ACUTE INTESTINAL OBSTRUCTION

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During the last two decades, a vast amount of interesting work has been done on acute intestinal obstruction in an endeavor to find the lethal factors involved. Beyond keeping the subject alive in the experimental field, much of the work has added little to the ultimate settlement of the questions at issue. Complicated operative procedures, ether and morphin anesthesia, methods used because of preconceived premises of toxic substances, and the failure to recognize different types of obstruction have been the main stumbling blocks.

In the preceding papers,[1] we have demonstrated that experimental obstruction produced under local anesthesia gives most ideal results. The postoperative animals are in practically the same condition as the human patients seen clinically. The only complicating factors in the experimental work are the procain anesthesia and the 3 inch (7.6 cm.) incision through the linea alba.

Carefully checked control animals on which only the abdominal incision has been made show that these two factors cause little or no systemic reaction.

Obstruction thus produced demonstrates clearly that there are two types of acute intestinal occlusion: (1) Acute strangulation. This includes those cases in which there is an interference with the venous, arterial and lymphatic circulation in the bowel wall and mesentery as well as complete obstruction of the intestinal lumen. Under this heading are grouped volvulus, strangulated hernia and intussusception. This condition is characterized by rapid pulse and respiration, subnormal temperature and low blood pressure, as well as the usual obstruction symptoms. Death here is due to shock, toxemia and peritonitis. (2) Acute simple obstruction. This comprises those cases in which there is a complete blockage of the bowel lumen only, with practically no circulatory involvements. Here the obstruction is produced by gall-

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1. Hausler, R. W., and Foster, W. C.: Studies of Acute Intestinal Obstruction, I, Different Types of Obstruction Produced Under Local Anesthesia, Arch. Int. Med. **34**:97 (July) 1924; II, Acute Strangulation, ibid. **34**:697 (Nov.) 1924.

stones, enteroliths, foreign bodies, adhesions and bands. The temperature, pulse, respiration and blood pressure show no marked deviation from the normal, even after ten days, although the obstruction symptoms are very pronounced.

Acute strangulation has been dealt with in a previous paper. In this paper, we shall endeavor to show that death in simple intestinal obstruction is not due to a toxemia, as is generally assumed, but instead is due to starvation.

In experimental simple obstruction, occlusion of the bowel lumen is usually produced by clamps, ties or section of the intestine and inversion of the cut ends. All three of these methods have their disadvantages and introduce certain complications which must be considered when interpreting the results obtained. The ideal method of producing obstruction is naturally that seen in human cases, namely by bands of living tissue. The aluminum clamps, as first used by Hartwell and Hoguet,[2] are not very satisfactory because of the difficulty in determining definitely whether the obstruction has been complete. Tissue destruction, abscess formation and peritonitis are rather frequent complications. Ligation of the intestine, with silk or cord ties, produces good results for several days, but unfortunately the ligature cuts through the intestinal wall in four or five days, and the bowel reunites or death follows from perforation and peritonitis. Section of the intestine and inversion of the proximal and distal stumps gives a complete obstruction that is easily produced and very permanent. This technic, however, causes considerable tissue destruction and is followed by a severe constitutional reaction. The postoperative record shows that for the first five or six days, or until the inverted stumps have healed, there is a quite pronounced rise in temperature, pulse and respiration. Unless subcutaneous injections of fluid are given during this reparative period, many of the animals die. However, if the dogs survive, we have an ideal obstruction exactly comparable to that seen in human cases. The obstruction is complete, and little or no devitalized tissue is present. The writers have used all three methods and find that the section method gives the most satisfactory results.

LITERATURE

No attempt will be made to review the extensive literature on the subject of intestinal obstruction. Instead, the reader is referred to a recent article by Ellis.

The literature shows five main theories as to the cause of death in acute intestinal obstruction:

1. Bacteremia. The theory that death is due to a bacteremia at first sight seems very plausible. McClure [3] has shown that the fluid accumu-

2. Hartwell, J. A., and Houget, J. P.: An Experimental Study of High Intestinal Obstruction, Am. J. Med. Sc. **143**:357, 1912.

