.4 | 460 | 38.1 |
| | 1 | 40.0 | 18.9 | 410 | 40.0 |
| | 2 | 35.3 | 21.7 | 360 | 38.1 |
| 5 | 0 | 25.2 | 13.31 | 480 | 34.3 |
| | 1 | 24.2 | 12.61 | 460 | 38.1 |
| | 2 | 28.2 | 16.81 | 400 | 40.0 |
| | 3 | 23.5 | 11.91 | 400 | 36.2 |
| | 4 | 20.2 | 9.81 | 400 | 38.1 |
| | 5 | 56.3 | 31.51 | 400 | 27.5 |
| 6 | 0 | 32.3 | 13.3 | 470 | 34.3 |
| | 1 | 33.0 | 12.6 | 410 | 45.7 |

TABLE I—*Concluded.*

| Dog No. | Day after operation | Blood | | | CO ₂ -combining power |
|---------|---------------------|----------------------------|---------------|------------|----------------------------------|
| | | Amount per 100 cc. | | | |
| | | Total non-protein nitrogen | Urea nitrogen | Chlorides | |
| | | <i>mg.</i> | <i>mg.</i> | <i>mg.</i> | <i>vol. per cent</i> |
| 6 | 2 | 47.2 | 17.5 | 370 | 43.8 |
| | 3 | 31.2 | 15.4 | 360 | 52.0 |
| | 4 | 31.8 | 16.8 | 380 | 41.9 |
| | 5 a.m. | 34.5 | 17.5 | 350 | 14.3 |
| | 5 p.m. | 45.8 | 22.4 | 320 | 10.5 |
| 7 | 0 | 28.0 | 10.2 | 450 | 40.0 |
| | 1 | 31.9 | 14.0 | 390 | 40.0 |
| | 2 | — | 51.3 | 320 | — |
| 8 | 0 | 23.4 | 8.4 | 490 | 35.3 |
| | 1 | 22.1 | 8.4 | 480 | 29.6 |
| | 2 a.m. | 53.2 | 36.8 | 440 | 22.1 |
| | 2 p.m. | 89.8 | 49.0 | 370 | — |

the nitrogenous elements usually started from 1 to 3 days before the death of the animal and increased until death. The carbon dioxide-combining power did not show any constant change. In some instances there was little or no change, in others a slight increase and in still others a decrease (Table I).

In all animals included in this report a well developed general peritonitis throughout the entire abdominal cavity was found at autopsy. The animals lived from 2 to 12 days following the operation with an average length of life of $4\frac{1}{2}$ days.

DISCUSSION.

The changes here noted in the blood chlorides, urea nitrogen and non-protein nitrogen resemble those observed in pyloric and high intestinal obstructions. In those two conditions an alkalosis develops which is not observed in general peritonitis.

Since the clinical manifestations of acute high intestinal obstruction and general peritonitis are strikingly alike and the chemical changes in

the blood are similar, it seems quite probable that the cause of death may be somewhat similar.

CONCLUSION.

1. A study of the blood chlorides, urea and non-protein nitrogen and the carbon dioxide-combining power in experimental general peritonitis is here reported.

2. The similarity between the chemical changes in high intestinal obstruction and general peritonitis is noted. These chemical changes suggest that the cause of death may be, at least in part, the same in the two diseases.

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