

STUDIES ON ACUTE INTESTINAL OBSTRUCTION

II. ACUTE STRANGULATION

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In the preceding paper¹ we have shown that acute intestinal obstruction may be divided both clinically and experimentally into acute simple obstruction and acute strangulation; that the symptomatology, length of life and lethal factors are different in each case. In uncomplicated simple obstruction death is probably due to starvation and its sequelae.

In this paper we report a series of experiments demonstrating that the foregoing factors in simple obstruction play little or no part in acute strangulation but instead that death is due to shock and toxemia. The rapidity with which these factors act varies with the length of intestine involved, the degree of arterial and venous obstruction and the location of the lesion.

No attempt will be made to review the extensive literature on the subject of intestinal obstruction. Only those articles which have dealt with experimentally produced strangulation will be briefly summarized. Von Albeck² was the first to produce experimental strangulation. He used loops from 6 to 8 inches (from 15 to 20 cm.) in length. His dogs died in collapse in from twenty-four to forty-eight hours, showing vomiting, diarrhea, subnormal temperature and spasms prior to death. He concluded death was due to the absorption of putrefactive toxins formed in the strangulated loop.

Eisberg³ also produced strangulation but under morphin ether anesthesia. In his animals the minimum length of life was from three to seven hours, the maximum duration forty-two hours. His statements are rather indefinite and not detailed. In speaking of the cause of death he says: "Surely at times a devitalized segment appears to be responsible for a lethal outcome, long before there has been time for bacterial action to play a part in the results."

* From the Departments of Anatomy and Physiology, University of Oregon Medical School.

1. Hausler, R. W., and Foster, W. C.: Studies in Acute Intestinal Obstruction. I. Different Types of Obstruction Produced under Local Anesthesia. *Arch. Int. Med.* **34**:97 (July) 1924.

2. Von Albeck: *Arch. f. klin. Chir.* **65**:569, 1902.

3. Eisberg, H. B.: *Ann. Surg.* **74**:584 (Nov.) 1921.

Murphy and Vincent⁴ are the only other authors who have paid any particular attention to circulatory conditions in obstruction. Their work was on cats. Strangulation, venous obstruction and anemia were produced under ether anesthesia. Blood pressure tracings under ether anesthesia were taken by them on the carotid artery at irregular intervals. Such procedures have many disadvantages and introduce other factors, especially in a very sick animal. To quote them further, "the animals after from four to six hours were in such poor condition that it was difficult to etherize them and obtain a second reading, since the slightest excess of ether was fatal, and the low blood pressure tended to the formation of a clot in the cannula." The local anesthesia method used on the femoral artery in our series permits of hourly tracings with no inconvenience to the dog, and excludes vagal and sympathetic stimulation and ether intoxication. Murphy and Vincent report that with venous obstruction the height of intoxication was reached in from four to six hours. They believe the symptoms and death to be due to "bacterial endotoxemia."

MATERIAL AND METHODS

The operative detail has been given in a previous paper. All operations were performed on dogs under local anesthesia with careful aseptic technic. Definite lengths of bowel were twisted and tied with rubber tubing in such a way as to cause a complete venous obstruction. Temperature, pulse, respiration and blood pressure were taken before and at definite intervals after operation. Blood chemistry estimations were made in the chemical laboratory. All solutions were adequately checked. The following methods were used: Blood sugar: Haskins-Holbrook⁵ modification of Shafer titration method, total nitrogen, Microkjeldahl, urea nitrogen, Microkjeldahl, chlorids, Folin.⁶

The dogs were kept under almost continuous observation from the beginning of the experiment until death. Necropsies were performed immediately. The abdominal organs were fixed and sectioned for microscopic examination.

EXPERIMENTAL OBSERVATIONS

From the first series of twelve dogs with acute strangulation the following five typical protocols illustrate the postobstructive course when different lengths of the intestine are involved.

4. Murphy and Vincent: Boston M. & S. J. **165**:684, 1911.

5. Haskins, H. D., and Holbrook, W. P.: J. Lab. & Clin. Med. **8**:747 (Aug.) 1923.

6. Folin, O. K.: Lab. Manual of Bio. Chem. Ed. 2, 1919.

PROTOCOL OF EXPERIMENTS

EXPERIMENT 1.—Dog 50, adult, tan terrier, male, weight, 23 pounds (10 kg.). Five inch (12.7 cm.) segment. Death in twenty-nine hours.

10:05 a. m. Temperature (rectal), 102.4 F.; pulse, 84; respirations, 20; blood pressure, 150 systolic.

10:15, operation. Five inches (12.7 cm.) of jejunum and ileum strangulated. Off table 10:30. Ran about the room.

12:00 noon. Temperature, 101.8; pulse, 110; respirations, 24; blood pressure, 146 systolic. Animal in good condition. Drank and vomited at frequent intervals.

2:00 p. m. Temperature, 98; pulse, 130; respirations, 28; blood pressure, 144 systolic. Whined frequently and seemed distressed.

5:00 p. m. Temperature, 99.2; pulse, 130; respirations, 28; blood pressure, 140 systolic. Howled frequently and was very nervous.

7:30 a. m. Next day. Temperature, 104.4; pulse, 190; respirations, 36; blood pressure, 110 systolic; the dog appeared to be very toxic and vomited frequently.

10:30 a. m. Temperature, 104.6; pulse, 190; respirations, 34; blood pressure, 90 systolic. Hardly able to walk.