3. McClure, R. D.: An Experimental Study of Intestinal Obstruction, J. A. M. A **49**:1003 (Sept. 21) 1907.

lating in the intestines above the obstruction acts as a very good culture medium. It furnishes all the requirements for bacterial growth, i. e., food, water, proper temperature and an alkaline reaction. Furthermore, the normal intestinal tract is the habitat of innumerable pathogenic bacteria. Under such ideal conditions, they multiply very rapidly and thus might easily work their way through the intestinal mucosa into the blood stream.

However, Krafft,[4] McClure,[3] and Hartwell and Hoguet [2] have definitely disproved this theory by showing that at death the heart's blood, peritoneum and organs are practically always sterile.

2. Perverted secretion. Supporters of this theory state that, after obstruction, the intestinal mucosa of the proximal segment secretes a toxin which when absorbed is rapidly fatal.

Dragstedt, Moorhead and Burcky [5] showed the fallacy of this theory by draining open isolated duodenal and jejunal loops into the peritoneal cavity. Their dogs lived indefinitely and showed no toxic symptoms.

3. Shock theory. The clinical evidence of tachycardia, low blood pressure and profound collapse, combined with wide dilatation of all splanchnic vessels, point to severe disturbance of the cardiovascular system.

These symptoms are true indications of shock and, as previously stated, appear in practically all cases of acute strangulation.

The protocols of dogs with simple intestinal obstruction, produced under local anesthesia, show an almost complete absence of shock symptoms. The pulse and respiratory rates are always practically normal; the temperature curve shows only the usual diurnal variation, and the blood pressure remains the same even after twenty days.

Shock, however, is an acute affair always manifesting its symptoms within the first twenty-four hours. These simple obstruction dogs live three or four weeks. It is thus evident that shock is not a lethal factor in simple intestinal obstruction.

4. Toxemia. This theory states that death in acute intestinal obstruction is due to the absorption of poisons produced by bacterial action on the stagnant bowel fluids which accumulate in the intestine above the obstruction. This theory, which has received almost universal

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4. Krafft, quoted from Enderlain, Hotz: Mitt. a. d. Grenzgeb. d. Med. u. Chir. **23**:755, 1911.

5. Dragstedt, L. R.; Moorhead, J. J., and Burcky, F. W.: Experimental Study of Intoxication in Closed Intestinal Loops, J. Exper. Med. **25**:421 (March) 1917.

acceptance, has been supported by experiments demonstrating that death is quickly produced when these fluids are injected intravenously into normal animals. Thus far, no one has been able to demonstrate toxins in the blood stream or their absorption by the intestinal mucosa.

5. Dehydration. In 1912, McLean and Andries [6] and Hartwell, Hoguet and Beekman,[2] each group working independently of the other, concluded that the water loss from drainage of the body fluids into the intestinal lumen above the obstruction was the cause of the systemic symptoms and the eventual cause of death.

This dehydration theory seems very plausible, since clinically we know that the mechanical obstruction causes an almost immediate vomiting of all foods and liquids taken, and, in addition, that the profuse vomitus contains a considerable quantity of bile, pancreatic, gastric and intestinal secretions.

The last two theories have never been definitely proved or disproved.

METHODS

The operative procedures used were substantially the same as those described in the preceding papers. All work was done on healthy dogs under aseptic technic. Blood chemistry estimations were made at frequent intervals by the methods previously reported. Blood pressure readings were made on the femoral artery under local anesthesia.

EXPERIMENTAL WORK

Rôle of Toxemia.—In our previous work on acute intestinal strangulation, it was noted that even massive doses of ten hour strangulated loop fluid was relatively nontoxic when injected intraperitoneally into normal dogs. This bloody exudate is an ideal culture media, even more so than intestinal secretions.

It thus seemed probable that the vomitus of simple obstruction was likewise nontoxic, especially since dogs of the simple obstruction group vomit at least three or four times a day, and thus prevent prolonged bacterial action on the accumulating fluid. Most high obstruction vomitus is not exposed to bacterial action for more than a twelve hour period. Selective action of the intestinal mucosa and vomiting also prevent much toxin absorption.

