Upsala J Med Sci 81: 97–102, 1976

Gastric Evacuation and Propulsive Intestinal Motility in Acute Afferent Loop Syndrome in the Rat

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ABSTRACT

The acute afferent loop syndrome, i.e. occlusion of the afferent loop after partial gastrectomy by the Billroth II method, was produced in the rat. In a primary session a gastrojejunostomy with division of the pylorus was performed. 2–3 months later the afferent loop was ligated. The gastric evacuation and the propulsive motility of the intestine were studied quantitatively, using an inert radioisotope. Both the gastric evacuation and the propulsive intestinal motility were considerably delayed in ALS, both in relation to the laparotomized controls and in relation to previous findings in mechanical intestinal obstruction and paralytic ileus due to retroperitoneal irritation or bacterial peritonitis.

INTRODUCTION

The gastric evacuation and propulsive intestinal motility in different pathological conditions of the abdomen and after different intra-abdominal operations have been described by several authors (7–11, 13, 14).

Both gastric evacuation and intestinal propulsion thus appear to be easily affected by different forms of trauma to the abdomen and abdominal organs. The stomach evacuates, however, even in the presence of a low small bowel obstruction (11). Similarly, according to the same authors, a slow propulsion of the intestinal contents takes place above the obstruction. It was considered of interest to find out how the gastrointestinal propulsive motility is affected by an even more violent acute abdominal catastrophe than obstructive ileus. For this purpose the acute afferent loop syndrome (ALS) was used. This condition comprises a total obstruction of the afferent loop following a partial gastrectomy by the Billroth II method. In ALS, which may appear in the immediately postoperative stage after a gastrectomy or much later (2), there is a rise in pressure in the occluded loop, and in dog experiments the pressure increased in some cases up to 100 mmHg. Within 24 hours the occluded loop shows signs of necrosis, there is evidence of hepatocellular degeneration in the liver, and a fully developed pancreatitis is observed (3). Death may follow within a few days, both in man and in experimental animals (2). The syndrome thus fulfils the criteria of an acute severe abdominal catastrophe. The present study was considered of importance also for understanding of the acute afferent loop syndrome.

MATERIAL

112 male rats of the same strain (Sprague-Dawley) were used. The animals were part of two series of a study of the pathophysiology in the acute afferent loop syndrome (1). The distribution of the animals into groups, and their body weights, are given in Table I.

METHODS

Operative technique. In a primary session a gastroenterostomy with division of the pylorus was performed. Two months later the main experiment was carried out, whereby the afferent loop was ligated with a 3–0 silk ligature close to the gastroenterostomy (ALS series) (Fig. 1). The abdomen was closed with single silk sutures in two layers. Further details of the operation have been given in (1). The control rats (laparotomized controls—LC) also underwent gastroenterostomy 2 months prior to the main experiment; this comprised the same dissection in the abdomen, the afferent loop being brought forward and a silk ligature being placed around it close to the gastroenterostomy, but without tying of the ligature.

The gastric evacuation and propulsive intestinal motility were analysed by a method previously described in detail (5). A standardized test meal containing a radioactive substance ($Na_2{}^{51}CrO_4$) was deposited in the stomach im-

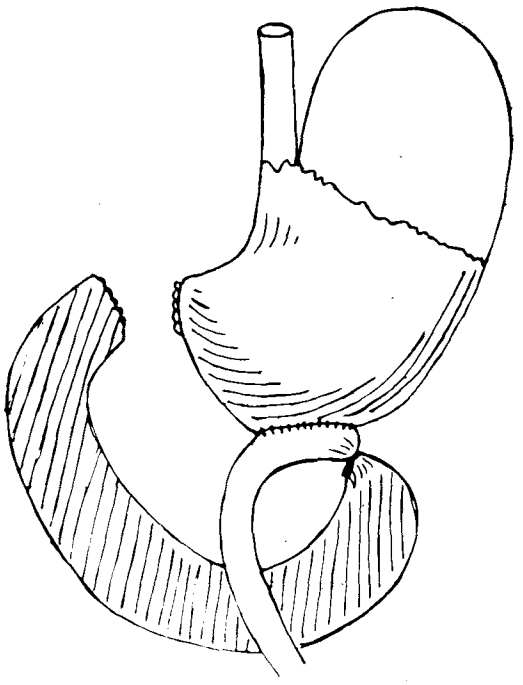

Fig. 1. Acute afferent loop syndrome in the rat.

mediately after the afferent loop had been ligated or, in the control experiments, after the dissection of the afferent loop. After 0.5, 4, 8 and 12 hours (ALS series) and 0.5 and 4 hours (control series) the animals were anaesthetized with ether on an open mask and killed by cardiac puncture and exsanguination. The abdomen was opened by a large incision and ligatures were placed around the cardia and around the efferent loop at the gastroenterostomy. Prior to this the entire afferent loop had been excised (1).

