TREATMENT OF ADVANCED CASES OF INTESTINAL OBSTRUCTION OR STRANGULATION*

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A clinician on being taken to see a case of acute intestinal obstruction inquires if regurgitant vomiting has commenced. For he knows that before regurgitant vomiting sets in the patient has a good chance of surviving, but that once it is established the prognosis is grave. By the time the vomit has become faecal the prognosis is bad.

Patients suffering from advanced intestinal obstruction are liable to die from known and appreciated risks, such as the inhalation of vomit during anaesthesia, or the perforation of a damaged bowel into the peritoneal cavity. But they are also liable to die suddenly and unexpectedly. To account for these unexplained deaths two theories have been put forward. The first is that death is caused by toxins, either chemical or bacterial, produced in the obstructed bowel. The second is that death is due to the loss of water and electrolytes from vomiting.^{1 2} It is chiefly the effects of the loss of water in an advanced case that I wish to discuss.

DEHYDRATION IN INTESTINAL OBSTRUCTION

The dehydration of intestinal obstruction is comparable to that seen in cholera, where water loss is universally acknowledged to be a contributory cause of death. Lawrence³ has recently drawn attention to water loss as being the cause of non-recovery in diabetic coma.

In cholera the fluid is lost in the stool, in diabetes in the urine, and in intestinal obstruction in the vomit. The fluid loss from gastric secretion alone may reach six litres in twenty-four hours.⁴

In the dehydration of these three diseases the following conditions are common to them all. Lawrence drew attention to their presence in diabetic coma and in cholera, and called them the "dehydration complex."

1. Tissue depletion of fluid.
2. Low blood volume and pressure.
3. Scanty secretion of urine.
4. High blood urea.
5. Low blood chloride.

All these conditions of the dehydration complex are present in an advanced state of intestinal obstruction.

1. The tissue shortage of water can be seen by the dry tongue, the thirst, the dry skin, the shrunken features—hollowed eyes and low ocular tension.

2. The pulse is usually rapid, but always of a low tension and volume.

3. The decreased secretion of urine has been described⁵ as one of the diagnostic features of small intestine obstruction. That anuria is an occasional symptom of intestinal obstruction was brought home to me by the following case.

A man, aged 35, was admitted to Ancoats Hospital, with a history of anuria for four days, vomiting, pain in the back. According to a blood urea estimation, done under the impression that it was a case of calculus, anuria was 250 +. The patient died that night, and necropsy revealed a volvulus of the small intestine. The kidneys appeared healthy.

In the oliguria of intestinal obstruction there is usually a trace of albumin, and in some cases renal casts and blood.

4. Tileston and Comfort,⁶ in an investigation of the blood ureas in a series of diseases, found that in intestinal

obstruction the blood urea was far higher than in any other disease, except those affecting the kidneys. The following case reported by Leslie Smith and Heritage⁷ indicates the high level the blood urea may reach, and yet on recovery the kidneys may apparently be normal.

A man, aged 28, was admitted in a comatose condition to Ancoats Hospital. The history of his illness, obtained from his relatives, began five days previously with colicky pain round the umbilicus. His bowels had not opened for three days, and vomiting had occurred for the same period. On his admission the vomit was faeculent. The abdomen was distended, visible peristalsis being seen. Blood urea was 240. The urine was dark and showed a few casts. Later, cerebro-spinal fluid urea was 350. The comatose condition seemed so profound that death was considered imminent, and operative intervention useless. Improvement took place after intravenous saline had been given, and Mr. Hughes operated. The obstruction was caused by a band four feet above the ileo-caecal valve, division of which resulted in the patient's return to consciousness the following morning. Blood urea five days later was 40; fourteen days later, 21. The urine contained no albumin or casts.