To determine, therefore, the relative toxicity of high obstruction vomitus, the total daily vomitus of four dogs with simple duodenal cord tie obstruction was collected, centrifugated, filtered through ordinary filter paper and slowly injected subcutaneously into normal dogs of approximately the same size. All vomitus was prepared, and injected as

6. McLean, A., and Andries, R. C.: Ileus Considered Experimentally, J. A. M. A. **59**:1614 (Nov. 2) 1912.

soon as possible after it was obtained in order to prevent further bacterial action. The vomitus was not heated because of the objection that this might destroy the toxins as well as the bacteria. However, by this technic we were injecting millions of bacteria into the subcutaneous tissue of these dogs. Apparently, dogs have good resistance to bacteria since only one of these dogs developed skin abscesses following the three days' injection period.

Table 1 shows the most severe reaction noted.

Two days later, the dog had recovered entirely.

The experiments in Table 1 show practically no toxic effect from the absorption of this high obstruction vomitus, and we therefore conclude

TABLE 1.—*Data Proving That High Duodenal Obstruction Fluid Is Relatively Nontoxic*

Dog 1, a male, weight 16 pounds (7.3 kg.). Prepared vomitus injected subcutaneously.

Day	Time	Vomitus	Result
1	9 a. m.	60 c. c.	Vomitus appeared to have no effect on dog
	12	60 c.c.	
	1 p. m.	60 c.c.	
	3	60 c.c.	
	5	60 c.c.	
2	9 a. m.	50 c.c.	Temperature, 103.2
	12	65 c.c.	Dog slightly indisposed
	1 p. m.	60 c.c.	
	3	60 c.c.	
	5	60 c.c.	
3	9 a. m.	65 c.c.	Temperature, 103
	12	60 c.c.	Dog looked sick; evidently had bacteremia from injections
	1 p. m.	50 c.c.	
	3	50 c.c.	
	5	50 c.c.	

that even the total daily vomitus is relatively nontoxic when injected subcutaneously, as soon as possible after it is obtained. Since we know that the fluid current is mainly out of the body by way of the upper intestinal tract and is very large, it is evident that only slight absorption of toxins can occur.

Death from toxemia thus seems very improbable. What then is the cause of death? Perverted secretion and bacteremia have been disproved. Shock, we have shown, is not present. Starvation and dehydration appear to be the only remaining possibilities.

Rôle of Dehydration.—Most workers assert that, since death is early and vomiting almost negligible, dehydration cannot be a prominent lethal factor. Their operations were invariably under ether anesthesia, and the animals usually died in from three to five days. No fluids were given subcutaneously, and little vomiting was noted.

On the other hand, in the experimental work of Hartwell and Hoguet,[2] Bacon and Anslow and Eppler,[7] reports are made of the

7. Bacon, D. K.; Anslow, R. E., and Eppler, H. H.: Intestinal Obstruction, Arch. Surg. **3**:641 (Nov.) 1921.

marked daily fluid output and loss of body weight in dogs of the simple obstruction type. They, however, had given daily subcutaneous injections of large quantities of saline solution and, under these conditions, the vomitus and urinary excretions were naturally much larger than they would have been had they allowed the body fluids to be gradually depleted, as they are in the ordinary cases.

With this fluid loss in mind, the clinical literature was examined for reports of total fluid output in human cases of obstruction, but none were found.