2:00 p. m. Temperature, 105.6; pulse, 210; respirations, 68; blood pressure, 50 systolic. Dog moribund.

3:15 p. m., the dog was killed, and there was an immediate necropsy. Findings: Lungs, liver, spleen and kidneys were grossly normal. Peritoneal cavity contained considerable foul smelling fluid. Strangulated segment was ruptured, purplish black in color and necrotic. Generalized peritonitis.

EXPERIMENT 2.—Dog 53, brown female, weight, 30 pounds (13.6 kg.). Twelve inch (30 cm.) segment. Death in twenty-five hours.

4:15 p. m. Temperature, 101.4; pulse, 84; respirations, 18; blood pressure, 145 systolic.

4:30 p. m. Operation. Strangulation of 12 inches (30 cm.), upper ileum. Off table at 4:50. Walked to cage; few minutes later vomited.

9:00 a. m. Following day. Temperature, 101.6; pulse, 152; respirations, 26; blood pressure, 130 systolic. The animal looked sick. Frequent vomiting after drinking. Gait steady.

11:00 a. m. Temperature, 101.6; pulse, 164; respirations, 28.

12:00 noon. Temperature, 102.4; pulse, 200; respirations, 27; blood pressure, 120 systolic. The dog appeared much weaker.

1:30 p. m. Temperature, 103; pulse, 220; respirations, 32. The animal was still able to walk.

2:40 p. m. Temperature, 102.4; pulse, 210; respirations, 36; blood pressure, 98. Much weaker.

3:45 p. m. Temperature, 104.4; pulse, 210; respirations, 48; blood pressure, 80. Dog lay in the cage, unable to stand.

4:45 p. m. Temperature, 105.8; pulse, 280; respirations, 70; blood pressure, 50 systolic.

5:20 p. m. Dead. Immediate necropsy. Findings: Peritoneal cavity contained about 300 c.c. of dark, bloody, foul smelling fluid; the strangulated loop was ruptured. Other viscera were grossly normal.

EXPERIMENT 3.—Dog 56, adult female collie, weight, 28 pounds (12.7 kg.). Fifteen inch (37 cm.) segment. Death in twenty-two hours.

10:45 a. m. Temperature, 102.8; pulse, 72; respirations, 18; blood pressure, 160 systolic.

11:00 a. m. Operation. Fifteen inches (37 cm.) of lower ileum strangulated. Off table at 11:20. Ran about room.

2:30 p. m. Temperature, 105.6; pulse, 74; respirations, 72; blood pressure, 152 systolic. There was marked thirst and frequent vomiting during the last two hours.

5:00 p. m. Temperature, 104.4; pulse, 84; respirations, 42; blood pressure, 145 systolic.

9:00 p. m. Temperature, 104.6; pulse, 160; respirations, 24; blood pressure, 124 systolic. Dog looked dejected; marked thirst and much vomiting.

8:00 a. m. Following day. Temperature, 107; pulse, 240; respirations, 56; blood pressure, 74 systolic.

9:00 a. m. Dead. Necropsy at once. Necropsy findings: Gangrenous, strangulated loop ruptured; belly full of bloody exudate, tissue very friable. Generalized peritonitis. Other viscera were grossly normal.

EXPERIMENT 4.—Dog 35, white bull, male, weight, 22 pounds (10 kg.). Eighteen inch (45 cm.) segment. Death in ten hours.

8:30 a. m. Temperature, 101.8; pulse, 94; respirations, 18; blood pressure, 152 systolic; white blood count, 6,900.

9:15 a. m. Operation. Eighteen inches (4.5 cm.) of upper ileum strangulated. Off table at 9:35. Ran around room. Whined, drank and vomited a few minutes later.

9:50 a. m. Temperature, 101.6; pulse, 160; respirations, 20; blood pressure, 150 systolic.

10:40 a. m. Temperature, 101.6; pulse, 162; respirations, 20; blood pressure, 118 systolic. Vomited frequently, very thirsty.

12:00 noon. Temperature, 99.1; pulse, 160; respirations, 22. Dog very listless. Vomited blood-stained bowel contents.

2:20 p. m. Temperature, 101; pulse, 180; respirations, 21; blood pressure, 102 systolic. Animal markedly shocked. Still able to walk; staggering gait.

4:20 p. m. Temperature, 101.2; pulse, 180; respirations, 22; blood pressure, 88 systolic. Hardly able to walk, fell frequently. Respirations irregular and loud. Drank occasionally and retched.

5:05 p. m. Temperature, 99.6; pulse, 180; respirations 26; blood pressure, 71 systolic. Advanced stage of shock.

7:06 p. m. Temperature, 99.6; pulse, 178; respirations, 26; blood pressure, 62 systolic; white blood cells, 10,700. No response to sensory stimuli.

8:05 p. m. Dead. Necropsy immediately. Findings: Lungs, liver, spleen and kidneys were grossly normal. Strangulated segment enormously distended, glistening black in color; contained 150 c.c. of dark bloody fluid, only slight odor. Stomach, duodenum and large intestine very anemic. Forty cubic centimeters of blood stained fluid in the peritoneal cavity.

EXPERIMENT 5.—Dog 36, adult Airedale male, weight, 30 pounds (13.6 kg.). Twenty-four inch (60.96 cm.) segment. Dead in seven hours.

10:30 a. m. Temperature, 102.2; pulse, 84; respirations, 16; blood pressure, 138 systolic; white blood count, 14,500.

11:30 a. m. Operation. Twenty-four inches (60.96 cm.) of lower ileum strangulated. Off table at 11:42. Ran about room, appeared normal.

