

## AN EXPERIMENTAL STUDY OF HIGH INTESTINAL OBSTRUCTION.

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INTESTINAL obstruction in man, if unrelieved, speedily causes death. The fatal outcome is too rapid to be the result of starvation, and three general theories have been advanced to explain it: (1) A disorder of the nervous mechanism controlling the cardiac and vasomotor systems. (2) A bacterial infection of the organism by the passage outward of bacteria from the intestinal lumen. (3) An intoxication from poisonous substances imprisoned in the intestine orally to the obstruction.

The advocates of these various theories have done a vast amount of experimentation to uphold one or the other, but up to the present time the question remains unsettled.

THE THEORY OF A NERVOUS DISORDER. In favor of the first theory, clinical evidence has been adduced. The tachycardia, the low blood pressure, the profound collapse of the patient, and the wide dilatation of the splanchnic bloodvessels all point to a loss of activity on the part of the medullary nerve centres. The early writers saw in these the result of a reflex disturbance from stimuli acting on the nerve endings in the intestinal wall. It is undoubtedly true, however, that exactly this train of events may arise from a toxemia or a bacteremia without the intervention of any afferent nerve impulses.

Recently, Braun and Boruttau,<sup>1</sup> have modified this theory somewhat, and assign the above symptoms to a disturbance of the circulation resulting from interference, chiefly mechanical, with the inherent nerve plexuses in the intestinal wall. They see a parallel between a gradual bleeding to death and the death in intestinal obstruction. The loss of body fluids resulting from failure of absorption, the enormous outflow of fluid into the intestine, the persistent vomiting, and the stagnation of blood in the splanchnic area lead to an anemia of the brain centres which progresses till these centres cease their activity, and death results. They experi-

<sup>1</sup> Deutsch. Zeitsch. f. Chir., 1908, xevi, S. 544.

mented most exhaustively on rabbits to support this view, but the evidence is all indirect, and seems to us not to amount to a proof of the absence of a toxemia. We are unable to find in the literature any experimental work which is convincing that the nervous system is primarily at fault. Certainly, as death approaches, there is a profound disturbance of the nervous control of the heart, bloodvessels, and respiration, but this, we believe, is in no sense a reflex disturbance, nor the result of an anemia, *per se*, of the nerve centres.

**THE THEORY OF BACTERIAL INFECTION.** The infection theory has much more to substantiate it. The intestinal lumen is the normal habitat of innumerable microorganisms which are potentially pathogenic. When an obstruction exists, these no longer have their normal outlet *per anum*. They are dammed up in the intestine under favorable conditions for their growth (McClure<sup>2</sup>). The damage to the intestinal mucosa may allow their passage outward, either into the blood stream, the lymphatics, or directly into the peritoneal cavity. Proof of this possibility is seen clinically in the presence of a peritonitis as a complication of intestinal obstruction without perforation; also in the frequent finding of the colon bacillus in the blood as a pathogenic microorganism, under very varied conditions of intestinal disturbances less severe than an obstruction, and in the bacteremia from the *Bacillus typhosus* when the intestine is ulcerated by the action of that organism.

Von Khantz,<sup>3</sup> experimenting on rabbits in a low intestinal obstruction, often found a bacterial invasion of the blood and the peritoneum, but never the former without the latter. Borszesky and von Gencsich,<sup>4</sup> on the other hand, found a blood infection with no invasion of the peritoneum. There can be no doubt that a bacterial invasion of both the peritoneum and the blood may take place in intestinal obstruction, but it is not an essential accompaniment. It occurs late as a complication, often a terminal one. Both clinically and experimentally it will result most often when the obstruction is complicated by a strangulation. Without this complication, intestinal obstruction will kill with no passage of bacteria beyond their normal place of occurrence in the bowel lumen.

The tables on pages 362 to 363 give the result of our experiments bearing on this point. In the first series the obstruction was produced by dividing the intestine from 10 to 30 cm. below the pylorus, and closing the ends by inversion. It will be noted that in all these cases some microorganisms were found in the organs examined.

<sup>2</sup> Jour. Amer. Med. Assoc., 1907, xlix, 1003.

<sup>3</sup> Arch. f. klin. Chir., 1909, xxxviii, p. 412.

<sup>4</sup> Beiträge z. klin. Chir., Band xxxvi, S. 448.

This, we believe, was due to two factors, namely, the necessary local soiling of the peritoneum by the method of operating, and the fact that all the examinations were made several hours post mortem, because the animals died during the night. In the second series, however, the obstruction was produced by the clamp described below, and the animals were either killed when it was believed they were too sick to live over night, and immediately examined, or the examination was made very soon after the animal died from the obstruction itself. This series of 7 cases furnishes irrefutable proof that death results from an obstruction of the upper intestine in the dog, without any invasion of the peritoneum, blood, liver, or spleen by bacteria, which are demonstrable by the methods employed. The methods in the two series were exactly similar and their efficiency in detecting organisms is amply demonstrated in the first. The cultures were taken at autopsy on slant agar or Loeffler blood serum or hoth. Usually four tubes, sometimes only three of each were inoculated from each tissue, and grown under both aerobic and anaerobic conditions at incubator temperature. For the anaerobes the Buchner method or the Cornell modification of it was used. The tubes were examined at the end of one, two, and three days. All the cultures were made in the sterile room of the Cornell bacteriology laboratory. This consists of a closed cabinet, ventilated through a bacterial proof filter, in the roof of which is suspended an ordinary revolving lawn sprinkler. The water is turned through this for fifteen or twenty minutes prior to taking the cultures. Thus the air is mechanically sterilized by washing out the microorganisms. All the needed apparatus for taking the cultures and smears is assembled before beginning the inoculations, and the air-tight door is kept closed until all cultures are completed. The technique in making the inoculations is that usually employed in obtaining cultures from postmortem material. The efficiency of the room and the method is demonstrated by the fact that only three air contaminations were found in several hundred tubes.

In some of the later cases of the second series no cultures were taken from the spleen, since it was noted that this organ never showed infection when the liver and peritoneum were without infection. Often, too, the spleen cultures gave no growth when the other organs were actively infected. A summary of this series shows that the femoral blood was cultured seven times and always was without growth; the liver was cultured six times, and showed one colony of Gram-positive cocci, in one tube of one case, probably an air contamination; the results from the peritoneal cultures were exactly the same as from the liver; the spleen was cultured only twice, with negative findings both times; the jejunal or duodenal contents above the clamp were cultured seven times, and all tubes showed an abundant growth of various microorganisms.

SERIES I

Case.	Organs.	Cultures taken.	Tubes showing cocci.	Tubes showing bacilli.	Characteristics.	Remarks.
12	Spleen	2 aerobic 3 anaerobic	None	1 aerobic 2 anaerobic	Morphology and growth characteristic of colon bacillus on usual differential media	Intestine sectioned and closed 34 cm. distal to pylorus. Lived eighty hours. Cultures taken ten hours postmortem.
	Liver	2 aerobic 4 anaerobic	All	All	(1) Gram + coccus. (2) Bacillus identified as colon on differential media. (3) Also very many colonies of a large gram + anaerobic bacillus with morphology of bacillus aerogenes capsulatus.	
	Jejunum	2 aerobic 4 anaerobic	All	All	(1) Gram + coccus. (2) Large anaerobic Gram + bacillus. (3) Gram - bacillus of two kinds, first long and slender; second, short and plump. Both grew in anaerobic tubes, while the former only grew in aerobic tubes.	
13	Femoral blood	4 aerobic 4 anaerobic	None	None		Intestine sectioned 36 cm. from pylorus and end closed. Autopsy about thirteen hours post-mortem.
	Peritoneum	4 anaerobic 4 aerobic	4 aerobic 4 anaerobic	None	(1) Gram + and (2) Gram - coccus.	
	Spleen	4 aerobic 3 anaerobic	1 aerobic	None	Gram + coccus. Same organism as in peritoneum.	
	Liver	4 each	None	None		
	Jejunum	4 each	All	All	Same as in Case 12.	
14	Jejunum at operation	2 aerobic 2 anaerobic	All	All	(1) Gram negative bacillus. (2) Gram + coccus. (3) Large Gram + bacillus	Intestine sutured after section 31 cm. from pylorus. Autopsy thirty-six hours post-mortem.
	Jejunum at autopsy	4 each	All	All	(1) Anaerobic gas forming Gram - bacillus. (2) Gram + bacillus. (3) Gram + coccus.	
	Femoral blood	4 each	2 aerobic	All	(1) Gram positive coccus, ferments lactose, and glucose, without gas, no change in neutral red and saffranin. (2) Large anaerobic Gram + bacillus with morphology of aerogenes capsulatus bacillus.	
	Peritoneum	2 each	All	All	(1) Gram + coccus, same as in femoral blood. (2) Gram - bacillus-like colon bacillus.	
	Spleen	4 anaerobic 4 aerobic	2 aerobic	2 anaerobic	(1) Gram - coccus. (2) Gram - bacillus having the characteristics of coli communis.	
	Liver	2 anaerobic 4 aerobic	2 anaerobic 3 aerobic	None	(1) Gram + coccus as in peritoneum and femoral blood.	