The whole gastrointestinal tract from the stomach to the anus was then taken out by careful dissection. The organs were then placed on a Plexiglas plate which was moved under a scintillation detector synchronously with an automatic linear recorder, connected to the recording unit of the scintillation detector. A quantitative study of the distribution of the radioactive test dose was then made, a planimetric analysis being performed on the area corresponding to the stomach and on each of 10 equal fractions into which the area of the small intestine had been divided. The caecum and colon were anlysed planimetrically as one combined area. Each planimetrically determined area was expressed in percent of the total area of the curve, i.e. in per cent of the total amount of radioactivity in the gastrointestinal tract, corresponding to the radioactive dose.

Further information was obtained by analysing the results by the so-called quotient method described by Grevsten, Johansson & Nylander (6):

$$\mu_i = \frac{x_i}{V + \sum_{j=1}^{i-1} X_j}$$

where

μ_i = the propulsive quotient for intestinal fraction i

x_i = the radioactive content of fraction i

V = the radioactive content of the stomach

$\sum_{j=1}^{i-1} X_j$ = the radioactive content of all fractions proximal to fraction i

RESULTS

The increase in weight in the different groups between the primary operation and the main experiment is given in Table I. The same table gives the haematocrit value at the primary operation and at the main experiment and the number of animals in each group. Only a few animals in the group with 12 hours' occlusion of the afferent loop died. The animals were in surprisingly good condition at the time of the main experiment, with the exception of the 12-hour group, which appeared extremely lethargic and ruffled and showed a very pale oral mucosa and a poor tendency to bleeding from the tail, as signs of peripheral vascular contraction.

Gastric evacuation

The gastric evacuation is given as the proportion of the radioactive dose remaining in the stomach at a given time point, expressed in per cent of the total dose administered. The mean values and the errors of the means for the percentage of the test dose recorded in the stomach and analysed planimetrically are given for the different groups in Table II. Figure 2 shows the corresponding values plotted in a coordinate system, where the radioactive gastric content is given on the ordinate and the duration of occlusion of the loop on the abscissa. It is seen that the gastric evacuation is exponential, and is markedly delayed in the afferent loop syndrome compared with the laparotomized controls.

Since the comparison of importance was that between laparotomized controls and ALS animals, the motility in intact controls with a gastroenterostomy alone was not studied in these experiments. The gastric evacuation in such animals has been shown by Nylander & Wikström (10), and their results are also included in Fig. 2 (IC). The difference between the gastric evacuation in the control and ALS groups is significant, both at experimental times of 0.5 and 4 hours ($p<0.0005$).

Table I. *Data concerning the two series*

Group	No. of animals	Initial wt.	Final wt.	Initial haematocrit	Final haematocrit
Control series					
0.5 hr	16	233±10.7	385±10.3	48±0.7	33±1.1
4 hrs	19	244± 7.9	400± 8.0	49±0.4	32±1.2
ALS series					
0.5 hr	17	230± 4.8	394±12.6	49±0.5	36±1.6
4 hrs	24	244± 6.2	231± 7.5	50±0.7	35±1.5
8 hrs	18	238± 5.3	318±10.5	47±0.7	36±1.2
12 hrs	18	238± 5.7	406± 7.8	49±0.4	31±1.0

Table II. *Planimetric analysis of the percentage distribution of the radioactive dose in the stomach, in fractions of the small intestine and in the colon*

	Laparotomy controls		Afferent loop syndrome			
	0.5 hr	4 hrs	0.5 hr	4 hrs	8 hrs	12 hrs
Stomach	67.8±4.20	30.8±7.75	89.9±2.36	65.5±6.05	63.4±6.69	42.7±7.32
Fraction						
1	19.5±2.10	5.9±1.38	8.8±1.82	14.1±1.97	18.2±2.49	15.3±2.43
2	8.4±1.66	5.2±0.98	1.4±0.69	5.3±1.10	10.8±3.03	9.1±2.36
3	3.4±1.12	1.8±0.63	0.2±0.20	3.3±0.97	4.3±1.48	6.1±1.34
4	1.0±0.70	2.6±0.94		3.1±1.08	2.9±1.29	6.4±1.75
5	0.1±0.14	2.9±1.05		3.0±1.20	1.0±0.71	6.4±2.12
6	0.4±0.38	6.2±1.67		3.4±1.50	0.2±0.24	4.1±1.30
7	0.1±0.09	12.5±3.70		1.4±0.74		2.8±1.03
8		11.0±3.60		1.3±0.24		2.7±1.95
9		6.8±3.30		1.1±0.22		1.7±1.09
10		8.0±3.57		0.8±0.19		0.7±0.48
Caecum+colon		8.8±4.89				2.6±2.15