TABLE 2.—*Fluid Loss in Low Duodenal Obstruction*

Dog	Weight	Days	Urine and Vomitus	Result
1	18.5 pounds (8.4 kg.)	1	180 c.c.	Dead in 60 hours from perforation and peritonitis
		2	520 c.c.	
	15.4 pounds (7 kg.)	3	100 c.c.	
2	43 pounds (19.5 kg.)	1	610 c.c.	Recovered after 90 hours
		2	415 c.c.	
		3	180 c.c.	
	37.1 pounds (16.8 kg.)	4	95 c.c.	
3	24 pounds (10.9 kg.)	1	285 c.c.	Died after 68 hours from perforation
		2	335 c.c.	
	19.2 pounds (8.6 kg.)	3	950 c.c.	
4	20 pounds (9 kg.)	1	375 c.c.	Recovered on third day
	19.1 pounds (8.6 kg.)	2	275 c.c.	
		3	195 c.c.	
	17.2 pounds (7.8 kg.)			
5	11 pounds (5 kg.)	1	100 c.c.	Recovered on third day
	10.1 pounds (4.5 kg.)	2	115 c.c.	
	9.5 pounds (4.3 kg.)	3	80 c.c.	
6	20.4 pounds (9.2 kg.)	1	210 c.c.	Recovered on fifth day
	19.9 pounds (9 kg.)	2	180 c.c.	
	19.3 pounds (8.7 kg.)	3	240 c.c.	
	18.9 pounds (8.6 kg.)	4	130 c.c.	
	18.1 pounds (8.2 kg.)	5	65 c.c.	
7	30.5 pounds (13.8 kg.)	1	250 c.c.	Recovered on fourth day
	29 pounds (13.2 kg.)	2	185 c.c.	
	28.3 pounds (12.8 kg.)	3	195 c.c.	
	27.8 pounds (12.6 kg.)	4	105 c.c.	

We therefore experimented on a series of seven dogs with simple duodenal and ileal obstruction; an accurate record was kept of daily fluid loss by urine and vomitus. Operations were performed, under local anesthesia, to prevent the usual atonic postoperative conditions of the bowel. The dogs were kept in metabolism cages so constructed that all fluid excretions could easily be collected. The obstruction was produced by ligating the bowel with a heavy cord tied just tight enough completely to block the bowel lumen. These ties cut through the bowel wall at the end of four or five days, and the vomiting immediately ceased, thus indicating that the obstruction had been relieved.

These results, although observed over only a short period of time, show that fluid loss is quite pronounced.

In another series of six dogs in which obstruction was produced by section of the intestine and stump inversion, this daily fluid loss was replaced by subcutaneous injections of physiologic sodium chlorid solution. The results were as follows: One dog died of snuffles on the fifth day; another was killed on the twelfth day because of a large skin abscess, and the remaining four lived fourteen, sixteen and seventeen days, respectively.

Table 3 shows the usual temperature, pulse and respiratory variations noted.

The foregoing results confirmed the work of Hartwell and Hoguet,[8] who first showed that, in simple intestinal obstruction, if the fluid loss

TABLE 3.—*Typical Temperature, Pulse and Respiration in Obstruction of Lower Jejunum*

Dog 2, weight 55 pounds (24.9 kg.), a large, black male.

Days Before	Rectal Temperature	Pulse	Respiration	Blood Pressure	Sodium Chlorid, C.c.	Result
1	101.4	84	18	150		
1	99.2	86	20		250	
2	101.0	126	18		250	
3	102.2	102	22		300	
4	102	88	20		300	
5	102.8	84	18		300	Slight nasal discharge
6	102.4	88	20		300	
7	102	76	18		350	
8	101.9	90	10		250	
9	101.1	66	18		350	
10	100.4	58	16		460	Snuffles worse
11	100.4	56	16		350	
12	100.4	54	20		460	Snuffles better
13	101	58	22		350	
14	100.6	74	18		360	
15	101	92	20		340	
16	100.4	172	20	100 systolic	300	
17		Died				Weight 41 lbs. (18.6 kg.)

Necropsy: Obstructed viscera grossly normal; no peritonitis; proximal and distal stumps well healed; lower 6 inches (15.2 cm.) of proximal intestine dilated slightly, and walls hypertrophied.

is replaced, the animals will live two or three weeks. They also indicated that fluid loss is a very important lethal factor in simple intestinal obstruction. That dehydration, however, is the sole cause of death seems very impropable. It certainly does not explain the rapid death obtained in experimentally produced obstruction. Fluid loss through vomitus and excretions is never sufficiently pronounced in obstruction to cause death in one, five or even ten days. All high obstruction dogs died in less than a week unless given fluid subcutaneously. It seemed apparent, therefore, that in experimental work there must be some complicating factor due to the operative procedure.