Case.	Organs.	Cultures taken.	Tubes showing cocci.	Tubes showing bacilli.	Characteristics.	Remarks.
15	Jejunum at operation	4 each	4 anaerobic	4 anaerobic 2 aerobic	Gram — bacillus, mobile same as in peritoneum.	Intestine ligated with tape 35 cm. from pylorus. Autopsy four hours post mortem. General peritonitis present.
	Jejunum at autopsy	4 each	1 anaerobic	4 anaerobic	(1) Gram — bacillus having characteristics of colon bacillus (2) Gram — bacillus intermediate in colon group, changes neutral red and safranin, does not ferment lactose. (3) Gram + coccus.	
	Peritoneum	4 each	4 anaerobic	4 anaerobic 4 aerobic		
	Femoral blood	4 each	None	None		
16	Spleen	4 each	None	None	Not identified	Bowel sectioned and inverted 48 cm. from pylorus. Lived about thirty-two hours. Autopsy twelve hours post mortem. No peritonitis.
	Liver	4 each	None	None		
	Jejunum at operation	4 aerobic 4 anaerobic		4 anaerobic 4 aerobic		
	Jejunum at autopsy	4 aerobic 4 anaerobic	4 anaerobic 1 aerobic	4 anaerobic 4 aerobic	(1) Short Gram — bacillus. (2) Gram + coccus resembling streptococcus. (3) Large Gram + bacillus.	
	Femoral blood	4 aerobic 4 anaerobic	None	None	Gram — short, stout, bacillus mobile (2 strains), resembling colon bacillus. (2) Gram + bacillus. (3) Gram + coccus. (1) Gram + coccus. (2) Gram + bacillus. (3) Gram — bacillus, short and stout, mobile. Same as in liver.	
	Spleen	4 aerobic 4 anaerobic	None	None		
	Liver	4 aerobic 4 anaerobic	1 anaerobic 4 aerobic	4 anaerobic 4 aerobic		
	Peritoneum	4 aerobic 4 anaerobic	4 anaerobic 4 aerobic	4 anaerobic 4 aerobic		
17	Jejunum at autopsy	None taken	None taken	.....	.....	Jejunum clamped 37 cm. from pylorus with Crile clamp, which at autopsy was found to have cut through. Autopsy eight hours post mortem, lived about eighty-eight hours. General peritonitis present.
	Femoral blood	4 aerobic 4 anaerobic	None	None	Gram + coccus. One colony outside line of inoculation. Probable air contamination.	
	Spleen	4 aerobic 4 anaerobic	1 anaerobic	None		
	Liver	4 aerobic 4 anaerobic	None	None	(1) Gram — bacillus, having characteristics of coli communis. (2) Gram + coccus. (3) Gram + bacillus short and fat and growing in long chains.	
	Peritoneum	4 aerobic 4 anaerobic	4 anaerobic 3 aerobic	4 anaerobic 3 aerobic		

Case.	Organs.	Cultures taken.	Tubes showing cocci.	Tubes showing bacilli.	Characteristics.	Remarks.
18	Jejunum	None taken	.....	.....	.....	Jejunum clamped with Crile clamp 20 cm. from pylorus. Killed with chloroform ninety-one hours after operation. Clamp cut through, local peritonitis resulting.
	Femoral blood	4 aerobic	None	None		
	Peritoneum	4 anaerobic 4 aerobic 4 anaerobic	None	4 anaerobic 4 aerobic	(1) Gram — bacillus. (2) Gram + bacillus.	

# SERIES II.

19	Jejunum	3 aerobic 3 anaerobic	3 anaerobic	3 anaerobic 3 aerobic	(1) Gram — bacillus. (2) Gram + coccus.	Intestine occluded with special clamp 18 cm. from pylorus. Lived one hundred and seventy-two hours after operation. Killed with chloroform. Autopsy immediately. No peritonitis; local abscess around clamp.
	Femoral blood	4 aerobic 3 anaerobic	None	None		
	Spleen	4 aerobic 4 anaerobic	None	None		
	Liver	4 aerobic 3 anaerobic	None	None		
	Peritoneum	3 anaerobic 4 aerobic 2 aerobic 2 anaerobic	None	None		
	Abscess around clamp		.....	1 anaerobic 2 aerobic	Gram-negative bacilli.	
20	Jejunum	None taken	.....	.....	.....	Occlusion clamp 16 cm. from pylorus. Lived ninety hours. Died. Autopsy. No peritonitis.
	Femoral blood	4 aerobic 4 anaerobic	None	None		
	Liver	4 anaerobic 4 aerobic	None	None		
	Peritoneum	4 aerobic 4 anaerobic	None	None		
	Exudate around clamp	3 aerobic 3 anaerobic	None	None		
21	Jejunum	4 aerobic 4 anaerobic	4 anaerobic 4 aerobic	4 anaerobic 4 aerobic	(1) Gram + coccus. (2) Gram + bacillus. (3) Gram — bacillus.	Occlusion clamp on jejunum 16 cm. from pylorus. Dog lived 68 hours. Autopsy 8 hours postmortem. Peritoneum clean.
	Femoral blood	4 aerobic 4 anaerobic	None	None		
	Spleen	4 aerobic 4 anaerobic	None	None		
	Liver	4 aerobic 4 anaerobic	None	None		
	Peritoneum	3 aerobic 4 anaerobic	1 aerobic	.....	Gram + coccus. One colony only, probably air contamination.	
22	Jejunum	3 aerobic 3 anaerobic	2 aerobic 1 anaerobic	.....	Gram + cocci	Occlusion clamp 12 cm. from pylorus. Lived twenty-one days. Killed with ether. Clamp not absolutely tight. No peritonitis.
	Femoral blood	4 aerobic 4 anaerobic	None	None		
	Liver	3 aerobic 3 anaerobic	None	None		
	Peritoneum	4 aerobic 4 anaerobic	None	None		

Case.	Organs.	Cultures taken.	Tubes showing cocci.	Tubes showing bacilli.	Characteristics.	Remarks.
23	Jejunum	None taken	.....	.....	.....	Occlusion clamp 12 cm. from pylorus. Lived two hundred and forty hours. Killed with cyanide and autopsied immediately. No peritonitis. Clamp tight.
	Femoral blood	3 aerobic 3 anaerobic	None	None		
	Peritoneum	4 aerobic 4 anaerobic	None	None		
24	Jejunum	2 aerobic 2 anaerobic	.....	2 aerobic 2 anaerobic	Gram positive and negative bacilli	Occlusion clamp 16 cm. from pylorus. Lived two hundred and five hours. Autopsied two hours post mortem. No peritonitis. Small abscess around clamp which was tight.
	Femoral blood	3 aerobic 4 anaerobic	None	None		
	Liver	4 aerobic 4 anaerobic	None	None		
	Peritoneum	4 aerobic 4 anaerobic	None	None		
	Clamp Abscess	2 aerobic 2 anaerobic	2 anaerobic 2 aerobic	.....	Pure growth of Gram + coccus.	
26	Jejunum	4 aerobic 4 anaerobic	.....	4 aerobic 4 anaerobic	(1) Small Gram negative bacillus. (2) Large, long Gram + bacillus	Occlusion clamp 16 cm. from pylorus. Lived two hundred and forty hours. Killed with ether. Autopsy immediate. No peritonitis.
	Femoral blood	4 aerobic 4 anaerobic	None	None		
	Liver	3 aerobic 4 anaerobic	1 aerobic	None	One colony outside inoculation of a Gram positive coccus.	
	Peritoneum	4 aerobic 4 anaerobic	None	None		
28	Jejunum	2 aerobic 2 anaerobic	2 anaerobic	2 aerobic 2 anaerobic	Gram positive and negative bacilli and negative cocci. Anaerobic tubes show gas production.	Occlusion clamp 11 cm. from pylorus. Lived fifty-six hours. Autopsy ten hours post mortem. Abscess around clamp. No peritonitis. Clamp tight.
	Femoral blood	4 aerobic 4 anaerobic	None	None		
	Liver	4 aerobic 3 anaerobic	None	None		
	Peritoneum	4 aerobic 4 anaerobic	None	None		
	Clamp abscess	2 aerobic 2 anaerobic	None	None		

Case 22, is not included in the above summary because the obstruction was not complete. It is worthy of note here, however, that the jejunal tube only showed a growth of a Gram-positive coccus in one-half the tubes inoculated, showing that the long period (twenty-one days) with only sterile water by mouth did much toward sterilizing the upper bowel.

**THE INTOXICATION THEORY.** This theory has experimental evidence to support it, and no experiments have proved the absence of a toxemia. Clairmont and Ranzi<sup>6</sup> demonstrated that the stagnated material above an obstruction, after passage through a Reichel or Pukal filter, was exceedingly toxic when injected into the blood of an animal. Kukula<sup>7</sup> found a toxic material in the alcoholic extracts of the intestinal contents in experimental ileus. Roger,<sup>8</sup> and Roger and Garnier<sup>9</sup> went farther and proved that the normal content of both the stomach and the intestine are poisonous when thus injected. They did not filter it, but death occurred too early to be the result of an infection. The toxicity was only one-third as great when injected into the portal vein as when injected into a peripheral vein. They also injected it into a mesenteric artery, against the current, so that it was carried backward and then redistributed through branches to a considerable section of the intestine. Here it produced emboli and stopped the absorption, so that large doses were not fatal. This power to cause clotting in the vessels of the intestine is mentioned in this connection because it may account for the gangrene resulting in some of our experiments (see page 386). Murphy and Vincent<sup>9</sup> found the material from the obstructed or strangulated intestine very poisonous when injected into the peritoneal cavity, and that its poisonous properties were destroyed by boiling or passage through a Berkefeld filter. They concluded, therefore, that living bacteria are the important factors. It does not seem to us that this conclusion is tenable. Their experiments only prove that living bacteria, if they obtain entrance into the peritoneum, will kill. They do not disprove the presence of other poisons in the obstructed intestine, which, being slowly absorbed over a period of some hours, or days, will kill with the symptoms of ileus. Such poisons may be constantly elaborated and absorbed, but may never be present at any one time in sufficient quantity to cause death when injected into the peritoneal cavity. The symptoms arising from the injection of the intestinal contents into the blood, as reported, differ very markedly from those appearing in the obstructed animal, and there are many objections as pointed out by Guibe,<sup>10</sup> Braun and Boruttau,<sup>11</sup> Korentchevsky,<sup>12</sup> and others, against accepting these experiments as directly bearing on the cause of death in intestinal obstruction. The lessened absorption from the obstructed intestine is one objection offered. Clairmont and

<sup>6</sup> Archiv f. klin. Chir., 1904, Band lxxiii, S. 696.