Propulsive intestinal motility

The percentage distribution of the test dose in the 10 equal, consecutive fractions of the small intestine and the colonic fraction, as obtained by planimetry, is given in Table II. The same values are illustrated graphically in Fig. 3 for control and ALS animals at experimental times of 0.5 and 4 hours, and in Fig. 4 for 4-hour control animals and for ALS animals at 8 and 12 hours. Fractions containing less than 5% of the total test dose were not included in the subsequent analysis. It was found that the propulsion in the distal direction after 0.5 hours had reached the second fraction in the control cases and the first fraction in the ALS group. After 4 hours the colonic fraction was reached in the controls and the second fraction in the ALS group. After 8 and 12 hours the second and fifth fractions, respectively, had been reached in the ALS group. Thus the propulsion was considerably delayed in the acute afferent loop syndrome. The relationship between gastric evacuation and propulsive intestinal motility is evident from the propulsive quotients given numerically in Table III and diagrammatically in Fig. 5. In the acute afferent loop syndrome there is thus a strong inhibition of the intestinal propulsion.

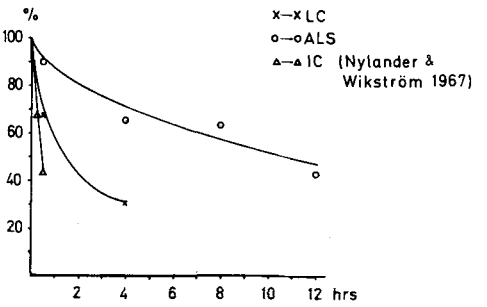

Fig. 2. Diagram illustrating gastric emptying.

Table III. *The propulsive quotient*

Groups		Fraction 1	2	3	4	5	6	7	8	9	10	Colon
LC	0.5 hr	0.29	0.10									
LC	4 hrs	0.19	0.14	0.04	0.06	0.06	0.13	0.23	0.16	0.09	0.09	0.09
ALS	0.5 hr	0.10										
ALS	4 hrs	0.22	0.08									
ALS	8 hrs	0.29	0.13									
ALS	12 hrs	0.36	0.16	0.09	0.09	0.08						

DISCUSSION

The inhibitory effect of laparotomy on the gastrointestinal motility has been pointed out previously by Nylander & Wikström (10), Nylander & Svensson (11) and Kylberg (8), among others. In the present control rats the laparotomy was combined with a dissection of the afferent loop corresponding to that required in order to place a ligature around the afferent loop and thereby produce an afferent loop syndrome. In this way the inhibitory effect was even more pronounced than in the previously described laparotomies. Despite the relatively strong inhibition of the intestinal motility after the laparotomy, there was a significantly more delayed gastric evacuation in the ALS groups and a delay of the propulsive motility. In ALS the entire small intestine is intact, since the pathological process lies in the blind portion which is occluded from the efferent intestinal segment leading from the stomach. Even though this efferent part is completely intact, as well as the communication between the stomach and the efferent loop, ALS leads to pronounced gastric and intestinal paralysis, which was found in these studies to be more marked than has been described in experimental intestinal obstruction in the rat (11). There are certainly several reasons for this. In ALS the pathological process is localized to the upper part of the abdomen in the vicinity of the stomach, while that in a low small bowel obstruction lies in the lower part of the abdomen. ALS has, in addition, a direct effect on three organs, the duodenum, liver and pancreas. Of