11:50 a. m. Temperature, 102.6; pulse, 100; respirations, 26. Very thirsty, drank, vomited in a few minutes.

1:00 p. m. Temperature, 101.5; pulse, 170; respirations, 26; blood pressure, 120 systolic. Stuporous and dazed.

3:00 p. m. Temperature, 101.4; pulse, 180; respirations 36; blood pressure, 88 systolic. Dog weaker, when forced to get up staggered and leaned against cage for support. Respirations rapid and stertorous.

3:40 p. m. Temperature, 101.4; pulse, 182; respirations, 42; blood pressure, 44 systolic. Gradually getting weaker.

4:50 p. m. Temperature, 101.4; pulse, 220; respirations, 46; blood pressure, 42 systolic. Profound shock.

6:00 p. m. Temperature, 100.2; pulse, 228; respirations, 46; blood pressure, 42 systolic; white blood count, 18,400.

6:30 p. m. Dead. Necropsy findings: Abdominal and thoracic viscera grossly negative. Strangulated visceral segment distended, gangrenous, con-

tained 35 c.c. of dark, bloody fluid. Fifty cubic centimeters of similar fluid were found in the peritoneal cavity. No perforation.

Table 1 shows that there is a direct relationship between the length of bowel strangulated and the rapidity with which death ensues.

Naturally slight variations occur, due to the size and the type of the dog and the degree of venous occlusion produced at operation. However, the results definitely indicate that the longer the segment, the more pronounced the shock and the earlier the death. With the short segments, 12 inches (30 cm.) or less, shock although present, is not sufficient to cause a pronounced derangement and disorganization of the cardiovascular mechanism. The fall in blood pressure is only moderate, the pulse rate is not extremely rapid and the animals are able to overcome this primary phase, only to succumb later to a terrific toxemia and peritonitis. In the cases with long segments, 18 inches (45 cm.) or more, shock appears very early and is extremely severe. The blood

TABLE 1.—*Relationship Between Length of Bowel Strangulated and Death*

Dog No.	Weight in Pounds	Inches of Bowel Strangulated	Length of Life, Hours
50	23	5	29
60	42	6	28
53	30	12	25
37	26	12	24
34	17	12	23.5
56	28	15	22
40	35	15	20
31	50	18	12
35	22	18	10
32	47	24	9
36	30	24	7
39	35	26	8.5

pressure falls rapidly, the pulse rate climbs to a very high level and the temperature falls. The animal goes into collapse and dies from shock before toxemia can develop.

TOTAL ANEMIA

In this group, segments of bowel from 12 to 20 inches (from 30 to 50 cm.) in length were used. Three of the loops were found perforated at necropsy. This group does not parallel any clinical entities, but serves as a check on other observers' experiments. The following typical protocol well represents this series, of four dogs, in which the intestine was tied so tightly that a complete and instantaneous anemia was produced.

EXPERIMENT 7.—Dog 38, adult mongrel male, weight, 35 pounds (15 kg.). Twenty inch (50 cm.) segment. Dead in twenty hours.

10:30 a. m. Temperature, 102.8; pulse, 90; respirations, 16; blood pressure, 146 systolic; white blood count, 8,900.

11:00 a. m. Operation. Total anemia of 20 inches (50 cm.) of upper ileum. Off table at 11:20. Dog vomited two minutes after leaving the table.

12:00 noon. Temperature, 102.8; pulse, 140; respirations, 20; white blood count, 11,600. The animal vomited frequently and appeared distressed. Blood pressure, 134 systolic.

2:30 p. m. Temperature, 101.3; respirations, 28; pulse, 140; blood pressure, 125 systolic. Frequent vomiting. Gait steady, condition fair.

7:00 p. m. Temperature, 102.9; pulse, 140; respirations, 36; blood pressure, 115 systolic; white blood count, 10,500. Condition somewhat weaker but presented none of the marked shock symptoms seen in the previous series. No further observations were made during the night.

7:00 a. m. Temperature, 98.6; pulse, 240; respirations, 40; blood pressure, 38 systolic. Dog moribund.

7:35 a. m. Dead. Immediate necropsy. Findings: Lungs, spleen, kidneys and liver grossly normal. Peritoneal cavity contained small amount of blood stained fluid. Strangulated segment was slightly distended with gas. Pale, yellowish, pasty substance in the bowel lumen. Tissue of segment very friable, of a yellowish color, showed no perforation.

In this group it is obvious that the pathologic and the clinical pictures are entirely different from those of venous obstruction. The total anemia intestine shows little or no dilatation and contains no fluid. On the other hand, with venous obstruction the strangulated loop is dilated to two or three times its normal size and enormously distends the abdominal cavity. Furthermore, this group, in which there is no blood or fluid loss and only slight distention of intestine, does not show the acute collapse symptoms of the venous obstruction series. Instead, we have only a slight, gradual rise in pulse rate and a practically normal blood pressure during the first fifteen hours. Vomiting is less profuse, thirst slight, and the animal is much stronger. The temperature and respirations, however, show extreme elevation, which are not seen in the strangulation animals. The respiratory rate always rises in a few hours to 40 or 50 per minute, and in one case it averaged 120 per minute for over sixteen hours. The temperature shows a gradual continuous rise to an average maximum of 105 during the last five to eight hours.

The above experiments prove (1) that in acute strangulation blood loss and loop distention are important factors in the production of shock; (2) that a rapid rise in respiration and temperature, along with only slight variations in pulse rate and blood pressure, are indicative of toxemia and peritonitis.