<sup>7</sup> Ibid., Band lxxiii, S. 773.

<sup>8</sup> La Revue Scientifique, January 10, 1907, vol. i; La Presse Médicale, January 4, 1911.

<sup>9</sup> Archiv. de med. Experiment, July, 1906; Revue de Médecine, August 10, 1906; La Presse Médicale, May 23, 1906.

<sup>10</sup> Boston Med. and Surg. Jour., November 2, 1911.

<sup>11</sup> La Presse Médicale, April 3, 1909.

<sup>12</sup> Roussky Vratcheb, 1908, p. 1572. (Cited by Guibe.)

<sup>11</sup> Loc. cit.



Ranzi<sup>13</sup> found that potassium iodide placed in the bowel above the obstruction, was excreted from the kidneys in greater quantities than normal, up to eight or ten hours after the obstruction was produced, the excretion rapidly decreased after that, and came to a stop in about fifteen hours. Braun and Boruttau<sup>14</sup> gave strychnine to their obstructed animals, and from the resulting symptoms they concluded that absorption became markedly reduced from the start. On this experiment they base their chief objection to the intoxication theory. However, as we shall show in our experiments, the development of symptoms is very slow, and death, under proper precautions to exclude damage to the intestinal wall, may be long postponed, so that a minimal absorption of poison is all that is required. A more important objection, than the lessened absorption, to using the evidence of the injection experiments as a proof of the intoxication theory, is that many substances injected directly into the circulation are poisonous, whereas, if they have to pass through living cells to get into the blood, their poisonous properties are entirely destroyed. Peptone is such a substance, and is a good illustration in this connection. The injection of peptone into the blood produces symptoms of poisoning, which may end in death. The absorption of peptone from the intestine is, on the contrary, a normal physiological process. Therefore, the mere presence of substances in the obstructed intestinal tract, which act as poisons when injected into the blood, is no indication that these substances cause the death resulting from an obstruction, since they are normally found in the intestine in conditions of continued health. To accept the view that such a death results from a toxemia it must be conceived either that new poisons are elaborated and absorbed or that an abnormal absorption of the poisons normally present takes place. In undertaking our work, after a study of the literature, we tentatively accepted the first hypothesis and planned our experiments to determine by a process of exclusion from what sources such new poison or poisons arise. In this endeavor we were only partially successful, but as the work developed the second conception was forced upon us, namely, that the essential factor in causing the symptoms and death in intestinal obstruction does not lie in the poisons *per se*, but in the production of lesions which favor their abnormal absorption. We believe our readers will get a clearer understanding of our findings if he will follow, step by step, the reasoning and experiments which led to this change of conception in our minds.

In searching for the source of the poison we accepted three possibilities: (1) Foodstuffs or substances derived from them; (2) true bacterial toxins; (3) secretory substances from the ali-

<sup>13</sup> Loc. cit.

<sup>14</sup> Loc. cit.

mentary tract and digestive glands, or their derivatives. It was our intention to exclude one after the other of these possible sources and study the conditions resulting after an obstruction was produced. It is readily conceivable that there may enter into all of these the result of bacterial activity. While, as detailed above, our experiments have shown that a bacteremia is not necessarily present, they have not excluded either a bacterial toxin or substances developed by virtue of the presence of bacteria in the occluded bowel. Indirectly, however, there is some clinical and experimental evidence that bacteria are not an essential in the process. Clinically, we know that a high obstruction produces much more severe symptoms, and is more rapidly fatal, than a low obstruction. The bacteria, however, are much more numerous in the lower bowel than in the upper. Roger and Garnier<sup>15</sup> found the most toxic substance in the duodenum; the least in the colon. The toxicity, too, was greater in unobstructed conditions, than in the obstructed conditions, and it grew less the longer the obstruction continued, while McClure<sup>16</sup> has shown that the bacteria increase rapidly when an obstruction is produced. Vidal<sup>17</sup> found that the toxicity could be decreased by injecting, simultaneously with the duodenal contents, glycerin extract from the jejunal mucosa lower down, and this cannot be explained on the ground that bacteria are the exciting cause of the poison. V. Baracz<sup>18</sup> found that dogs with a double occlusion of the lower ileum and cecum might live for many weeks. At the end of this time only a moderate quantity of material was found in the loop. The contents were rich in bacteria. The intestinal wall showed marked changes; mostly in the form of an hypertrophy, but sometimes an atrophy was present. The microscopic findings are not given. In one case the animal was killed after four hundred and twenty-five days while in a condition of perfect health. Here the bowel loop, about 20 cm. in length, was found enormously distended, filled with 365 grams of a foul-smelling, greenish material, which was rich in bacteria; a bacillus, the *Bacterium aërogenes* and a leptothrix being isolated in cultures. These cases seem to point to the conclusion that the stagnation of bacteria and their toxins in the intestine is not sufficient to cause the symptoms seen in intestinal obstruction. Indeed, they, with the facts cited immediately above, seem to make it doubtful whether microorganisms play any essential part, either directly or indirectly, in producing these symptoms. This question, however, must remain an open one for the present, and the writers can offer no positive answer to it.

There still remain the stagnated foodstuffs, and the stagnated glandular secretions of the stomach, intestine, liver, and pancreas

<sup>15</sup> Loc. cit.

<sup>16</sup> Loc. cit.

<sup>17</sup> *Revue de Chir.*, October 10, 1900, xxii; *Congres. Français de Chir.*, 1905, xxviii, 1237.

<sup>18</sup> *Archiv f. klin. Chir.*, Band lviii.

as the source of the poisonous materials. Draper,<sup>19</sup> of Rochester—formerly Maury of New York—has worked on the theory that the duodenum secretes a substance or substances which normally are rendered non-toxic by passing over the jejunal mucous membrane lower down. When obstruction is present the duodenal substances are not brought into contact with the antibodies, and, hence, remain poisonous and cause death. Vidal earlier advanced this view.

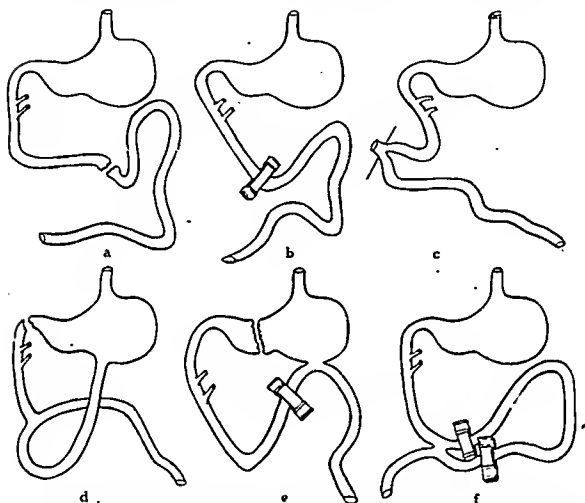


FIG. 1.—Showing the types of operation done: a, obstruction by sectioning and inverting the cut ends; b, obstruction by means of the special clamp; c, attempt to produce sterile condition of gut by fistula and washing; d, elimination of duodenal contents from upper bowel; e, double occlusion of one-half of the stomach and the duodenum with reestablishment of the alimentary canal; f, the same in the small intestine distal to the duodenum.

Draper has done some elaborate experiments in the way of side-tracking the various secretions to support this theory, but his reports are somewhat contradictory, and he himself says very inconclusive. Recently he has reported that feeding dogs which have an obstruction just distal to the duodenum, with the excised mucous membrane from the jejunum and ileum of other dogs,

<sup>19</sup> Jour. Amer. Med. Assoc., October 21, 1911; Johns Hopkins Hosp. Bull., 1909, p. 838; AMER. JOUR. MED. SCI., vol. cxxxvii, p. 725, and Jour. Amer. Med. Assoc., 1910, liv, 5.

prolongs the life of the obstructed dogs. The difference between the control dogs and the fed dogs, however, was not marked, and the exceptions where a fed dog died in a shorter time, or one not fed lived a longer time, were too numerous to make the total result at all convincing.

The possible relation of the stagnated foodstuffs to the production of the poisons was studied in the following experiments. The type of operation is shown in Fig. 1.

#### EXPERIMENTS.

All animals were fully etherized before operation. No food was given in any case after the operation, unless so stated. Water, however, was given freely, unless otherwise noted.

