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Many different factors have been cited as the cause of the steatorrhea which is not an uncommon sequel of partial gastrectomy. Those structures involved in the digestion and absorption of fat—the gastric pouch, afferent loop, and small intestines—have all been the subject of scrutiny, both clinically and in the laboratory. Several authors[5, 13, 24] have noted that fat malabsorption occurs more commonly and to a greater degree with a Billroth II than a Billroth I reconstruction.

Because of the greater impairment of fat absorption with Billroth II operations, the present study has focused attention on the possible role of the afferent loop in the production of steatorrhea. Particular attention has been given to the effect of bacterial overgrowth within the afferent loop on the development of steatorrhea. An experiment was designed first, to determine whether progressive increase in the length of the afferent loop was predictably associated with increasing fat malabsorption and, second, to determine if overgrowth of bacteria within this loop could be correlated with increased fecal fat loss.

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METHOD

Normal healthy dogs weighing between 10 and 20 kilograms were dewormed and 2 weeks later were started on a special diet consisting of 1 can daily of standard dog food weighing 450 Gm. per can, which assayed 6 percent fat, to which 100 Gm. of lard was added.

Any animal which did not tolerate the high fat diet was eliminated from the study. After 5 days of this diet, a 72 hour total stool collection was obtained. The specimen was stored in a deep freeze. At a convenient time, the stool was thawed and homogenized in a blender. Aliquots were analyzed for fat content by the method of Van de Kamer and associates[17] as modified by Anderson and his group.[1] If the aliquots were not within 3 percent equivalence, reanalysis was done.

The results were tabulated as percent of ingested fat excreted per 24 hours. Any animal which failed to absorb 95 percent or more of the dietary fat per 24 hours was eliminated from the study at this point. The rest of the animals then underwent a 50 percent distal gastrectomy with an antecolic Polya-type Billroth II anastomosis. Afferent loops of 30, 60, and 90 cm. were constructed.

Before opening the gastrointestinal tract, bacteriologic samples were taken from the duodenum. A 10 cm. length of midduodenum was isolated between the fingers of the

Table I. *Thirty centimeter afferent loop*

Dog	*Ingested fat in stool per 24 hours*	
	Preoperative (%)	*Postoperative (%)*
F 61	3.6	5.5
F 62	1.3	1.5
F 55	3.3	4.1
Average	2.7	3.7

Table II. *Sixty centimeter afferent loop*

Dog	*Ingested fat in stool per 24 hours*	
	Preoperative (%)	*Postoperative (%)*
F 67	2.5	7.8
F 70	3.5	14.0
F 95	1.5	8.7
Average	2.5	10.2

Table III. *Ninety centimeter afferent loop*

Dog	*Ingested fat in stool per 24 hours*	
	Preoperative (%)	*Postoperative (%)*
F 96	1.5	23.7
F 72	3.5	31.0
F 73	2.1	19.4
F 88	1.4	38.5
Average	2.1	28.2

Table IV. *Sixty centimeter afferent loop*

Dog	*Ingested fat in stool per 24 hours*		
	Preoperative (%)	*Postoperative (%)*	*With Achromycin*
F 67	2.5	7.8	15.4
F 70	3.5	14.0	22.2

assistant surgeon. Ten cubic centimeters of sterile saline was injected into the occluded segment and thoroughly agitated. Serial dilutions of 0.5 c.c. of the aspirated material were carried out. The various dilutions were then cultured on 5 and 7 percent blood agar, on McConkey agar, LBS agar plate, sodium azide blood plate, and Sabouraud's agar (to which Chloromycetin was added). The plates were incubated both aerobically and anaerobically at 37° C. for 48 hours.

Bacterial colonies were counted. The dilution which gave between 25 and 50 colonies per plate was used in the calculation of the total bacteriologic count. The total count in each of the various media was then added to obtain the total count in the original sample.

Following operation, the animals were given antibiotics for 5 days and when fully recovered were returned to the animal farm until further metabolic studies were carried out. Upon return from the farm, each animal was again dewormed and the original experiment was repeated. The animals were then re-explored, the condition of the afferent loop noted, and repeat bacteriologic studies were obtained from the afferent loops.

Two animals with 60 cm. afferent loops were placed on a 10 day course of Achromycin following recovery from the second exploratory laparotomy. Repeat metabolic and bacteriological studies were carried out, exactly as in the first two steps.