RUBBER BAG SERIES

In order to show the relative importance of toxemia and shock in acute strangulation, five dogs were treated as follows. At operation the bowel segments were introduced into thin rubber bags, and a rubber catheter was tied around the base sufficiently tight to cause complete venous obstruction. This procedure prevents the absorption of toxins from the strangulated segment by the peritoneal serosa, without interfering with the distention of the segment by blood and gas.

EXPERIMENT 8.—Dog 12, black and white mongrel, male, weight, 31 pounds (14 kg.). Twenty-four inch (60 cm.) segment. Dead in eight and one-half hours.

9:00 a. m. Temperature, 102.6; respirations, 20; blood pressure, 145 systolic; white blood count, 14,200.

9:30 a. m. Operation. Twenty-four inches (60 cm.) of upper ileum and jejunum strangulated in rubber sac. Off table in twenty minutes. Drank freely, vomited.

10:00 a. m. Temperature, 102.6; pulse, 95; respirations, 24; blood pressure, 145 systolic.

11:00 a. m. Temperature, 102.6; pulse, 200; respirations, 30. Dog drank, retched and vomited frequently and appeared weaker.

12:00 noon. Temperature, 102.6; pulse, 240; respirations, 36; white blood count, 20,600. Frequent whining and vomiting.

1:05 p. m. Temperature, 102.8; pulse, 240; respirations, 50; blood pressure, 86 systolic. Animal rapidly going into shock, leaned heavily against cage.

2:30 p. m. Temperature, 102.3; pulse, 250; respirations, 42; blood pressure, 68 systolic. Dog very weak, in collapse.

3:30 p. m. Temperature, 104.3; pulse, 250; respirations, 48; blood pressure, 68 systolic; white blood count, 9,200.

4:45 p. m. Temperature, 104.6; pulse, 250; respirations, 52; blood pressure, 62 systolic. Could hardly walk. When aroused dog stood up and then fell.

5:30 p. m. Temperature, 104.6; pulse, 260; respirations, 48; blood pressure, 40 systolic. Moribund.

6:08 p. m. Dead. Immediate necropsy. Findings: Peritoneum normal, small amount of fluid present. No perforation. Intestine was greatly distended with gas and contained 250 c.c. of dark, bloody fluid. Gallbladder contained 20 c.c. concentrated fluid. Proximal intestine empty and anemic, of mottled appearance.

EXPERIMENT 9.—Dog 13, adult mongrel, male, weight, 30 pounds (13 kg.). Ten inch (25 cm.) segment. Death in Sixty-six hours.

March 9, 2:00 p. m. Temperature, 102.8; pulse, 86; respirations, 20; blood pressure, 150 systolic.

2:30 p. m. Operation. Ten inches (25 cm.) of jejunum strangulated in rubber sac. Off table in twenty minutes.

5:00 p. m. Temperature, 102.8; pulse, 110; respirations, 24; blood pressure, 145 systolic. Occasional vomiting. Condition good.

March 10, 8:00 a. m. Temperature, 102.6; pulse, 96; respirations, 26. Dog appeared in excellent condition; wagged tail, walked about with no difficulty. Respirations were slightly labored.

2:00 p. m. Frequent vomiting, appeared weaker.

March 11, 8:30 a. m. Temperature, 103.2; pulse, 108; respirations, 26. Animal in good condition.

5:00 p. m. Temperature, 104; pulse, 130; respirations, 28; blood pressure, 138 systolic. Dog looked toxic.

March 12, 8:00 a. m. Dog moribund. Killed. Necropsy findings: Sac distended with gas and contained about 100 c.c. bloody fluid. Perforation just proximal to tie. Generalized peritonitis.

In the foregoing experiments long and short loops were used. From the previous experiments it was evident that shock was the predominating lethal factor when long segments were used. Thus the use of the rubber bag to prevent any possible absorption should not materially effect the duration of life. A comparison of Experiment 4 (segment 24 inches, death in seven hours) with Experiment 8 (segment 24 inches placed in rubber sac, death in eight and one-half hours) shows that

the change in temperature, pulse, respiration, blood pressure and duration of life are practically identical in each case. It is thus evident that toxemia is only of minor importance, since the prevention of the absorption of toxin does not materially affect the outcome. With short loops, however, where toxemia is more predominant and shock of a milder degree, the length of life should be markedly affected. A comparison of Experiment 2 (segment 12 inches, death in twenty-five hours) with Experiment 9 (segment 10 inches, in rubber bag, death in sixty-six hours) reveals this difference in a very striking manner. The foregoing experiments prove the following: (1) In the rapidly fatal cases of long segment strangulation toxemia is not an important lethal factor; (2) in the short segments toxemia is very pronounced.

RELEASE OF STRANGULATION

To show the effect of release of the strangulation after shock symptoms had become moderately advanced experiments of the following nature were performed.

EXPERIMENT 14.—Dog 47, adult brown male, weight, 50 pounds (22 kg.).
11:00 a. m. Temperature, 101.2; pulse, 62; respirations, 16; blood pressure, 146 systolic.

11:45 a. m. Operation. Strangulation of 20 inches (50 cm.) of upper ileum. Off table in fifteen minutes. Vomited slight amount of blood tinged mucus. Voided 175 c.c. urine. Small stool.

12:45 p. m. Temperature, 97; pulse, 50; respirations, 28; blood pressure, 132 systolic. Dog appeared to be very sick. Whined continuously, seemed cold. Heater placed in cage.