No. 3. A cat which had received no food for ninety-six hours before operation. Duodenum sectioned and ends closed by inversion 10 cm. below pylorus. For three days the animal remained in good condition. Vomited. Fourth day showed marked weakness and some rigidity of muscles of extremities. Just before death had convulsions involving extremities. Killed with chloroform ninety-six hours after obstruction. Autopsy immediate. No peritonitis. Closure of bowel ends tight. Stomach contained small amount of normal appearing gastric content. Duodenum empty. Neither stomach or duodenum was distended.

No. 4. Fox terrier. Fed twenty hours before operation. Repetition of No. 3, the section being 11 cm. distal to the pylorus. Except for some weakness and vomiting, seemed like normal dog for four days. Died with progressive weakness and vomiting on seventh day. Autopsy. No intestinal leakage. No peritonitis. Stomach slightly distended. Duodenum not distended.

No. 5. Bitch. Weight 9.8 kgr. Fed last time forty-eight hours before operation. Repetition of No. 3. Post-operative course the same. Lived one hundred hours. Autopsy. No intestinal leakage. No peritonitis. No distention of stomach or duodenum.

No. 6. Bitch. Weight 10.85 kgr. Starved for seventy-two hours. Repetition of No. 3, section being 12 cm. below pylorus. Post-operative course the same. Temperature varied from 36.6° to 39° C. Lived for fifty-five hours. Autopsy. Same findings as those given above.

No. 7. Dog. Weight 19.7 kgr. Fed last time forty-eight hours before operation. Same operation 16 cm. below pylorus. Some peritoneal soiling with intestinal contents. Post-operative course same as others, except more acute. Temperature as high as 39.9° C. Lived only thirty-six hours. Autopsy yielded same findings as others.

No. 8. Dog. Medium weight. Fed six hours before operation. Same operation 12 cm. below pylorus. Post-operative course the same. Temperature 39°. Lived one hundred and eighteen hours. Autopsy. Same findings and in addition there were noted three superficial areas in the duodenum which resembled ulcers.

No. 9. Leakage of intestinal contents at closed end of proximal gut.

No. 10. Same.

No. 11. Dog. Weight 5.3 kgr. Fed twenty-six hours before operation. Same operation 33 cm. below pylorus. Lived only ten to fourteen hours. Autopsy. No leakage. No peritonitis. Stomach was not distended. Mucosa showed small punctate hemorrhages. Intestine proximal to section and for 80 cm. distal to it was much congested and showed hemorrhagic areas in it. We are unable to explain these last postmortem findings as being the result of the operation. Our belief, however, is that the early death resulted because of them.

No. 12. Bitch. Weight 9.2 kgr. Fed twenty-six hours before operation. Same operation 36 cm. below pylorus. Post-operative course as above. Lived eighty-two hours. Autopsy. Same findings as in other cases.

No. 13. Dog. Weight 8.2 kgr. Fed twenty-six hours before operation. Bowel sectioned 36 cm. below pylorus and ends inverted. Symptoms same as others. Temperature 35.5° C. Pulse 180 after twenty-four hours. Lived thirty-eight hours. Autopsy. No peritonitis. Ends of bowel tight. At distal closure an intussusception 14 cm. long had taken place and the intestinal wall was much damaged. This, we believe, contributed to early death.

No. 14. Young dog. Weight 5.75 kgr. Fed seven hours before operation. Bowel sectioned and ends inverted 31 cm. below pylorus. Never fully recovered from operation and died in twelve hours. Autopsy. No peritonitis. Closed ends tight.

No. 15. Dog. Weight 10.5 kgr. Fed forty-eight hours before operation. Bowel sectioned and ends closed 48 cm. below pylorus. Post-operative symptoms same as in other dogs. Lived thirty-two hours. Autopsy. No peritonitis. Closed ends tight; 10 cm. intussusception of distal end in gut, with much damage of gut wall. Proximal intestinal mucosa showed much congestion and many punctate hemorrhages.

These animals were operated upon at varying periods after eating, and none was fed anything after operation. The one which lived the longest, one hundred and sixty hours, was fed twenty hours before operation, and the one that died in ten hours was fed the last time twenty-seven hours before operation. One dog which ate a full meal only six hours before operation lived one hundred and sixteen hours, while one that had fasted for seventy-two hours lived fifty-five hours after operation. The dogs had

all the water, both before and after the operation, that they would take.

This same lack of correspondence between the state of digestion at the time the obstruction was produced on the one hand, and the severity of the symptoms and length of life afterward, was noted in all subsequent experiments. It seems, therefore, reasonably certain that decomposition of food residues in the tract has little to do with causing death, although the ingestion of food after the obstruction might readily be a factor.

The condition of the dogs after operation can be very briefly summarized. They showed a remarkable absence of active symptoms. Recovery from operation was usually prompt and complete, and on the next day they seemed well, but less lively than before. There was a slight rise in temperature. Water was drunk freely, and promptly vomited, washing out bile and mucus. Emaciation was marked. After a varying time the animal began to grow weak, but there was a lack of many of the symptoms we are accustomed to see in the human subject. Vomiting, as a rule, took place only after drinking. There was no abdominal distention, and no sign that cramps or colic were present. Once or twice we noted some rigidity of the muscles of the hind legs, as described by Maury, but in most cases no such rigidity could be demonstrated.

An autopsy was done in every case, and in none was there any gross alteration in the organs that accounted for death. The peritoneum was not inflamed. The stomach and the intestine above the section were never markedly distended. They usually contained only a very small amount of bile and mucus. The mucosa showed a severe congestion. In one or two cases there was a superficial ulceration in the intestinal mucosa. We were struck with the fact that the most severe changes of this character were present in the dogs living the shortest time. The gall-bladder was not distended. The kidneys and liver showed congestion, and the former evidence of degeneration.

From these cases it was determined that animals with a high obstruction might live as long as five to seven days; that the length of life was not dependent on the presence of food residue in the tract above the obstruction, and that autopsy gave no gross evidence of the cause of death. A complete microscopic study of these cases was not made. Where sections were examined, however, the same lesions were found in the intestinal mucosa, the kidney and the liver, as those to be described in the second group of cases.

We next turned our attention to the production of an obstruction which should not be complicated by any operative damage to the intestinal wall. This was considered advisable because there seemed to be a direct relation between the severity of this damage on the one hand, and the severity of the symptoms and

the early death of the dogs on the other (Nos. 11, 13, 15). In one case the bowel was closed by tying a tape around it and burying this with a row of Lembert sutures. The tape cut through and a perforative peritonitis followed. The same thing resulted in two cases where the Crile carotid clamp was used.

A special clamp was then devised which proved to be efficient in causing a complete obstruction, without opening the gut, or damaging the blood supply. The clamp consisted of two rubber-covered aluminum plates, about 1 cm. in width and 4 cm. long. Over each end a second covering of rubber tubing was placed. One plate was then passed through a slit in the mesentery, with the bowel passing over it. The second plate was placed across the bowel at this point, and the two plates bound together near their ends. The interval left between them because of the rubber coverings over the ends allowed sufficient room for the intestine, tightly compressed, but not strangulated, to pass through. By bending the plates in or out the degree of obstruction was accurately regulated. We found by experience that the proper degree was obtained when the thin handle of a scalpel could just be passed within the bite of the plates, but not through it. (See Fig. 1, *b*.)

No. 19. The first dog on which the clamp was used was a male, weighing 13.5 kgm. He had been in the laboratory seventy-two hours without feeding, and had been given only sterile water to drink. This was done with the idea of getting the stomach and upper intestine as nearly free from bacteria as possible. Cushing and Livingood<sup>20</sup> have shown that the number present can be reduced very markedly in this way. The clamp was placed 18 cm. below the pylorus. The dog remained in excellent condition for five days following the operation. The temperature ranged from 37.5° to 38.5° C. and the pulse from 132 to 150. He drank freely of sterile water, which he vomited soon afterward, washing out bile and mucus with it. He passed small quantities of urine, which contained traces of bile and albumin. During this period he lost 1.8 kgr. He showed no active symptoms of the disease, and was in no apparent pain. The abdomen was not tender to palpation, nor was it distended. At operation the circumference of the abdomen was 47 cm. and on the fifth day only 43 cm. After the fifth day he began to show marked weakness and a more rapid loss of weight. The pulse and temperature remained about the same, and no new active symptoms developed. On the seventh day he was markedly weak and walked with difficulty. Emaciation was marked, there having been a loss equal to 19 per cent. of his body weight. He was killed with chloroform one hundred and seventy-two hours after the operation and the autopsy, with bacterial examination, immediately done.

The obstruction was tested under water pressure, and was found complete, no water passing through the clamped point. Coils of intestine and omentum were matted around the clamp. On separating these, a small local abscess of green odorless pus was opened. The peritoneal coat of the intestine, at this point, was eroded by pressure, but no serious damage was done to the rest of the wall. The general peritoneal cavity was entirely free from evidence of inflammation. The liver and kidneys showed a marked congestion. There was a marked dilatation of the stomach and upper intestinal segment, and both contained water with bile and mucus. Their mucosae were markedly congested.

*Microscopic Examination.* The intestine proximal to the clamp shows intense congestion and considerable exudative inflammation of the subserous tissue. The submucosa shows hemorrhagic and leukocytic infiltration. There is an acute hyperplasia of the lymph follicles. Distal to the clamp the intestine is normal. The kidneys show intense capillary congestion, especially of the glomeruli. The epithelium is not well preserved.

The spleen shows congestion.

The pancreas is normal.

The liver sections were unsatisfactory.