A careful review of the literature showed that, of the three methods used in producing obstruction, only one gave satisfactory results. Cord

8. Hartwell, J. A.; Hoguet, J. P., and Beekman, F.: An Experimental Study of Intestinal Obstruction, Arch. Int. Med. **13**:701-736, 1914.

tie and clamp occlusion were too uncertain and too temporary, and perforation and peritonitis were very common. With section of the intestine and inversion, the obstruction was very satisfactory, but the animals died in about five days unless given fluids subcutaneously. It appeared evident, therefore, that in the latter method the time during which healing of the inverted raw ends was taking place was a critical period. The animals that survived this stage should give ideal experimental results.

Consequently, on a series of four dogs, cut and inversion obstruction was produced, and physiologic sodium chlorid solution was given subcutaneously, during the first five days, to tide them over this reparative

TABLE 4.—*Simple Obstruction of Lower Duodenum*

Dog 4, an adult, female collie, weight 34.2 pounds (11 kg.).

Days	Temperature	Pulse	Respiration	Vomitus, C.c.	Saline Solution, C.c.	Chlorids, Mg. per 100 C.c.	Carbon Dioxid	Nitrogen, Mg. per 100 C.c.	Blood Pressure	Observation
0	102.2	110	18	...	...	298	43.2	23	not	
1	103.2	152	24	...	250	...	....	..	taken	
2	102.9	144	24	...	250	...	....	..	...	
3	102.6	148	22	...	240	...	....	..	...	
4	103.1	145	24	...	240	...	....	..	...	
5	104	140	22	...	250	...	....	..	...	Urine scanty during remainder
6	103.6	135	22	...	...	...	....	..	...	of starvation period
7	103.5	140	20	...	...	...	....	..	...	
8	103	138	20	...	...	...	....	..	...	
9	103.1	135	20	...	...	...	....	..	...	
10	102.7	128	16	...	...	...	....	..	...	
11	103.4	128	18	...	...	320	46.2	..	138	systolic
12	102.6	160	12	...	...	...	....	..	...	
13	102.6	164	18	...	...	298	60.49	..	...	
14	102.7	176	16	...	...	...	....	..	...	
15	102.6	176	12	...	...	...	....	..	...	Mucosa of nasal and buccal
16	102.1	172	9	...	...	...	....	..	...	cavity dry and crusted
17	142.1	172	11	...	...	310	53.6	40	...	
18	101.8	176	10	...	...	...	....	..	...	
19	102.7	160	10	150	...	320	36.8	54	...	
20	102.6	165	12	...	...	...	...	..	105	
21	102.1	Killed in afternoon				330	42.5	67	...	

Necropsy (done immediately): Weight 19.3 pounds (8.7 kg.). Abdominal viscera grossly normal; no peritonitis; stumps well healed; walnut sized abscess in mesentery just below proximal intestine. Weights of viscera, as follows: Liver, 272 gm.; kidneys, 76 gm.; spleen, 10.5 gm.; thyroid, 4 gm.; suprarenals, 3.2 gm.; pancreas, 21 gm., and ovaries, 1.4 gm.

period. Complete food and water starvation was then begun. One animal died on the third day from hemorrhage in the proximal segment; another developed snuffles and was sacrificed on the fifth day. The remaining two dogs, to our amazement, lived twenty-one and twenty-eight days, respectively. In other words, these animals lived sixteen and twenty-three days without food, water or salts, with complete obstruction of the small intestine. Tables 4 and 5 show the post-operative results.

These two animals, especially during the last ten days, showed the typical picture of starvation. The loss of weight was extreme. The skin became dry and inelastic, and the shedding of hair was pronounced.

Folds produced in the skin flattened very slowly. Salivary secretion ceased entirely, and as a result the tongue and oral mucosa became crusted with foul smelling, dry serous placques. The last five days, the nasal apertures became plugged with similar secretions and nasal breathing was harsh and difficult. The skin was extremely anesthetic, so much so that incisions caused no pain. The extremities were cold and the blood flow through them of such small volume that the necessary amount of blood for chemistry estimations was difficult to obtain. The rectal

TABLE 5.—*Simple Obstruction of Upper Jejunum*

Dog 5, an adult, female mongrel, weight, 51 pounds (23.1 kg.).