RESULTS

The average fecal excretion in the 10 dogs on a 127 Gm. diet was 2.4 percent of

Table V. *Thirty centimeter afferent loop*

	Dog	*Gram – rods*	*Gram + cocci*	*Anaerobes*	*Others*	*Total*
Preoperative	F 61	4×10^4	8×10^3			5×10^4
Postoperative		2×10^4	3×10^6		2×10^4	3×10^6
Preoperative	F 62	5×10^3				5×10^3
Postoperative		7×10^3	1×10^5	10^3		1×10^5
Preoperative	F 55	1×10^4				4×10^4
Postoperative		1×10^3				1×10^3

Table VI. *Sixty centimeter afferent loop*

	Dog	*Gram – rods*	*Gram + cocci*	*Anaerobes*	*Others*	*Total*
Preoperative	F 67	1×10^3	2×10^5			2×10^5
Postoperative		7×10^3	2×10^3	1×10^7	4×10^3	1×10^7
Preoperative	F 70		No data available			
Postoperative		1×10^3				1×10^3
Preoperative	F 95			5×10^4		5×10^4
Postoperative		7×10^5		5×10^4		8×10^5

the ingested fat. This is similar to results both in dogs [5, 21] and in humans[2, 15, 22] carried out in other institutions. The animals with 30 cm. afferent loops were able to digest and absorb the fat diet without any apparent difficulty (Table I).

As the afferent loops became longer, the steatorrhea increased. With a 60 cm. loop, the average fecal fat excretion increased to 10.2 percent (Table II) and with a 90 cm. loop, to 28.2 percent (Table III).

The stools in the animals with 90 cm. afferent loops were bulky, frothy, foul-smelling, and often similar in color to the ingested food. These dogs all lost weight and became emaciated despite ravenous appetites. All succumbed to malnutrition after 5 to 6 months.

The 2 animals which were given a 10 day course of Achromycin had no decrease in steatorrhea and, in fact, an apparent increase (Table IV).

Bacteriology. A sterile duodenal culture was found in only 1 of the 10 fasting animals. The presence of small numbers of bacteria in the upper small intestine of dogs is normal. Meleney and co-workers[14] found only 1 sterile duodenum in a study of 25 animals.

There were 3 principal groups of organisms noted: aerobic gram-negative bacteria of the B coli group; aerobic gram-positive cocci of the streptococcus group; and anaerobic bacilli. In the last group, bacteroides was more common than clostridia, the latter being cultured in only one animal (Tables V, VI, VII).

Postoperatively, there was an over-all increase in the number of bacteria found in the afferent loop, although there was very considerable overlap between the normal and the postoperative count. However, there was no apparent correlation between the increase of bacteria and the degree of steatorrhea noted. The animals with 30 cm. afferent loops and no steatorrhea had about the same increase in total bacterial count as the animals with 90 cm. loops and massive steatorrhea.

Qualitatively, there was no significant or consistent pattern between the flora of the normal duodenum and that of the afferent

Table VII. *Ninety centimeter afferent loop*

	Dog	*Gram – rods*	*Gram + cocci*	*Anaerobes*	*Others*	*Total*
Preoperative	F 96			6×10^2		6×10^2
Postoperative			4×10^3	2×10^4		2×10^4
Preoperative	F 73					
Postoperative		2×10^3			1×10^4	1×10^4
Preoperative	F 72	7×10^2	2×10^4		3×10^5	3×10^5
Postoperative				1×10^6		1×10^6
Preoperative	F 88	3×10^5	5×10^2			3×10^5
Postoperative					7×10^3	7×10^3

Table VIII. *Sixty centimeter loop with Achromycin*

	Dog	*Gram – rods*	*Gram + cocci*	*Anaerobes*	*Other*	*Total*
Preoperative		1×10^3	2×10^5			2×10^5
Postoperative	F 67	7×10^3	2×10^3	1×10^7	4×10^3	1×10^7
Postoperative, with Achromycin				2×10^3		2×10^3
Preoperative			No data available			
Postoperative	F 70	1.3×10^3				1×10^3
Postoperative, with Achromycin		5×10^2				5×10^2

loop. In general, the organisms present originally were found again postoperatively with other flora on occasion being cultured in addition. In only 2 animals, F 72 and F 88, were entirely different flora noted between the preoperative and the postoperative study.