The cultures from the abscess around the clamp showed a pure culture of a Gram-negative bacillus, having the characteristics of colon bacillus. Cultures taken from the inside of the intestine, just above the clamp, showed the same bacterial growth, and in addition, a gram-positive coccus. Our effort to sterilize the upper bowel was not successful, and the local abscess probably resulted from the passage of organisms outward through the intestinal wall, where the peritoneal coat was damaged by the clamp pressure. Other organs were sterile (see table).

No. 20. Dog. Weight 9.5 kgr. No food for one hundred hours. Sterile water during this time. Repetition of No. 19. Post-operative symptoms the same. Lived ninety hours. Autopsy (five hours postmortem during which time the body was on ice) showed no peritonitis. Obstruction complete. Stomach a little dilated. Bowel above clamp twice the normal size. In the intestine, distal to the clamp were seen many hemorrhagic points.

*Microscopic Examination.* Intestine proximal to clamp shows fibrinous peritonitis, marked congestion of all coats, and exfoliation of lining mucosal cells. Stomach normal.

Liver. Congestion. Moderate granular and fatty degeneration.

Kidney. Moderate congestion: Tubules show granular coagulum. Cells swollen and show marked granular degeneration. Some cells show partial necrosis.

Cultures from femoral blood, peritoneum and liver show neither aerobic nor anaerobic growth.



No. 21. Puppy. Weight 8 kgr. Fed four hours before operation. Clamp applied 16 cm. below pylorus. The stomach contained food and the lymphatics and vessels were distended from full digestion. The post-operative course was the same as in the others. Pulse, 190. Temperature, 39° C. He died fifty-eight hours after operation. Autopsy, eight hours post mortem, the body being on ice. No peritonitis. Moderate distention stomach and duodenum. Few drops of purulent looking fluid around clamp, buried in adhesions. The obstruction was complete. The gastric and intestinal mucosa, proximal to the clamp, showed very little change. Distal to the clamp there were areas which resembled submucous hemorrhages in the intestinal wall. The other abdominal organs appeared normal.

*Microscopic Examination.* Proximal intestine not examined. Distal intestinal mucosa shows hyperplasia of lymph follicles, but no hemorrhages.

Liver. Marked congestion.

Kidney. Congested. Tubule cells swollen, and in places intensely degenerated. Apparent necrosis present in some foci of the tubules.

Spleen and pancreas much congested.

*Bacteriology.* Cultures from femoral blood, peritoneum and liver all sterile, both aerobic and anaerobic, with exception of one colony on one peritoneal aerobic slant agar tube. This showed pure growth of a Gram positive coccus and probably was an air contamination, one of the three found in the whole series.

No. 22. Bitch. Weight 10.25 kgr. Fed eighteen hours before operation. Clamp applied 12 cm. below pylorus. This animal was killed on the twenty-first day, up to which time she had showed no symptoms of suffering or of disease, except for loss of weight to 7.4 kgr. During this time she had eaten nothing. She drank water freely, however, for the first three days. After this she received 150 to 250 c.c. of normal saline solution subcutaneously, and then she refused water to drink. She only occasionally vomited. The temperature ranged between 37° and 38° C. The pulse for the first four days was around 130. It then became slower and during the last two weeks was between 70 and 100. Urine was passed freely and contained bile and albumin in small quantities. Several blood-cell counts were made during the later days of the experiment and the red cells numbered between 5,240,000 and 7,000,000, nearly all counts being slightly over 5,000,000. Killed with ether on the twenty-first day while still in good health. Autopsy. No peritonitis. Clamp allowed minimal drainage into gut below (compare No. 27). Stomach and duodenum were somewhat dilated and the intestine distal to clamp was contracted, showing very little flow into it. However, it contained bile, and under pressure water was forced through clamp into

bowel below. The mucosa of intestine and stomach appeared normal. The kidneys and liver showed congestion.

*Microscopic Examination.* Stomach and intestine not examined.

Kidneys. Tubules filled with granular coagulum. Cells show swelling and granular degeneration.

Liver. Intensely congested.

Spleen. Congested.

Pancreas. Normal.

*Bacteriology.* Neither aerobic nor anaerobic growth on tubes from femoral blood, liver or spleen.

No. 23. Dog. Weight 11 kgr. Starved five days, receiving only sterile water. Clamp applied 20 cm. below pylorus. He was killed on the tenth day. His symptoms were the same as in cases above. He received saline subcutaneously with apparent benefit. Pulse, 132 to 150. Temperature, 37.9° to 39.2° C. Weight at death 8.5 kgr. Urine contained albumin and bile. An extensive noma developed on face which accounted for high temperature. Autopsy immediately after death by cyanide. Obstruction complete. Stomach and proximal gut much dilated. Distal gut collapsed (Fig. 3). They contained about 300 c.c. of bile, mucus, and water. The specimen was preserved intact so the mucosa was not seen. No peritonitis. Liver and kidneys much congested. The latter showed acute degeneration, the markings being indistinct and the papillae pale.

*Microscopic Examination.* Kidneys show granular degeneration, in places very marked.

Liver. Intensely congested. Considerable fatty and granular degeneration.

Spleen and pancreas show congestion.

*Bacteriology.* Aerobic and anaerobic cultures from femoral blood and liver were all without growth.

No. 24. Small bitch. Weight 6 kgr. Fed seventy-two hours before operation. Clamp applied 16 cm. below pylorus. Lived two hundred and five hours—a little less than nine days—with symptoms as in other cases. Pulse, 120 to 180. Temperature average, 39° C. Received saline injections throughout. Red-blood cells increased from 5,000,000 before operation to 6,720,000 on eighth day showing some loss of water. Weight 4.9 kgr. Died during night. Autopsy at 10 A.M. in morning. Obstruction complete as tested by absence of bile in distal gut, although under pressure, water could be forced beyond the clamp. No peritonitis. Around the clamp in a mass of omentum there was a collection of dirty yellowish fluid. The intestinal wall showed some damage within the bite of the clamp (see microscopic examination). The stomach and proximal intestine were moderately dilated, and contained a small amount of bile and mucus. The mucous membrane of the whole tract appeared normal to gross examination, as did the kidneys and liver.

*Microscopic Examination.* The intestine within the clamp showed an advanced purulent peritonitis, destroying longitudinal muscular coat. Congestion of mucosa. Exfoliation of epithelium.

Kidney. Congested. Tubules filled with granular coagulum. Cells swollen and show marked granular degeneration in places approaching necrosis.

Liver. Section lost.

Pancreas. Marked congestion.

*Bacteriology.* Aërobic and anaërobic cultures from femoral blood, peritoneum, and liver showed no growth. A Gram positive coccus was obtained in pure culture from the abscess around the clamp in all tubes both aërobic and anaërobic. This was undoubtedly an infection passing through the gut wall where the clamp damaged it, since the same organism was isolated from the intestine.

No. 25. Dog. Weight 6 kgr. Fed twenty-seven hours before operation. Clamp applied 16 cm. below the pylorus. He took ether badly and made a poor recovery. He was not given the hypodermoclysis of normal saline solution. Condition seemed good the next day. Died that night. Autopsy not done until day following, about thirty hours post mortem. Obstruction complete under water pressure. No peritonitis. Stomach and proximal gut somewhat distended. Distal gut collapsed. Mucosa of stomach and proximal gut markedly congested. Liver, spleen, pancreas, and kidneys showed congestion.

*Microscopic Examination.* Stomach congested. Proximal intestinal mucosa shows intense congestion. Marked round-cell infiltration of tips of villi and exfoliation of cells.

Liver. Intense congestion. Considerable granular and fatty degeneration.

Kidney. Not examined.

No bacteriological examinations made because of delayed autopsy.

No. 26. Bitch. Weight 7.2 kgr. Exact duplicate in every way of No. 25, except that she received the saline injections daily, the first one of 150 c.c. being given at the close of the operation. She lived ten days, and showed the same symptoms as in other dogs described. There was a marked contrast to dog No. 25, which did not receive the saline. Killed with ether and autopsied immediately. Obstruction complete against a column of water 90 cm. high. No peritonitis. The stomach and proximal gut were dilated to about three times normal. The mucosa of stomach and proximal intestine were congested. The liver, kidneys, and spleen were congested.

*Microscopic Examination.* Proximal intestinal mucosa shows intense congestion and exfoliation of living cells.

Kidney. Shows congestion. Tubules contain coagulum and

occasional blood cells and show granular degeneration, in places very marked.

Liver shows intense congestion, atrophy of liver cords about distended capillaries and moderate granular degeneration.

Spleen shows much hypertrophy of the follicles.

Pancreas normal.

Cultures from femoral blood, liver, spleen, and peritoneum were taken. These were all without growth after four days, under both aerobic and anaerobic conditions, except one aerobic tube (four were taken) from the liver. This showed one colony of Gram positive cocci growing well to one side of the stroke of the loop.

No. 27. Bitch. Weight 11.5 kgr. Fed twenty-eight hours before operation. Clamp applied about 15 cm. below the pylorus. Lived twenty-four days with only moderate development of symptoms during first week. Then behaved like a normal dog. Re-operated upon on July 20. The clamp was not sufficiently tight to make a complete obstruction (compare with No. 22). It was tightened and the next day it had cut its way through the gut wall and the dog died of a peritonitis.