The high blood urea observed when the patient has begun to vomit is not due to the failure of the kidney to excrete this, as the urine passed contains a high concentration of urea. It is due presumably to increased destruction of tissue protein. Later, when casts appear in the urine, there must be a definite nephritis (? toxic), and this is shown by the failure of the kidneys in renal dye tests.⁸

5. The decreased blood chloride of intestinal obstruction is especially stressed by Haden and Orr.⁹ They endeavour to correlate the blood chloride findings with the prognosis. The normal limits of blood chloride may be taken as between 0.27 and 0.32. They consider that the prognosis becomes worse and worse in proportion to the fall of the blood chloride below 0.26. As treatment they advocate giving 500 c.cm. of 5 per cent. saline morning and evening. Exactly what causes the fall in the blood chloride is a matter for conjecture. Some chloride is lost in the vomit, yet Haden and Orr found that a fall of chlorides occurred in the experimentally obstructed rabbit, which does not vomit. Yet in the rabbit the lost chloride might well be in the dilated stomach and duodenum.

A clinical symptom in intestinal obstruction, which may be due to the fall of the blood chlorides, is the cramp that occurs in the calf muscles of some of these patients. Is this similar to the cramp of stokers who lose salt from sweating?

That death occurs earlier in high intestinal obstruction than in low obstruction can be ascribed partly to the earlier onset of vomiting in high obstruction and subsequent loss of fluid. In low obstruction of the ileum Wilkie² was able to show by animal experiment that the fluid secreted into the afferent bowel is re-absorbed by the bowel higher up. Similarly the lack of symptoms in colonic obstruction could be ascribed to the well-known powers of the colon in absorbing fluid.

From these observations I consider that the severe dehydration seen in intestinal obstruction shows all the points of the dehydration complex seen in cholera or severe diabetic coma.

TOXAEMIA OF INTESTINAL OBSTRUCTION

In regard to the toxæmia of intestinal obstruction, Whipple¹⁰ isolated and obstructed a loop of dog's duodenum. An extract from the occluded loop, when injected intraperitoneally into another dog, caused it to die vomiting, with symptoms resembling intestinal obstruction. This toxin, which Whipple considered to be a proteose, could be heated to 70° C. without losing its efficacy.

A certain amount of evidence has been brought against this proteose toxin of Whipple. Magnus Alsleben¹¹ found

* A paper read to the Oxford Clinical Club.

normal duodenal secretion to have the same effect. In a series of experiments Wilkie² found the duodenal toxin to be harmless to normal intestinal mucosa. Williams¹² suggests that these proteose toxins are produced by heating the duodenal contents to 70° C., as was done by Whipple in their preparation. If prepared by filtration and centrifuging, they are non-toxic. I will not discuss at present whether the toxin is a bacterial one produced by *B. welchii*, but state that there is good experimental evidence to encourage the use of anti-gas serum.

TREATMENT

The above considerations have caused me since May, 1929, to treat my severe cases of intestinal obstruction by giving 2½ to 3½ pints of intravenous saline during operation, which is rather a larger quantity than is generally advocated in such cases. I have used normal saline, as it is convenient, and it seems to me that it is essentially fluids that these patients require. There might be something to be said in favour of 1.2 per cent. saline with traces of other salts as used by Rogers in treating cholera patients. The 5 per cent. saline of Haden and Orr certainly gives them salt, but he gives them less than a pint of fluid at a time. In the intravenous saline I give *B. welchii* antitoxin.

The operations are done under local anaesthesia. On return to the ward a continuous rectal saline and glucose is given by the Murphy drip method at the rate of a pint an hour. A dehydrated case will absorb another three or five pints during the night. On return to the ward 20 units of *B. welchii* antitoxin are given intramuscularly, and morning and evening till the bowels open. I have been agreeably surprised at the general condition of these patients next day.

NOTES ON ADVANCED CASES

Case 1.—A female, aged 29, was seen on May 2nd, 1929, with a right inguinal hernia, which had been strangulated for seven days. The skin over the hernia was red and shiny. Faecal vomiting was present. Using novocain as a local anaesthetic I operated, and in incising the skin found pockets of pus in the subcutaneous fat, but no gas. (A culture of this pus grew *B. welchii*.) The sac contained 8 inches of gangrenous small intestine, which was removed by resection; thereafter lateral anastomosis was done, and the peritoneum was drained. Three pints of saline with 50 c.cm. of *B. welchii* antitoxin were given intravenously during the operation, at the rate of one pint every ten to fifteen minutes. A rectal saline of 50 c.cm. *B. welchii* antitoxin was given intramuscularly in the first twenty-four hours, and a similar dose during the second twenty-four hours, following operation. The patient was very fit the morning after operation, and the bowels were open on the third day. The wound was granulating satisfactorily eighteen days after the operation, when the patient, who had had previous mental treatment, became cataleptic, and was transferred to a mental institution.