Following the Achromycin therapy in 2 animals, the bacterial count decreased in the afferent loop. In 1 dog, F 67, anaerobes became the predominant organism (Table VIII).

DISCUSSION

Many factors have been implicated as the cause of increased fat loss following partial gastrectomy and Billroth II anastomosis. Some investigators believe that failure to maintain weight is related to decreased caloric intake rather than to any primary absorption defect.[21]

Others feel that changes in gastrointestinal motility or reservoir function are responsible for the steatorrhea. In a clinical study, Saxon and Ziese[16] stated that loss of the reservoir function of the stomach was of primary etiologic importance. He noted that loss of body weight in his patients could only be correlated significantly with the amount of stomach removed at operation and with no other factors. Waddell and Wang,[20] on the other hand, felt that abnormal motility rather than lack of reservoir function was the basic physiologic disturbance involved. Glazebrook and Welbourn[6] indicted intestinal hypermotility as the cause of the postgastrectomy steatorrhea.

Inasmuch as several metabolic studies have demonstrated consistently greater malabsorption after the Billroth II compared to the Billroth I anastomosis,[4, 5, 13, 23] there has been a recent increasing interest in the etiologic role of the afferent loop. Some British authors[4] have related the presence of an afferent loop to subsequent ileojejunal insufficiency. Other authors believe that there is a physiologic derangement of pancreatic and biliary excretory function due to loss of cyclic coordination with gastric emptying[8-10, 12, 15] or lack of stimulation of these secretions.[3, 17] The last group of investigators have felt that bypassing of the duodenum by chyme, an important stimulus for the release of secretin and cholecystokinin, was the underlying pathologic mechanism.

The results of the present study support the concept that the afferent loop can be a most important factor in the cause of postgastrectomy steatorrhea, depending upon the details of its construction. Animals with short afferent loops did not demonstrate any significant steatorrhea. As the length of the afferent loop increased, a concomitant and dramatic rise in fecal fat excretion was noted.

The role of bacterial overgrowth within the afferent loop as the cause of steatorrhea has apparently been well documented clinically.[7] This is usually a demonstrable factor only in those uncommon cases in which there is a poorly draining afferent loop.[18] Since the decreased fecal fat loss with Billroth II anastomosis is the rule, however, rather than the exception, it could be predicted that overgrowth of bacteria in the afferent loop is not the usual cause of postgastrectomy fecal fat loss. The present study

confirms this reasoning. There was no significant correlation between the number or type of bacteria in the freely draining afferent loop and the degree of steatorrhea measured. In 2 animals to which antibiotics were given, the steatorrhea persisted and, in fact, increased despite a lowering of bacterial content of the afferent loop.

While the present study indicates that bacterial factors do not contribute to steatorrhea if there is an unobstructed afferent loop, the data do not delineate the cause for the defect in fat absorption. The malabsorption is probably not due to defunctionalization of the upper jejunum in view of Kremen's[11] demonstration in dogs that over half the jejunum can be bypassed under other circumstances without producing steatorrhea. A more likely explanation may be that a relative pancreatic or biliary insufficiency results from diversion of the respective digestive juices into a partially defunctionalized loop from which they may not efficiently mix with ingested intestinal contents. Alternatively, the exclusion of food from contact with the duodenum may prevent full activation of the secretin mechanism with consequent attenuation of exocrine pancreatic stimulation. Whatever the mechanism, the present study emphasizes the need for use of a short afferent loop if a Billroth II reconstruction is used.

SUMMARY

An attempt was made to correlate Billroth II gastrectomy in steatorrhea in dogs with the length of the afferent loop. With greater lengths of afferent loop, increasing steatorrhea was noted.

Qualitative and quantitative bacteriologic studies of the duodenum were carried out before and after creation of an afferent loop. Postoperatively, there was very little change in bacterial content with different lengths. Antibiotics which reduced the bacterial count of the afferent loop did not mitigate the steatorrhea.

This study indicates that postgastrectomy steatorrhea with a Billroth II anastomosis is related to the length of the afferent loop, although the precise physiologic mechanism is not clear. Possible explanations for the steatorrhea are discussed.

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