No. 28. Dog. Weight 6.5 kgr. Fed twenty-eight hours before operation. Clamp applied 11 cm. below pylorus. Post-operative course as in other cases. Died in fifty-six hours and body immediately placed on ice. Autopsy ten hours later. Obstruction complete. No peritonitis. Local infection around the clamp. Stomach and proximal intestine only slightly distended and contained a small amount of bile-stained fluid. The distal gut was totally empty. Two superficial ulcers were seen in the mucosa of proximal gut. The liver, kidneys, spleen, and pancreas appeared normal.

*Microscopic Examination.* Intestine not examined.

Kidney. Tubules swollen and show granular degeneration, very marked in places.

Liver. Intensely congested.

*Bacteriology.* Cultures from the femoral blood, the peritoneum, the liver, the spleen, and the local collection of fluid around the clamp all were without growth under aerobic and anaerobic conditions.

No. 29. Dog. Weight 8 kgr. Fed thirty hours before operation. Clamp applied 11.5 cm. below pylorus. Post-operative course same as in other cases. Received about one-fortieth of body weight of normal saline subcutaneously daily. He remained in excellent condition for eight days, during which time he lost a little over 2 kgr. in weight. He drank water each day which was always vomited. Pulse from 130 to 174. Temperature, 38.5° to 40° C. On the ninth day he was etherized and the abdomen opened. The obstruction was complete. The stomach and proximal intestine were much distended (Fig. 2), and filled with a greenish fluid of the thickness of pea soup. There was no peritonitis. The

splanchnic vessels were not much dilated, except those above the occlusion. The spleen was not enlarged. The liver and kidneys did not show much congestion. Sections were taken from the stomach, duodenum, distal intestine, liver, spleen, and kidney while the dog was still under ether, after which he was killed.

*Microscopic Examination.* The stomach is normal.

The proximal intestine shows marked congestion and an edema in the submucosa. The lesions here were not advanced, because the specimens were taken some hours before the animal would have died from the disease. The distal intestine is normal.



FIG. 2.—No. 29. Showing mucous surface of intestine at site of clamp. Note absence of damage to mucosa at this point and the congested appearance proximal to it. Also distention of duodenum above the clamp.

The liver is congested. The cells show granular degeneration and areas of necrosis are present.

The kidney is congested. Tubules contain granular coagula. The cells show granular degeneration.

The above series includes experiments on 11 animals which may be summarized. In 2 a complete obstruction was not obtained because the clamp was not sufficiently tight. These 2 animals were killed after twenty-one and twenty-four days respectively, while each was in good condition. They had emaciated a good deal, and one of them was weak, but otherwise they acted like normal dogs.

The remaining 9 dogs all showed a similar condition while living, and autopsy yielded the same findings in the organs. In some

dogs, particularly those which lived the longest, the dilatation of the stomach and the duodenum was very marked (Fig. 3, of No. 23).

Four dogs in this series were given sterile water by mouth only, and this they promptly vomited. They were, therefore, practically getting neither nourishment nor water, for if what they drank was not all vomited, very little absorption could take place from the stomach and duodenum. Beginning with the fourth dog, each dog

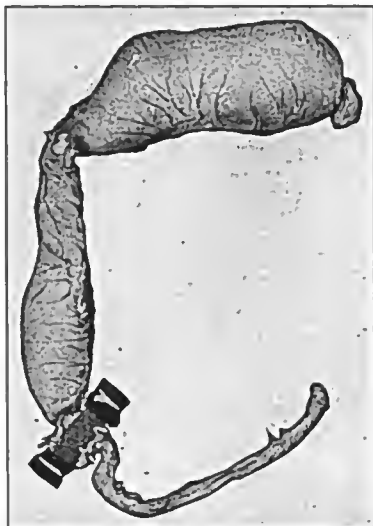


FIG. 3.—No. 23. Showing clamp in situ. Stomach and duodenum dilated. Vessels engorged.

received from 150 to 300 c.c. sterile normal saline solution, either subcutaneously, or by rectum, per day, and this was absorbed. In one case this was not given till the third day. This animal had then begun to act sick, but showed a marked improvement after the administration of the hypodermoclysis. In general, the dogs who received this saline, lived longer than the others. They sometimes drank sterile water in addition, but this they always vomited in a very short time. In many cases they refused to drink more than a few cubic centimeters. The 3 dogs which lived the longest

were all given the saline subcutaneously, and there seems no doubt that this procedure was very beneficial.

We attempted, by a count of red-blood cells, to determine whether the dogs not receiving it suffered from dehydration, with a consequent increase in the number of red cells per cm. There was not a sufficient variation from the normal, to throw any light on this point, the number ranging around 6,000,000 both before and after the saline injections. The saline may have acted in either of two ways. It may have prevented an actual dehydration of the tissues, which is a deleterious condition in itself, or it may have acted as a diuretic and thus aided the elimination of poisons by the kidneys. The longest post-operative life was ten days (2 dogs); a third dog lived just under nine days; the shortest period was thirty-six hours in a small fox terrier who never completely recovered from the operation. The average length of life was one hundred and thirty-nine hours in this series, as against sixty-seven hours in the series where the obstruction was produced by sectioning the intestine and inverting the ends, but it was only in the former that the aid of the saline injections was used.

It was, moreover, remarked that the second series in which no damage to the intestinal wall was produced was much nearer the normal throughout the experiment than was the first series in which the intestine was sectioned. So far as we know, no other experimenter has paid the same attention to producing an obstruction without any damage to the intestinal wall. There is evidence that the method is of advantage in excluding the complications which may arise from such damage. Clinically, we know that when a strangulation—however limited in extent—is superimposed on an obstruction, the course of the disease becomes much more acute. Medowoy,<sup>21</sup> Kocher,<sup>22</sup> and others, emphasize this point.

Murphy and Vincent<sup>23</sup> in their experiments, worked particularly on this phase of the question. In a simple obstruction in cats they found the intestinal wall very little damaged in four to six hours. When, however, the circulation was disturbed, particularly a venous stasis produced, a very severe change was present at this time. They conclude that "Interference with the circulation of the obstructed intestine is the vital factor in the production of the symptoms of ileus."

When the intestine is sectioned, and the proximal end sufficiently inverted to prevent the stomach and duodenal contents being forced through it, a local strangulation takes place. At autopsy there was always found in these cases a local damage to the intestinal wall at the inverted point, small in extent to be sure, but nevertheless sufficient to be the possible site of the absorption of

<sup>21</sup> Deutsch. Zeitsch. f. Chir., cv, S. 1.

<sup>22</sup> Mittel. Grenz. d. Med. u. Chir., Band iv, Heft 2.

<sup>23</sup> Loc. cit.

a considerable amount of poison. Often, too, a very considerable intussusception of the invaginated stump was present. This mechanical damage, of course, is not an essential in producing death, because it was not present in the second series, and they died. We feel, however, that it has an important bearing on the problem, as we shall later, more fully explain.

We desire to call particular attention to the microscopic findings in these cases. The intestinal mucosa, proximal to the obstruction, shows a marked congestion. Marked round-celled infiltration is present, and exfoliation of the lining cells. Hemorrhages are often seen. There is present an acute hyperplasia of the lymph follicles. The submucosa is edematous. Distal to the clamp there is no change, or only a very slight change from the normal. The gastric mucosa shows only a congestion. The kidneys are markedly congested. The tubules contain granular coagula. The tubule cells are swollen and the seat of granular degeneration; not infrequently necrotic areas are seen.

The liver is congested and the cells show granular and fatty degeneration. Necrotic areas may be present.

The spleen shows marked congestion, and some hyperplasia of the lymph follicles.

The pancreas shows only congestion.

The above changes are those seen in many forms of toxemia, both bacterial and non-bacterial. They are found in the toxemia of starvation, of pregnancy, of delayed chloroform poisoning (Howland and Richards<sup>24</sup>), and of many of the acute infectious diseases. They are, therefore, very strong evidence that in our dogs, some toxic substance was present in the circulating blood during life. The rapid emaciation, the progressive weakness and the high pulse rate, with a comparatively normal temperature, all support this evidence. The two cases, Nos. 22 and 27, in which the obstruction was not complete, indicate that the condition is not one of simple starvation, because these animals received no food and practically no water to drink (the saline was given subcutaneously as in the others) during a period of over three weeks, and yet remained in a condition of good health. They showed less emaciation and loss of strength during the experiment than did the fully obstructed dogs in one-third as long a period.

In order to test this point still farther, however, a dog was kept in the laboratory for two weeks, and received absolutely no food or water by mouth. The saline injections were given daily. Under this treatment he remained in an apparent condition of perfect health. He lost 29 per cent. of his body weight. There was no evidence of weakness. The pulse rate was from 70 to 120, and the temperature normal. A second dog had the clamp applied to the

<sup>24</sup> Jour. Exp. Med., 1909, ii, 344.



pylorus. He remained well (Fig. 4) for fifteen days, receiving only the subcutaneous saline injections. The toxemia of starvation is, therefore, not the determining factor in producing the symptoms or the pathological changes found in intestinal obstruction. We attempted, as mentioned above, to eliminate a bacterial origin, either direct or indirect, for the toxic substances, by withholding food from the dogs and allowing them only sterile water to drink, both before and after the operation, with the hope that we might produce a sterile condition of the stomach and duodenum above the occlusion. In this we were not successful, for in the 6 cases where a bacterial examination of the intestine above the clamp was made at autopsy an abundant growth of the usual intestinal flora was found. A



FIG. 4.—Dog with clamp on pylorus. Showing good condition after twelve days. Examination under ether on the fifteenth day, while the dog was still in good condition, showed the clamp to be tight.

further attempt to accomplish such sterilization was done in two cases (Nos. 30 and 31) by producing an intestinal fistula just distal to the duodenum (Fig. 1, c). We hoped by gastric and retrograde washing through this tract to get it clean and then obstruct it. Both dogs, however, died within a few days, and no further attempt in this direction was made.