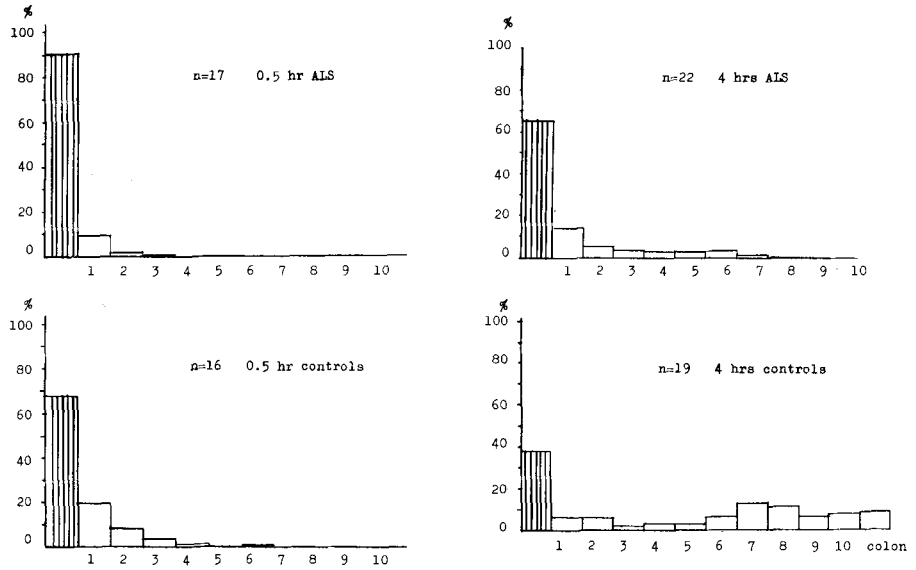

Fig. 3. Histograms representing the quantitative distribution of the radioactive substance in the gastrointestinal sample. Control groups 0.5 and 4 hours and ALS groups 0.5 and 4 hours.

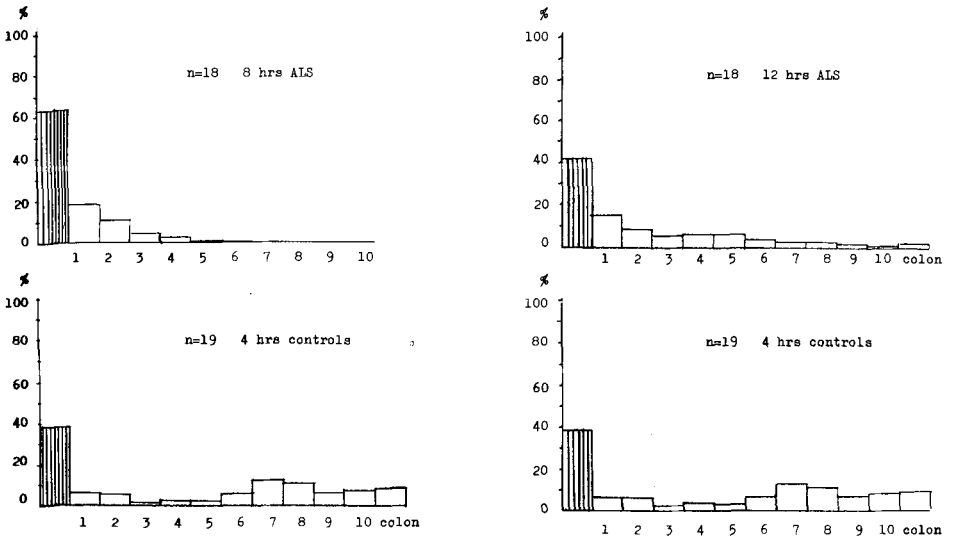

Fig. 4. Histograms representing the quantitative distribution of the radioactive substance in the gastrointestinal sample. Control group 4 hours and ALS groups 8 and 12 hours.

probable importance for the development is the dilated, tense duodenal loop, in which mucosal necrosis gradually occurs (3). This necrosis gives a possibility of leakage into the peritoneum of both bacteria from the bacteriarich duodenal fluid (1) and toxins. Supporting this assumption are the signs of peritonitis with increased fluid in the peritoneum which were observed in all cases.

In ALS, pancreatitis occurs regularly (3, 4, 13). This also contributes both to peritonitis and to paralytic ileus, which is a regular component in all cases of pancreatitis. Added to this is the fact that ALS also gives rise to biliary stasis and hepatocellular degeneration (3), which can contribute to deterioration of the liver function and thereby reduction of the capacity of general detoxification.

Paralytic ileus has been produced previously in the rat both by means of bacterial peritonitis (8) and through retroperitoneal irritation (9). The paralysis of the gastrointestinal tract occurring in the acute afferent loop syndromes seems to be more pronounced, however. Contributing to this is thus the combination of pancreatitis, intestinal necrosis, hepatocellular degeneration and peritonitis, and in the final phase the irreversible shock which develops.

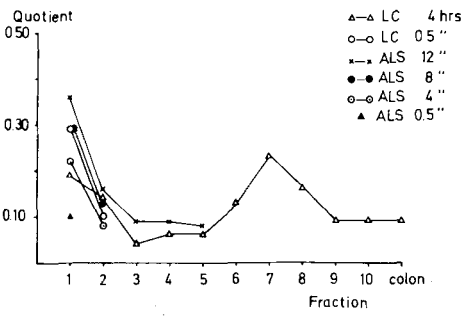

Fig. 5. Diagram illustrating propulsive quotient values.

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Received November 1, 1975

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