Days	Temperature	Pulse	Respiration	Vomitus, C.c.	Saline Solution, C.c.	Chlorids, Mg. per 100 C.c.	Carbon Dioxid	Nitrogen, Mg. per 100 C.c.	Blood Pressure	Remarks
0	101.9	90	18	...	...	315	45.2	23	not	
1	103.2	148	18	...	350	...	....	..	taken	Condition good
2	102.6	140	22	...	240	...	....	..	...	Condition good
3	102.1	126	20	...	240	...	....	..	...	Condition good
4	101.8	66	18	...	240	...	....	..	...	Condition good
5	102.9	84	18	...	250	...	....	..	...	Condition good
6	102.2	68	18	...	none	...	....	..	...	Condition good
7	102.3	64	18	...	...	...	....	..	...	Urine scanty
8	101.8	72	16	...	...	...	....	..	...	
9	102.2	68	18	300	...	...	....	..	...	
10	102	88	22	...	...	...	....	..	...	
11	103.3	96	20	130	...	310	60.4	40	143	
12	102.6	98	12	...	...	...	....	..	...	
13	102.4	126	20	250	...	300	66.5	38.2	...	
14	102.6	143	20	...	...	...	....	..	...	
15	101.5	160	18	15	...	...	....	..	...	
16	102.4	156	16	360	...	...	....	..	...	
17	102.1	138	16	180	...	300	56.4	46	...	Nose dry; respiration harsh;
18	101.6	134	17	...	...	...	....	..	...	crusts on nose and lips
19	101.6	114	16	...	...	...	....	..	...	
20	100.4	124	12	...	...	...	....	..	...	
21	100.1	146	14	...	...	270	53.7	45	...	
22	101	150	14	...	...	...	....	..	...	
23	101.2	140	12	...	...	...	....	..	...	
24	102.2	160	14	50	...	290	57	41.6	...	
25	101.8	132	15	...	...	...	....	..	...	
26	101.9	145	16	...	...	...	....	..	112	
27	101.8	160	16	410	...	280	62.7	42	...	
28	102.2	165	18	...	...	...	....	..	...	Sudden death

Necropsy (immediate): All thoracic and abdominal viscera grossly normal; no peritonitis; lower 3 inches (7.6 cm.) of proximal intestine slightly dilated and walls hypertrophied. Distal segment very small and atrophic. Weights of organs, as follows: Liver, 254 gm.; kidneys, 78 gm.; spleen, 12 gm.; thyroid, 3 gm.; suprarenals, 3 gm.; pancreas, 14 gm., and ovaries, 1.2 gm.

temperature was slightly subnormal; the pulse almost doubled in rate, and the respirations were very slow. The blood pressure fell to about 110 mm. of mercury systolic.

Throughout the experiment the dogs were active and playful and, except for thirst, vomiting and the symptoms mentioned, appeared absolutely normal. At no time after the first five days was there any subjective or objective evidence of toxemia. The temperature, pulse and respiratory changes were certainly not those of toxemia, and the appearance and action of the animals were essentially those of a normal animal undergoing starvation.

The red blood cell count was consistently above normal after the first week, the counts varying between six and eight million. Hemoglobin estimations (Haskins-Osgood modification of Sahli) fluctuated between 120 and 135 per cent. Viscosity determinations were made by comparing the time required (stop-watch) for normal oxalated blood to flow from a special 5 c.c. viscosity pipet with that for the oxalated blood of the obstruction animals. This method, although not absolutely accurate, gave approximate results and revealed a quite decided increase in blood viscosity. This increase was also visibly apparent, the blood appearing thick and flowing very slowly. Clotting time was decreased, and no bleeding occurred following small skin incision. A comparison of the foregoing charted blood chemistry figures with those of Dog 5 of Morgulis and Edwards'[9] starvation series (the only animal deprived of both food and water) shows that the changes are almost identical. The chlorids remain normal or slightly increase; the nonprotein nitrogen increases gradually although never to a very high figure, and the carbon dioxid combining power of the blood varies, usually slightly above normal limits. These changes are all indicative of the anhydremia of starvation.