2:00 p. m. Temperature, 104.7; pulse, 206; respirations, 30; blood pressure, 100 systolic. Condition worse.

2:55 p. m. Temperature, 105; pulse, 184; irregular, respirations, 32; blood pressure, 78 systolic. Anal sphincter relaxed.

4:00 p. m. Temperature, 106; pulse, 170; respirations, 42; blood pressure, 75 systolic. Appeared deeply shocked, hardly able to stand.

4:20 p. m. Belly opened under local anesthesia, strangulation relieved. Gut appeared very black, apparently not viable, but returned to belly cavity without resection.

5:45 p. m. Temperature, 106; pulse, 172; respirations, 40; blood pressure, 84 systolic. Passed small liquid stool twenty minutes after removal of strangulation. Given one grain morphin by hypodermic.

6:45 p. m. Temperature, 106.5; pulse, 170; respiration, 50; blood pressure, 90 systolic. Frequent small, fluid stools. Sleeping quietly.

7:30 p. m. Temperature, 105.2; pulse, 210; respiration, 50; blood pressure, 95 systolic. Five hundred c.c. of 5 per cent. glucose in physiologic sodium chlorid solution given subcutaneously. Condition much improved.

April 11, following day.

8:00 a. m. Temperature, 103.2; pulse, 100; respirations, 24; blood pressure, 115 systolic. Dog appeared to be in good condition.

From this time the recovery was uneventful.

This animal was sacrificed one week later. The segment previously strangulated was markedly thickened, hard and indurated and mottled purplish in color. Lumen patent but smaller than normal. Peritoneum smooth and glistening, with no signs of adhesions.

These experiments illustrate very clearly the degree of systemic depression produced by the violent peristalsis of the proximal segment and the stretching of both the parietal and visceral peritoneum by the strangulated intestine. These loops are always distended to about two or three times their former length and diameter, completely filling and distending the peritoneal cavity.

Before the release of the obstruction the animals were in a precarious condition. The blood pressure was low, the pulse rapid and feeble, respirations fast and stertorous, and muscular weakness was pronounced. The dogs were hardly able to stand, and paid little attention to external stimuli.

As soon as the obstruction is released a marked change occurs. The animal is visibly relieved, the depression passes, vomiting ceases and the bowels are emptied by two or three liquid stools. He is soon able to walk about and drinks large quantities of water. Soon the pulse becomes slower and of a better quality. The temperature returns to normal. The blood pressure and respirations show few changes during the first three hours. The following day the animal is in a good condition and usually makes an uneventful recovery. The loop fluid, which was always allowed to pass into the distal segment, produces a mild diarrhea, but apparently without any marked deleterious effect on the animal.

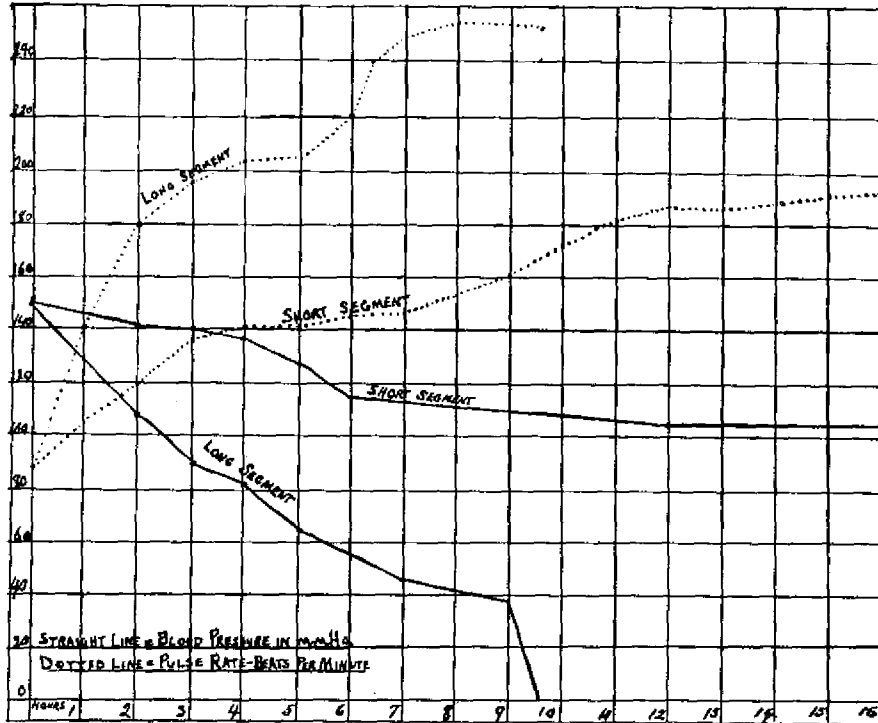
The foregoing results further indicate that shock is a predominating lethal factor in early strangulation. Toxemia apparently has not developed to any marked degree. Further discussion of these experiments will be given in a subsequent article on the treatment of acute intestinal obstruction.

OBSERVATIONS ON PULSE AND BLOOD PRESSURE

One of the most outstanding features of acute strangulation experimentally produced is a constant, pronounced fall in blood pressure, and a coincident and proportionate increase in pulse rate. These striking observations are graphically presented in the chart. A definite relation exists between the length of segment strangulated and the blood pressure and the pulse changes. In the long segments a rapid fall in blood pressure from an average of 150 mm. of mercury systolic to 90 mm. occurs within the first three hours. At the end of five hours it has dropped to 60 mm. and by seven hours has usually reached the 40 mm. level. At the same time the pulse rate jumps from an average of 80 to 90 per minute to about 140 at the end of the first hour, 180 or above at the end of two hours, 200 at four, and from there on until death it averages about 240 beats.