Case 2.—On August 25th, 1929, a female, aged 70, was seen on account of a right femoral hernia, which had been strangulated for three days. Vomiting was regurgitant and faeculent. At the operation, which was performed under local novocain anaesthesia and by the method of inguinal approach, a loop of strangulated small intestine was found to be viable. During the operation three pints of normal saline and 40 c.cm. of *B. welchii* antitoxin were given. The after-treatment was carried out as described above. Recovery was uninterrupted.

Case 3.—On October 27th, 1929, I operated on a male, aged 56, who had had a strangulated right femoral hernia for five days, and who was suffering from faeculent vomiting. Spinal stovaine was used, and the operation was carried out by the method of inguinal approach. A strangulated knuckle of bowel was found; it was gangrenous along the line of constriction. Resection and lateral anastomosis was done; 2½ pints of intravenous saline were given during the operation, with 50 c.cm. *B. welchii* antitoxin. The after-treatment was carried out as described above, and recovery was uninterrupted.

Case 4.—On December 3rd, 1929, a female, aged 72, was seen by me on account of a right femoral hernia, which had been strangulated for three days. She was having regurgitant vomiting. Using the method of inguinal approach, and employing novocain locally, I operated, and found that the sac contained 4 inches of gangrenous small intestine; I resected 8 inches, and then did a lateral anastomosis. During the operation three pints of normal saline and 40 c.cm. anti-gas serum were given; the routine of after-treatment followed the usual lines. There was some suppuration in the subcutaneous tissues, which were incised on the tenth day. The recovery was otherwise uneventful.

Case 5.—A female, aged 66, suffering from a right femoral hernia which had been strangulated for two days, was operated on by me on June 16th, 1930. Novocain was used as a local anaesthetic, and I chose the method of inguinal approach. Prior to the operation the patient was suffering from regurgitant and faeculent vomiting, but I found that the small intestine, though strangulated, was viable. During the operation 3 to 3½ pints of normal saline and 40 c.cm. *B. welchii* antitoxin were given. The after-treatment was as described above, and recovery was uninterrupted.

SUMMARY

Attention is drawn to the dehydration of intestinal obstruction similar to that stressed by Lawrence in diabetic coma, and by Rogers in cholera. All three diseases show tissue shortage of water, high blood urea, low blood chlorides. Large quantities of intravenous saline (2½ to 3½ pints) associated with *B. welchii* antitoxin can be given advantageously during the operation for intestinal obstruction, followed by rectal saline and glucose after operation.

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Memoranda

MEDICAL, SURGICAL, OBSTETRICAL

SERUM ANAPHYLAXIS

In view of the widespread use of serum in the treatment of various diseases, the two following cases are of importance.

A boy, aged 16, suffering from pyaemia, was receiving, with other treatment, intravenous doses of antistreptococcal serum. He had six of these without any untoward effect. While receiving the seventh dose he suddenly began to cry out that he could not get his breath, and exhibited all the signs and symptoms of anaphylaxis. The previous dose of serum had been given only three days before, and the make of serum used was the same throughout.

The other case is that of a woman, aged 30, suffering from suspected puerperal septicaemia. She had six doses of serum, and while receiving the seventh dose she exhibited the classical signs of anaphylaxis. The previous dose of serum had been given only two days before, and the make of serum used was constant.

In each case the serum was being given intravenously, and the symptoms commenced before the needle had been withdrawn from the vein. Both patients responded very well to adrenaline, and recovered completely in a short time.

These cases are of great interest in that the interval between the two last doses of serum was three days in one case, and two days in the other. Every textbook leads one to believe that serum given in doses at intervals