A rather unique case of simple intestinal obstruction was seen by us several months ago and will be reported here, as it represents the type of obstruction produced by gallstones, foreign bodies and enteroliths. The owner of a large Airedale brought the animal to the medical school for treatment. He stated that the dog was poisoned about two weeks before, as a result of eating fresh salmon (common in Northwest). During this time, the dog had vomited all food and water eaten, had lost markedly in weight and strength, and when seen was in a precarious condition. Subcutaneous injections of physiologic sodium chlorid solution were given at once, but with little results, the dog dying on about the eighteenth day after the onset of the obstruction symptoms. At necropsy, a round basalt stone, about 2 inches (5 cm.) in diameter, was found tightly lodged in the lower ileum above 5 inches (12.7 cm.) above the ileocecal valve. The small intestine above this point was markedly dilated, congested and contained a small quantity of fluid. No perforation or peritonitis was present, and the obstruction was complete. This case further substantiates the belief that starvation is the cause of death in simple obstruction.

COMMENT

It has been previously demonstrated that in uncomplicated cases of acute simple intestinal obstruction, bacteremia and perverted secretion

9. Morgulis, S., and Edwards, A. C.: Chemical Changes in the Blood During Fasting and Subsequent Refeeding, Am. J. Physiol. **68**:477 (May) 1924.

of the intestinal mucosa are not the causes of death. Shock, the predominating lethal factor in acute strangulation of a large segment of intestine, is never present in simple obstruction. A review of the temperature, pulse and respiration and blood pressure curves of the accompanying tables shows a complete absence of shock symptoms. This theory, therefore, appears untenable.

That death is due to a toxemia is also very improbable for the following reasons:

1. The relatively slight toxicity of even large quantities of vomitus.

2. The decreased absorption from the proximal segment.

3. The fact that dogs with closed, isolated loops full of extremely toxic fluids live months without showing marked toxic manifestations.

4. The quicker death in high obstruction (in this the function of the intestine is mainly secretory), and the slower death in low obstruction, in which normally much absorption occurs, and in which the intestinal secretions become more toxic because of delayed vomiting and more prolonged bacterial action.

5. Furthermore, the entire clinical picture, namely, the temperature, pulse, respiratory and blood pressure variations and the general appearance of the patients and the experimental animals, certainly is not that of a toxemia.

6. In addition, the fact that, in uncomplicated cases of experimentally produced intestinal occlusion, the subjects live three or four weeks without food or water or any treatment whatsoever proves definitely that toxemia is not the cause of death.

The experimental workers supporting this theory have attempted to prove it by demonstrating that fluid from closed isolated loops is rapidly fatal when injected intravenously into normal animals. By chemical extraction, they conclude that the toxins are proteoses, nucleoproteins and histamin derivatives. Undoubtedly, intestinal secretions become very toxic when exposed to prolonged bacterial action. Death following intravenous administration, however, proves nothing, since it has long been known that many substances are extremely fatal when introduced directly into the blood stream, whereas if allowed to pass through normal intestinal mucosa their poisonous properties are entirely destroyed. Intravenous peptone injections may cause rapid death, yet the absorption of peptone from the normal intestine is, on the contrary, a natural physiologic process.

Furthermore, the absorption or presence of these toxins in the blood stream has never been demonstrated, nor has the introduction of blood from animals dying from obstruction ever produced untoward symptoms or death.