In short segments the blood pressure falls to approximately 110 mm. systolic in the first six hours and remains at about this level until

several hours before death. At this time a very rapid fall begins, often amounting to 60 mm. in an hour's time. The pulse rate usually shows a fairly rapid rise within the first six hours to an average of 140 per minute. After this level has been reached there is generally only a slight elevation during the next ten hours. After sixteen hours, however, a profound toxemia has developed and the pulse shows a rapid increase to over 200, at which level it continues until death.



Composite curves of pulse and blood pressure variations in long and short segment strangulation.

TEMPERATURE

The temperature in acute strangulation is so subject to fluctuation that it does not give a reliable indication of the pathology or condition of the experimental animal. Some dogs with long strangulated segments, profound shock and early death show a temperature curve that does not vary over one-half a degree from the normal. Others, with exactly similar pathology, have a rapid fall of three or four degrees within the first two hours. In general, however, the longer segments show a slight fall at the end of two hours and then a more rapid progressive decline until death. The blood pressure and temperature curves are very similar in time and extent. In short segments there is

little or no variation within the first ten hours. After this time, however, a marked toxemia or peritonitis has developed, and the temperature is usually very high, averaging about 105 F.

The clinical value of external heat applications was well demonstrated in several cases of profound shock, a subnormal temperature of 97 F., rising in an hour's time to 103 F. with marked improvement in the animal's general condition.

RESPIRATION

In all cases of acute strangulation there was a definite increase in respiratory rate. The curve parallels that of the pulse rate very closely. In the long segment cases there is an early rise from a normal of approximately 20 to an average of 44 per minute. They usually become shallow and thoracic in type, and toward the last are very irregular. With the shorter segments there is an early increase of about ten respirations per minute, at which level it remains until the onset of profound toxemia, when a rapid elevation to 40 or more takes place.

TOXICITY OF LOOP AND PERITONEAL EXUDATES

Much work of a very technical nature has been done on the chemistry and toxicity of the loop fluid in dogs in which no circulatory disturbances were produced. The degree of toxicity was determined for the most part by intravenous injection of loop content, but since the normal bowel contains many proteoses and amines which are very toxic on intravenous injection, too much weight cannot be placed on the results of such experiments. There can be no question about the toxicity of body fluids and bowel content when these are mixed with bacteria and left in a virtual incubator for twelve hours or more. This is evidenced in the peritonitis following bowel rupture, in which cases death ensues in a relatively short time from a terrific toxemia.

The purpose in the following experiments was to determine the relative toxicity of the loop content of dogs dying in less than twelve hours, and to ascertain its relation to the sudden collapse beginning two or three hours after strangulation, to which certain authors refer as a profound toxemia.

In acute strangulation, obstruction of the venous and lymphatic channels is produced by the constricting band and the subsequent thrombosis of the mesenteric vessels. Thus the only possible path of absorption is by a transudation of the loop fluid through the strangulated bowel walls into the peritoneal cavity and thence by the peritoneal lymphatics into the blood stream. Further, the inflammatory reaction of the peritoneal serosa to this transudate may partially detoxicate it. Therefore, it seemed unphysiologic to inject this fluid intravenously,

and accordingly in the following experiments we have made all injections intraperitoneally. The fluid was drained from the strangulated segments immediately after death, filtered through sterile gauze to facilitate injection, and without further treatment, either by heat or chemicals, injected intraperitoneally. By this simple treatment any increase or decrease in toxicity by necropsy changes was prevented.

In the first experiment a mixture of the loop fluids of Dog 32, dying in nine hours, and Dog 31, dying in twelve hours, was injected into a normal, young dog, weighing 12 pounds (5.4 kg.), with the results seen in Table 2.

TABLE 2.—*Experiment 16, Dog 33, Young Mongrel, Male, Weight 12 Pounds (5.4 kg.).*

Time	Temperature	Respiration	Pulse	Amount of Fluid
6: 30 p. m.....	103.0	16	90	Normal
6: 40 p. m.....	103.0	16	90	20 c.c.
7: 30 p. m.....	102.8	22	96	20 c.c.
8: 30 p. m.....	102.4	28	110	20 c.c.
9: 00 p. m.....	20 c.c.
Next day				
8: 15 a. m.....	104.0	34	180	No injection

The dog showed signs of peritonitis for a few days but made an uneventful recovery.

In order to test the toxicity of early loop fluid in massive doses the entire loop contents, 100 c.c., of a dog dying in seven hours (Experiment 5, Dog 36), was injected in one dose, with the result seen in Table 3. The fluid was left on ice over night.

TABLE 3.—*Experiment 17, Dog 38, Young, Black and White, Male Mongrel Weight 15 Pounds (6.8 kg.).*

Time	Temperature	Respiration	Pulse	Amount of Fluid
9: 30 a. m.....	103.4	20	100	Normal
9: 45 a. m.....	103.4	20	100	104 c.c.
1: 20 p. m.....	103.3	28	134	
4: 00 p. m.....	104.0	28	160	
Next day				
9: 00 a. m.....	103.1	30	120	
11: 00 a. m.....	103.2	26	118	

Half an hour after the injection the dog showed marked salivation, dry nose and restlessness, and vomited once. Two hours later he appeared depressed, but walked about quite normally. After six hours the condition was much improved. On the following day the dog was in good condition, and made an uneventful recovery. An exploratory laparotomy on this animal three weeks later revealed a normal peritoneal serosa.