We then turned our attention to an elimination of the bile, pancreatic, and duodenal secretions as a source of the poisons. To this end, in five dogs (Nos. 32 to 36) the following operation was done (Fig. 1, d). The pylorus was divided and the two cut ends closed by inversion. The intestine was divided just distal to the

duodenum, and the proximal end implanted in the jejunum about 30 cm. lower down. The distal end was implanted into the stomach. By this means the stomach emptied into the intestine just below the duodenum, and the bile, pancreatic, and duodenal secretions passed into the jejunum about 30 cm. distal to this. Our intention was to cause an obstruction with the clamp, just orally to the new duodenal outlet when the animals should have recovered from the anastomoses, thus having an obstruction of the stomach and about 30 cm. of intestine, but not of the duct-bearing portion of the tract.

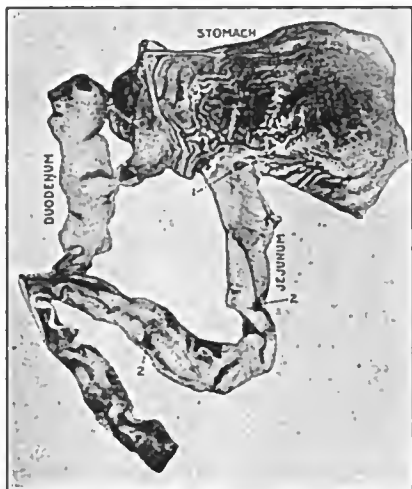


FIG. 5.—Showing damage to jejunum where the gastric juice passes into it without neutralization by entering duodenum; 1, perforation just below gastrojejunoanastomosis; 2, ulcerations lower down.

For a few days following the operation these dogs did well, fully regained their appetites and seemed to be in good health. After about a week, however, they all began to act sick. They did not eat well, and sometimes vomited. They lost weight rapidly. Some of them developed diarrhea with bloody appearing stools. Four of them died in about four weeks with evidence of malnutrition. The autopsy showed, in two of these, that a very extensive damage to the intestine between the stomach and duodenal anastomosis was present. Ulcers were seen which were about to perforate (Fig. 5).

The stomach and this part of the gut contained partially digested blood.

The fifth dog seemed to be in fair condition after two weeks and on the nineteenth day the abdomen was opened under ether anesthesia. The anastomoses were satisfactory and no evidence of ulceration was present from the peritoneal surface. The occlusion clamp was applied just above the anastomosis of the duodenum with the jejunum. The following day the dog was in good condition; he had not vomited. He was given saline subcutaneously.



FIG. 6.—Showing perforation: 1, at site of clamp where it was applied nineteen days after anastomosis; 2, gastrojejunosomy stoma; 3, artefacts; 4, duodenojejunal anastomosis.

He died during that night and autopsy was done in the morning. The anastomoses were satisfactory. A general peritonitis was present from a perforation within the bite of the clamp (Fig. 6). The stomach and intestine anastomosed to it were distended with a dirty brownish fluid. On opening them the gastric mucosa was seen to be much congested; the mucosa of the jejunal segment was markedly congested and contained many hemorrhagic areas. The occlusion clamp had caused a perforation through one of these areas. The intestine below the duodenal anastomosis was entirely

normal in appearance. Similar ulcerations have been observed in the jejunum of dogs, after a long loop anastomosis to the stomach, by Exalto,<sup>23</sup> and many clinical cases of jejunal ulceration are reported following gastro-enterostomies.

Our attention was attracted by these cases to the damage done to the intestinal wall when the gastric juice unneutralized by the duodenal secretions comes into contact with the intestinal mucosa. Our effort to produce an obstruction in which the duodenal contents should not be stagnated had not been successful. But the symp-



FIG. 7.—Showing perforation (1) and ulceration (2) in doubly occluded stomach and duodenum.

oms developing in these dogs and the findings in the intestinal mucosa turned our attention in another direction which we think led to important results. In many ways these dogs behaved like the ones with an obstruction, the symptoms and the course of the disease being, however, much less acute. The microscopic examination of the intestinal mucosa, the kidneys and the liver showed, too, the same lesions as found in the simple obstruction. It, therefore, occurred to us that the damage we had noted in the mucosa, above the occlusion, might be a factor in the production of the

toxemia which caused the animal's death. To further test this point, the following experiment was done three times (Nos. 37 to 39).

The stomach was bisected and the two cut ends closed. A gastro-enterostomy was made between the cardiac half of the stomach and the first part of the jejunum. The occlusion clamp was then applied just proximal to this anastomosis, or the proximal end of the divided jejunum was implanted into the pyloric half of the stomach. In this way the secretions from the pyloric half of the stomach, the liver, the pancreas, and the duodenum were occluded within half of the stomach and the duodenum (Fig. 1, *e*). Two of these dogs died in approximately thirty-six hours; one in eighteen hours. They were very sick during this time. The pulse was 200 and over. They vomited. At autopsy the occluded portion was found enormously distended with a mixture of bile and mucus which was blood tinged. The stomach and intestinal wall were markedly stretched, the latter to paper thinness. In one case, at a point near the lower end of the duodenum, an ulcer was present which had perforated through the peritoneal coat, and around it was an area of necrosis 5 cm. in diameter. Proximal to this point were five extensive ulcers (Fig. 7). The mucosa throughout was markedly congested. In the other 2 cases no ulceration was seen, but the damage to the mucosa was very severe. In the jejunum, immediately below the gastric stoma, the mucosa was damaged in a similar way to that seen in the previous series, due to the unneutralized gastric juice entering it. Microscopical examination of the tissues gave the same findings as in the obstructed cases.

In the next series of experiments, Nos. 40 to 43, a loop of intestine below the duodenum was occluded by sectioning and closing the ends, or by the use of the clamps, and the patency of the tract reestablished by an anastomosis between the proximal and distal arms, above and below the clamps (Fig. 1, *f*). One was done high up in the jejunum, two in the middle of the small intestine, and one just above the cecum. The first of these dogs lived about eighty-four hours, and during the first three days of this time he seemed perfectly well. He drank water and took food without vomiting. He suddenly began to act sick on the fourth day and died that night. The closed ends of the loop were not tight, and leakage had taken place, resulting in peritonitis. The occluded loop showed an intense congestion and it was distended with dark bloody fluid. The mucosa showed hemorrhagic areas. The peritonitis, however, renders the case useless except for a study of the damage done to the occluded loop.

In the second case, leakage in a similar manner also took place, vitiating the result so far as the course of the disease is concerned. The occluded loop, however, was much distended with a thin

bloody fluid. The mucosa was markedly congested and at one point a superficial erosion was present.

The third dog, in which the occlusions were done with the clamps, lived about one hundred and ten hours. He remained well for three days, and then began to show marked symptoms similar to those seen late in the obstructed cases. Autopsy. The anastomosis was tight and the clamps had produced a complete closure. There was no abnormality seen in the alimentary tract, except in the occluded loop, which was 44 cm. in length and began 76 cm. below



FIG. 8.—Showing distended nearly gangrenous loop of intestine after double occlusion in upper jejunum. Rubber tubes indicating points at which clamps were placed; (1) entero-enterostomy. Mesenteric vessels perfectly patent.

the pylorus. This loop was enormously distended and contained about 500 c.c. of a bloody exudate containing granular material resembling hashed up blood clot. The loop was nearly gangrenous<sup>24</sup> throughout the whole extent of its convexity, the process being much more marked in the outer coats than in the mucosa (Fig. 8). No localized ulcerations were seen. The mesenteric vessels were filled with fluid blood, showing that gangrene did not result

<sup>24</sup> Cf. Roger and Garnier, *loc. cit.*

from their thrombosis. The gastric mucosa was not congested, but that of the duodenum was slightly so. The kidneys and liver showed a very marked congestion.

The fourth dog in this series had the clamps applied in the lower ileum, closing off a portion of the bowel about 25 cm. in length, proximal to the cecum, with an anastomosis between the proximal and distal arms. He was an old animal, nearly blind, and a poor operative subject. For three days he remained in fair condition. He drank water and did not vomit. The pulse was about 150 to 170 and the temperature 40° C. On the fourth day it was noted that the wound was badly infected, and he began to suffer from the dyspnea which is so often seen in old dogs. Ether was given and the abdomen reopened. The wound was badly infected, and a large intramural abscess was present. There was no peritonitis. The anastomosis had not leaked. The clamps were tight. The occluded loop was not distended. It contained only a few cubic centimeters of dirty brown mucus. The mucosa showed no evidence whatever of congestion. The condition was in marked contrast to that seen in the over-distended, ulcerated and gangrenous loops of the upper tract. The sickness of this dog we attribute to his age and general condition before operation, and to the severe infection in the wound, more than to the occlusion itself. McClure<sup>27</sup> and v. Baracz<sup>28</sup> have found similar results to this. They are to be explained by the absence of any irritative agents in the occluded lower ileum loop which cause damage to the mucosa. Hence, no poisons, bacterial or otherwise, are able to pass the defense of the lining cells and the animal remains unpoisoned (cf. occlusion of pylorus, page 388). The experiments of the last two series demonstrate that when, in a dog, any portion of the upper small intestine, either alone or with a part of the stomach, is occluded at both ends, and the patency of the alimentary tract reestablished by an anastomosis, the animal lives even a shorter time than in the simple high obstruction. He rapidly develops symptoms which come on only late in the obstructed dogs. The occluded loop is always markedly distended, and its wall is severely damaged by ulcerations or by destruction of the mucosa, and the damage may go on to gangrene. There is hemorrhage in the loop. If the loop receive the active digestive juices the damage is most marked in the mucosa; if it be lower down, the process is most marked in the outer coats. The vessels in the mesentery are not thrombosed, though an examination of the gut wall suggests this as a cause of the lesion. In the lower ileum the damage after four days may be very slight. If these lesions be compared to those already described as appearing in a simple high obstruction, they are seen to be of the same character, though much more severe. They resemble also the lesions

<sup>27</sup> Loc. cit.