It is thus evident that, in uncomplicated acute simple obstruction, toxemia is not present, and that death is purely the result of starvation. All injected food and water is quickly vomited, and the result is therefore essentially the same as if these substances had been withheld. Fluid loss through urinary and respiratory excretion is probably similar in both starvation and obstruction. In addition, however, the obstructed animals are losing considerable quantities of the various juices secreted into the gastro-intestinal tract. In the normal starving animal, these fluids are all reabsorbed. Gastric secretory loss, as Pawlow has indicated, is negligible after the fifth day of starvation. Pancreatic juice secretion probably shows a similar inhibition. Intestinal and biliary secretion, however, continues throughout the obstruction period, as indicated by the large quantity of bile stained vomitus. Experiments with biliary, pancreatic and intestinal fistulas show that animals can survive these secretory losses for long periods of time, thus indicating that, in intestinal obstruction, this, in itself, does not cause death but instead merely hastens the starvation process.

That dehydration is very pronounced is evident. The dehydration theory, therefore, contains an element of truth. Fluid loss, however, is only a part of complete starvation for, in the latter, both food and fluids are withheld. Ordinary dogs deprived of all food and water live from fourteen to sixty-six days, with an average of about thirty days, whereas, if allowed water but no food, they may live more than four months. These figures indicate that death in simple obstruction falls within the same period of time as does ordinary starvation. The extra fluid loss in obstruction through vomitus merely accelerates the starvation process.

Furthermore, a review of the tables outlining the experiments on the two starvation dogs shows that changes are exactly similar to those reported for normal starvation animals. The increased viscosity of the blood, the high red cell and hemoglobin figures, the decreased blood volume flow through the extremities, the blood chemistry changes, the extreme loss of body weight and the temperature, pulse and respiratory curves have all been previously reported in starvation experiments.

The statement of Werelius that death in obstruction may be due to hepatic insufficiency and to a large amount of nonperistaltic bowel is not confirmed by our experimental work. Fluoroscopic examination of the proximal segments has always shown very active peristalsis even up to the time of death. That the distal intestine also is not aperistaltic, as generally stated, is proved by the fact that these obstruction dogs frequently pass small quantities of the white pasty feces that accumulate in the distal intestine. The vomitus is always darkly bile tinged and, during the last two weeks, appears to contain a gradually increasing amount of this hepatic secretion. At necropsy the fluid remaining in the proximal intestine is blackish green, and the gallbladder is distended with black, syrupy bile. Furthermore, blood sugar and urea estimations

show no extreme variation from the normal, and the clotting time, instead of being increased, is, on the contrary, markedly decreased. Aperistalsis and ahepatism seem to play no rôle in acute obstruction death.

Experiments with obstructed mucosal extract have never warranted serious consideration.

Haden and Orr [10] state that, in high, simple intestinal obstruction, the characteristic changes in blood chemistry are a fall in chlorids, an increase in nonprotein nitrogen and, usually, an increase in the carbon dioxid combining power of the plasma. Since the nonprotein nitrogen increase does not begin until after the chlorids are well depleted, they conclude that the chlorids act as detoxicating agents. They also assume that the toxins are absorbed from the intestinal tract, and suggest that they cause much tissue destruction. An examination of their protocols shows that most of their dogs died within from two to five days after the obstruction was produced, namely, during the healing period, unless treated by subcutaneous saline injection. Their conclusion, therefore, as to characteristic blood chemistry changes, toxin, absorption and tissue destruction appears unwarranted. On the contrary, we find that the blood chemistry changes in untreated, uncomplicated, simple obstruction dogs are almost identical with those reported by Morgulis and Edwards [9] for complete food and water starvation. The chlorids, instead of being decreased, remain normal or show a slight increase. The nonprotein nitrogen increases in the same ratio as that occurring in starvation. The carbon dioxid combining power of the plasma varies considerably, usually somewhat above the normal limits.

CONCLUSIONS

1. Death in uncomplicated cases of acute intestinal obstruction is due to starvation.

2. The blood chemistry changes noted in uncomplicated simple intestinal obstruction are almost identical with those found in complete starvation. Hypochloremia is not present.

3. Dehydration, in uncomplicated cases of simple intestinal obstruction, is usually very pronounced.

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10. Haden, R. L., and Orr, T. G.: Effect of Inorganic Salts on the Chemical Changes in the Blood of the Dog After Obstruction of the Duodenum, J. Exper. Med. **39**:321 (Feb.) 1924.

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