In dogs dying from experimental strangulation there is always a bloody exudate found at necropsy in the abdominal cavity. The total quantity of this fluid was gathered from one dog (Dog 31, dying in

twelve hours), 60 c.c. in amount, and injected intraperitoneally with the results seen in Table 4.

About two hours after the injection the dog appeared slightly depressed. He held his belly rigid and the respirations were shallow and thoracic in type. The nose was dry and he drank freely. He passed one liquid stool. The following day the animal appeared entirely normal.

In the preceding experiments no attempts were made to kill the bacteria in the loop fluid before its injection into the experimental animal. Accordingly, part of the reaction obtained is undoubtedly due to the subsequent peritonitis following this massive bacterial injection. The fact that no severe peritonitis followed demonstrates the enormous ability of the peritoneum to overcome bacterial invasion. All of the injected animals were small and young, and therefore, if the fluid was very toxic, should have shown extreme reaction to these doses.

The process of transudation from the strangulated intestine and absorption via the peritoneal serosa is a gradual one, beginning soon

TABLE 4.—*Experiment 18, Dog 37, Brown, Mongrel, Male, Weight 11 Pounds (5.0 kg.).*

Time	Temperature	Respiration	Pulse	Amount of Fluid
10: 35 a. m.....	103.2	22	120	Normal
10: 40 a. m.....	60 c.c.
11: 05 a. m.....	103.4	22	110	
1: 30 p. m.....	104.4	26	110	
3: 35 p. m.....	103.5	28	80 Irreg.	
6: 00 p. m.....	103.0	22	62	
Next day				
8: 00 a. m.....	102.3	18	60	

after the strangulation is produced. The amount of this transudation varies at different times, according to the pressure within the strangulated loop and the force of the incoming arterial flow. In the first experiment, therefore, amounts similar to what might have been absorbed were given at hourly intervals. Since the reaction was very slight and no great degree of toxicity was evidenced in the first animal, the second animal was given the entire loop contents in one massive dose. This procedure gave a more pronounced reaction, but this animal likewise quickly recovered. The free peritoneal fluid, which is a mixture of transudate and peritoneal exudate, proved to be relatively nontoxic.

The preceding protocols show that the fluids which accumulate in the peritoneal cavity and strangulated loops of less than twelve hours' duration are not highly toxic when introduced intraperitoneally, even in massive doses. Furthermore, they indicate that the sudden and extreme collapse following long segment strangulation cannot be explained entirely as a profound toxemia.

BLOOD CHEMISTRY

Table 5 shows the changes in blood urea, nonprotein nitrogen, sugar and chlorids in five of the strangulation dogs.

It will be seen that there is an increased nonprotein nitrogen in all cases. The final determinations, however, are not far above the normal level. They probably indicate both urinary retention and increased tissue destruction. Urea nitrogen also consistently increases, but does not always parallel the nonprotein nitrogen curve. Blood sugar shows marked fluctuations, usually within normal limits; at no time was there a definite continuous hypoglycemia or hyperglycemia. In two dogs the sodium chlorid percentage is decreased slightly, in two a slight

TABLE 5.—*Blood Chemistry in Five Dogs*

Dog No.	Hours After Operation	Blood Chemistry, Amounts per 100 C.c.				Length of Life		
		Total Nonprotein Nitrogen, Mg.	Urea Nitrogen, Mg.	Sugar, Mg.	Sodium Chlorid, Mg.			
36	0	28	9.6	114	501	7 hours		
	7	50	22.4	66	501			
32	0	30	10.0	101	478	9 hours		
	4	..	23.0	173				
	6	..	25.0	150	412			
	7	42	31.0	90				
	8	..	29.0	137				
	9	60	36.0	114	379			
35	0	32	9.8	98	462	10 hours		
	6	129				
	10	42	18.3	85	501			
31	0	28	11.5	104	478	12 hours		
	3	..	16.5	95				
	5	..	16.0	78				
	6	..	17.0	86	495			
	7	..	26.5	91				
	8	28	25.0	98				
	9	35	26.0	82				
	10	28	24.0	114	501			
	37	0	22	12.0	81		500	24 hours
		6	24	14.0	92		470	
12		30	19.0	67	445			

increase was noted, and in the other there was no change. The chlorid variations apparently have little significance.

Immediately after the strangulation is produced, violent peristalsis begins in the proximal segment and in five to fifteen minutes is followed by intense retching and vomiting. The character and frequency of vomiting and the type and amount of vomitus vary at different stages. At first it contains the typical stomach content, acid in reaction, not bile stained and composed largely of a watery fluid mixed with partially digested food. This primary vomiting gives only short relief and is soon followed by smaller amounts of bile-stained alkaline material, evidently the content of the bowel between the stomach and the point of obstruction.

After this initial cleansing of the proximal intestine has been completed the frequency of vomiting and the character and amount of vomitus depends entirely on the fluid intake. If the dogs are allowed to drink all of the water they desire, the vomiting is very frequent, from every ten to fifteen minutes, profuse in amount and consisting entirely of a clear, watery mucus, alkaline in reaction. At first sight it seems evident that the dogs are losing a large quantity of fluid by profuse intestinal secretion and this undoubtedly accounts for the prevalent belief that death in obstruction is due to dehydration. An accurate measurement of the fluid intake and the amount of vomitus shows, however, that the two are practically identical. Over 90 per cent. of the vomitus is ingested water and the remainder is thick, mucous intestinal secretion. Thus, with each attack of emesis the animal does not lose more than from 10 to 20 c.c. of fluid.