<sup>28</sup> Loc. cit.

produced when the gastric juice unnutralized by the duodenal contents is emptied into the jejunum. From these findings it would seem that in all our experiments a destructive agent was at work which seriously affected the mucosa of the intestinal wall.

As is well known, one of the functions of the intestinal mucous membrane is to so alter the substances which pass through it into the blood, that they are not toxic to the organism. The changes which we have found in the structure of the mucosa, we believe, deprive this membrane to a greater or less extent of this function, and in consequence the toxic substances, which other workers have demonstrated to be present, actually enter the blood in their poisonous condition. If the damage to the mucosa is great as in double occlusion, the poisons enter the system rapidly, and the course of the disease is short. If the damage is less severe, as in simple obstruction, where oral drainage by vomiting can take place, the course of the disease is lengthened. Therefore, change in the intestinal mucosa, proximal to the obstruction, is to our minds, the key to the solution of the cause of death in intestinal obstruction. This change results from the irritating influence of the retained digestive juices, and the mechanical damage due to the stretching of the intestinal wall. It deprives the mucosa of its defence against the passage of substances which are poisonous to the organism. These poisons, therefore, slowly enter the blood, and work the damage seen in the kidneys and liver, and ultimately produce the death of the animal. The poisons are present in the normal stomach and intestine,<sup>22</sup> and it is not necessary to consider that the obstruction causes any alteration in them. Abnormal absorption is the essential factor.

This explains the total lack of symptoms observed in our dogs over a period of nearly a week in some cases. The changes in the mucosa of the intestine were not sufficiently great to deprive it of its defence against the passage of unaltered poisons (No. 23). When the change once developed, this abnormal absorption took place rapidly, and active symptoms ending in death soon resulted. The subcutaneous injection of saline was beneficial because it probably aided in the elimination of the poisons by the kidneys, as well as prevented a dehydration of the tissues from lack of water. Any method of producing the obstruction, which causes a mechanical damage to the intestinal wall, hastens the development of symptoms and death, because the defence is then locally broken down. We see in this an explanation of the much more severe type of the disease when a strangulation is superimposed on an obstruction.

In one dog the clamp was applied directly to the pylorus. This animal lived in a condition of good health for fifteen days (Fig. 4).

<sup>22</sup> Roger and Garnier, *loc. cit.*



During the first twelve days he received the saline injection subcutaneously and refused to drink any water. His condition remained so nearly normal that it was concluded that the clamp was not causing a complete obstruction. The saline injections were discontinued, and he was given water by mouth and 50 grams of-chopped beef heart on the twelfth day and 75 grams on the thirteenth day. The next day he refused to eat anything more, but he drank water freely, some of which he vomited. On the fifteenth day he was etherized and the abdomen was opened. The clamp was found to have caused a complete obstruction. There was no peritonitis. The stomach was enormously dilated, filling nearly the whole abdomen. This dilatation had occurred only during the three days in which he received water by mouth, as determined by the measurement of his girth which increased from 30 cm., the normal size, to 50 cm. The contents of the stomach was water with remnants of finely digested meat. The mucosa was entirely normal in appearance. There was no congestion of any of the abdominal viscera. From this experiment it is evident that the gastric juice, when stagnated in the stomach itself, is not absorbed as a toxic substance, so long as the mucosa remains normal. As shown, however, in experiments 32 to 36, it produces changes in the small intestine, when unneutralized by the duodenal contents which permit the absorption of materials producing symptoms and pathological lesions in the organs analogous to those of an obstruction. These facts are very strong evidence that the damage to the mucosa occurring in ileus is a necessary factor in the production of the toxemia there seen.

The following is a summary of the findings of our work:

1. A high intestinal obstruction, that is, 10 to 30 cm. from the pylorus, in dogs, may not produce death for ten days, provided the gut wall is not damaged. If it is damaged by section and inversion the average life is only half as long.
2. There are found in the kidney and liver cellular changes which are the same as those found in many toxic diseases. The intestinal mucosa is found to be damaged to such an extent that it may readily be conceived that it has been deprived of its natural defence against the passage of toxic substances, unaltered, through it.
3. Bacterial invasion of the blood and organs does not necessarily occur.
4. Dogs deprived of food for forty-eight to seventy-two hours, may die as early as those fed ten to twenty hours before the obstruction is produced. Decomposition of foodstuffs is not, therefore, an essential element in causing death.
5. If a double occlusion of the alimentary tract, with reestablishment of the continuity of its lumen, above the lower ileum be produced, the damage to all the tissues is greater than with a simple

obstruction, and the course of the disease to a fatal termination is shorter. A double occlusion in the lower ileum produces much less damage than in the upper.

6. The action of the gastric juice, bile, pancreatic juice, and duodenal secretions are not a requisite in producing the symptoms and pathological changes seen in intestinal obstruction, because these are produced by a double occlusion in the upper ileum when none of these secretions are blocked.

7. Simple occlusion of the pylorus does not necessarily produce any evidence of a toxemia in two weeks, and the gastric mucosa at the end of this time shows no evidence of being damaged.

8. The above findings indicate that death from intestinal obstruction in dogs results from the presence of toxic substances in the circulating blood which produce fatal lesions in the kidney, liver, and other tissues. The essential factor which admits these substances into the blood is an injury to the lining cells of the intestine caused by the irritating action of the stagnated contents, together possibly, with the mechanical damage due to stretching. The poisons themselves may arise from the secretory activity of the various digestive glands, or from bacterial activity. They may be the same as those found in the normal tract or they may be substances newly formed under the conditions of stagnation. What ever their source, they are innocuous so long as the mucosa remains normal.

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## LOSS OF EMOTIONAL MOVEMENT OF THE FACE WITH PRESERVATION OR SLIGHT IMPAIRMENT OF VOLUN- TARY MOVEMENT IN PARTIAL PARALYSIS OF THE FACIAL NERVE.

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THE voluntary and emotional movements of the face are independent of one another, and one form may be lost without loss of the other. Von Beehterew<sup>1</sup> says a number of observations show

<sup>1</sup> Die Funktionen der Nervencentra, vol. II, p. 1144 et seq.

positively that the emotional movements in man are accomplished by means of special tracts which have no relation to the tracts concerned with voluntary movement. Cases of paralysis are known in which voluntary movements of the face, or of the lower part of the face, were lost and emotional movements (laughing, crying) of the face were performed in a normal manner. In discussing these cases, Nothnagel remarked that when the voluntary movement of the face is lost in hemiplegia from a focal lesion, but both sides of the face take part in equal degree in emotional movement, one may assume that the optic thalamus and its connections with the cortex are uninjured. Cases in which the emotional movements of the face have been lost and the voluntary movements preserved have been reported by v. Bechterew, Nothnagel, Rosenbach, Kirilzew, Gowers, and many others.

According to v. Monakow,<sup>2</sup> it has long been known that in many cases of hemiplegia the face on the affected side cannot be moved voluntarily, but the parietal facial muscles at once contract, as well as those of the other side, when the patient laughs or cries. v. Monakow expresses his belief that a part of the sensory reflex tract for laughing or crying is situated in the optic thalamus.

The experimental work of Roussy<sup>3</sup> throws doubt upon the optic thalamus as a reflex centre for emotional movements of the face, as does the work of Ernest Sachs,<sup>4</sup> who, in the report of his experiments upon the optic thalamus states that stimulation produced facial movements of the opposite side. He adds: "It is desirable, perhaps, to repeat here that these motor phenomena constitute no evidence of special centres in the thalamus for automatic or rhythmic movements, as has been assumed by some authors." One possibly might make the objection that the results obtained in animals are not applicable to man.

Lewandowsky<sup>5</sup> believes the opinion regarding the optic thalamus as an emotional centre is not well founded. There are so many negative cases in the literature that a relation of paralysis of emotional movement to thalamic lesions cannot be maintained. Even in the cases in which thalamic lesions have been found, different parts of the thalamus have been affected. It is not improbable, he adds, that the assumption of a relation between emotional facial movements and a thalamic lesion is without foundation.

The paralysis of one form of facial movement without impairment or with only slight impairment of the other may result from a lesion of the facial nerve, or may be a pseudoparalysis from imperfect innervation. I have observed persons who in smiling or speaking innervated one side of the face so much less than the other that paresis of one side seemed to be present, and yet the drawing up

<sup>2</sup> *Gehirnpathologie, Nothnagel's System.*  
<sup>4</sup> *Brain, Part 126, vol. xxxii, p. 1909.*

<sup>3</sup> *La couche optique, p. 185.*  
<sup>5</sup> *Allgemeine Neurologie, Part 2, p. 741 et seq.*