On the other hand, if the dogs are allowed no fluids by mouth the picture is greatly altered. The frequency of vomiting is markedly decreased, usually occurring only about once an hour. However, when the vomiting spells do come on they are much more severe, consisting almost entirely of a five to ten minute period of intense retching followed by the regurgitation of from 20 to 30 c.c. of a thick, tenacious, stringy mucus. This slimy material is the typical secretion of acute strangulation and it varies little, either in character or amount, from this point until death. Stercoraceous or fecal vomiting is never seen. This substantiates the belief that fecal vomiting never occurs in obstruction, but rather that the foul smelling fluid in low simple obstruction is due to the bacterial decomposition of the stagnant intestinal secretions accumulating in the proximal segment.

An analysis of this characteristic strangulation secretion, which begins within forty-five minutes after the obstruction is produced, reveals the following facts: Physically it is a clear, yellowish viscid fluid resembling egg albumin. Chemically it is mucin, a glycoprotein, secreted by the goblet cells of the intestinal mucosa. Titration tests for proteolytic and lypolytic enzyme action are practically always negative, thus indicating a complete absence of pancreatic juice and succus entericus. After the first vomitus of stomach contents the regurgitated fluid is always alkaline and gives a negative test for traces of free and combined hydrochloric acid. Gmelins' and Pettenkofers' tests for bile pigment are always negative. At necropsy the gallbladder is never distended and usually contains but little bile.

Kidney excretion shows a similar inhibition. Practically all dogs dying fifteen hours or less have an almost complete postobstructive anuria. Micturition does not occur and at necropsy the bladder is usually empty. The increase in blood urea and nonprotein nitrogen previously noted is evidently largely due to retention.

SUMMARY

The entire picture then reveals the following facts:

1. That in acute strangulation dehydration is an almost negligible factor, the total intestinal secretory loss being less than 200 or 300 c.c. Urinary secretion is almost nil.

2. There is no appreciable loss of bile, pancreatic, intestinal, or gastric juices, but rather there is a total inhibition of these secretions.

3. The absence of urine and the extreme fall in blood pressure indicate that the kidney and adrenals suffer in a like manner from this extreme systemic depression.

4. The absence of hydrochloric acid in the vomitus plus the relatively normal blood chlorids and the absence of alkalosis rules out of consideration hypochloremia as a lethal factor.

COMMENT

A careful clinical comparison of patients with simple obstruction and acute strangulation shows that the two processes are radically different. The former lives from three to eight days without food or water, dying eventually either of (1) inanition and dehydration, or (2) peritonitis following perforation. Collapse symptoms are not present until the last day. The course is a slow, gradually progressive one which terminates fatally, in toxemia or collapse. On the other hand, strangulation is an acute affair characterized by profound collapse and resulting in death usually in less than thirty-six hours. In a previous paper we have shown that experimentally and clinically this classification is justified.

Acute strangulation has been further subdivided into (1) long loop obstruction and (2) short loop obstruction. Clinically, the first group is represented by volvulus, and extensive internal strangulation, the second by strangulated inguinal, femoral and umbilical hernias and intussusception. We have shown that in both of these groups the course is so rapid that hypochloremia, alkalosis, dehydration and inanition do not have time to develop. It is thus evident that the usually assumed causes of death in simple obstruction play little or no part in acute strangulation.

With long loops the predominant lethal factor is shock. This is indicated by the early and extreme fall in blood pressure, fast thready pulse, rapid sighing respirations, marked thirst, subnormal temperature, and pronounced muscular weakness. The rapid recovery following the release of the strangulation and the slight toxicity of early loop fluid further verifies this statement. The factors producing this shock are as follows: (1) excruciating pain due to the constant stretching of the visceral and parietal peritoneum by violent peristalsis and abdominal

distention; (2) low blood pressure and its direct effects, namely, diminished rate and volume of blood flow, capillary stasis, decreased metabolism, glandular inactivity, etc.; (3) blood loss from hemorrhage into the bowel lumen.

If the loops are short, shock, although present, is not of sufficient intensity to cause death. The dogs withstand this primary systemic depression and thus sufficient time is allowed for toxemia and peritonitis to develop. The poisonous products responsible for the development of this toxemia are not formed in the intestine above the obstruction but are produced by bacterial decomposition within the strangulated loop.

In brief, then, death in acute intestinal strangulation is due to a varying combination of shock and toxemia. The preponderance of one or the other of these causes being largely determined by the length of bowel involved and the degree of venous occlusion.

A point of considerable clinical interest is the fact that an intestine segment when strangulated always stretches two or three times its former length. For example, a loop 12 inches (30 cm.) long at operation shows at necropsy a length of 30 inches (76 cm.) or more. It is thus evident that the measurements given in surgical reports of gangrenous bowel resection do not represent the actual length of intestine involved.

CONCLUSIONS

1. Dehydration, hypochloremia, alkalosis and inanition are not present to any appreciable degree in acute intestinal strangulation.
2. The blood chemistry estimations show a considerable increase in urea and nonprotein nitrogen. The other constituents remain practically normal.
3. The length of the strangulated segment determines the degree of shock, toxemia and the rapidity with which death ensues.
4. The loop fluid of long segment strangulation is relatively nontoxic when injected intraperitoneally.

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