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Afferent loop syndrome (bilious vomiting following partial gastrectomy)

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THE AFFERENT LOOP SYNDROME

(Bilious vomiting following partial gastrectomy)

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Bilious vomiting, or the "afferent loop syndrome" is a rare complication of sub-total gastrectomy. This symptom complex has emerged as a distinct clinical entity among the several post-gastrectomy complications.

Typical symptoms are quite distinctive. The hallmark of the syndrome is vomiting of a watery, bile stained, fluid, unmixed with food from one hour to ninety minutes following a meal. The attack is preceded by a subjective feeling of upper abdominal fullness developing into a sharp, stabbing pain. The episode is terminated by vomiting copious amounts of bilious, alkaline, fluid quite unmixed with the ingested meal. Vomiting is followed by a feeling of hunger and well being. Symptoms may progress in severity in the post-gastrectomy patient, eventually resulting in severe debilitation and undernutrition in some cases.

An encounter with these unusual symptoms in a post-gastrectomy patient has inspired an attempt to clarify the etiology and nature of the "afferent loop syndrome."

This patient was a fifty year old man who after ten years of increasingly disturbing peptic ulcer disease underwent a subtotal gastrectomy for a gastric ulcer five months prior to the onset of bilious vomiting. The symptoms attributed to the afferent loop syndrome became disabling and did not respond to active, non-operative treatment. A variety of operative maneuvers were employed to no avail until the secretions entering the duodenum were diverted from the stomach. The course of events challenged my comprehension of upper gastrointestinal tract interrelations, leading to this review.

A brief review of normal gastric physiology will be presented, along with a discussion of the altered state encountered in the gastrectomy patient.

In the normal person, the motor functions of the stomach are three: (1) storage until the ingested material can be accommodated in the lower portions of the gastrointestinal tract, (2) mixing of gastric secretions and the ingested food to form an isotonic, semi-fluid chyme, and (3) propulsion of the food into the small intestine at a slow rate.[1]

There are several neural and humoral mechanisms involved in gastric emptying. The simple presence of food in the stomach, with distention of the gastric wall, has been shown to initiate vagal stimulus to increase the secretions of the gastric glands. Secondarily, irritation of the gastric mucosa causes increased peristaltic waves which tend to move the material to the pylorus. Normal pyloric tone is inhibited as each antral wave approaches, so that only a small amount of chyme expelled into the duodenum with each wave.[2]

Chyme of the proper consistency and tonicity is presented to the duodenal mucosa. Upon increasing pressures in the duodenum, the "enterogastric" reflex is initiated. This neural phenomenon inhibits gastric emptying by different pathways. Feedback through the myenteric plexus to inhibit gastric contractions is a factor. Vagal afferent fibers to centers in the medulla serve to influence gastric secretion and peristalsis by way of vagal efferents. Sympathetic pathways through the celiac plexus also play back to regulate the speed of gastric emptying. The elements of the "enterogastric reflex", then are mechanisms by which the small

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1. Guyton, A.C. Textbook of Medical Physiology Philadelphia: W. B. Saunders Company, 1966, p. 882

intestine influences neural pathways. Thus stimuli, whether from low pH of the chyme, hypo-or hypertonicity, or simple distention of the duodenum, tend to limit gastric emptying.[3]

A humoral mechanism is also at play in the delay of gastric emptying. Fatty acids in the chyme stimulate the duodenal and jejunal mucosa to release a hormonal substance called "enterogastrone", which slows gastric emptying and allows increased time for digestion of the fatty chyme.[4]

Gastric secretions have served to sterilize the ingested material and to initiate peptic digestion of proteins. These actions have been augmented by the considerable time spent in transit of the food through the stomach.[5]

Following gastrectomy, much of the secretory and regulatory functions of the stomach are lost. The degree of alteration depends on the amount of the stomach resected. Bypass of the normal mechanisms and lack of dilution capacity provide a great stimulus to peristalsis as hypertonic material reaches the intestinal mucosa. Stasis and reflux into the gastric remnant also tend to become problems created by the altered anatomy. Reduction of the acid content of the food material interferes with absorption of different substances, including iron, vitamins and various food types. Colon bacteria grow uninhibitedly all the way up into the gastric remnant.[6] All of these factors play a role in the

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3. Ibid p. 885
4. Ibid p. 885

5. Davenport, H.W. Physiology of the Digestive Tract, Chicago: Year Book Medical Publishers, 1966, p. 117
6. Wells, C. and Welbourn, R. "Post-Gastrectomy Syndromes", British Medical Journal March 17, 1951 1:546

response of the patient after gastrectomy.

Gastric resection was first developed early in the 1880's by the Vienna surgeon, Christian Albert Billroth (1829-1894). In the beginning, the procedure was used only as treatment for gastric carcinoma.

Mortality and morbidity were quite high. As time passed, techniques improved and mortality from the procedure decreased. The use of Billroth's operations, I (gastroduodenostomy) and II(gastrojejunostomy) was gradually extended to the treatment of bleeding or intractable peptic ulceration of the stomach.[7]

Billroth's operations were, at first, regarded as a panacea in the treatment of gastric carcinoma and peptic ulcer disease. They were extensively and liberally used in the last of the nineteenth and early twentieth centuries. One of the earliest reports of difficulty following gastrectomy appeared in 1913, in which the first attempt was made to describe and classify the several post-gastrectomy syndromes that are recognized today.[8]

The literature, for the years from 1913 onward, is filled with unclear and often conflicting reports of anatomic, physiologic and biochemical problems after gastrectomy. Today, most authorities recognize a number of separate entities under the heading of post-gastrectomy syndromes. The "dumping syndrome" or"efferent loop syndrome", (early and late varieties), the"afferent loop syndrome" and the small stomach syndrome. Secondarily, metabolic complications such as malabsorption of fat, Vitamin B12 and iron deficiency are also seen.

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7. Woodward, M.D., The Post-gastrectomy syndromes, Springfield, Ill.: Charles C. Thomas Publishers, 1963, p. 56
8. Hertz, A.F. "The cause and Treatment of Certain Unfavorable Effects of Post-Gastrectomy Syndromes" Lancet, 2:479, 1956, p. 480

The "efferent loop" or "dumping syndrome" is the most common and well known of the post-gastrectomy syndromes. It will be described briefly for purposes of distinction. Symptoms are reported in from one to seventy percent of post-gastrectomy patients, depending on the series, criteria and follow-up methods. The frequency and severity tend to decrease as time passes, because the patient is able to adjust to the altered state. Serious, debilitating symptoms will persist in less than ten-percent of patients.[11] Symptoms of the early variety consist of subjective feelings of discomfort and abdominal distention, palpitations, weakness, warmth and sweating, dizziness, nausea and possibly diarrhea. Symptoms begin at the end of a meal or within thirty minutes thereof.[12, 13]

The mechanism of the "dumping syndrome" has been well established. A sudden overloading and distention of the jejunum with a hypertonic bolus of food begins the sequence. Fluid is drawn into the jejunal lumen with resultant depletion of plasma volume. Electrolyte loss into the bowel in an attempt to adjust the tonicity of the food material results in systemic depletion.[11] The body responds with reflex mechanisms which attempt to control plasma volume and electrolyte concentration. Catecholamine release by the adrenal medulla produces cardiovascular and neurovascular responses. Antidiuretic hormone from the posterior pituitary and aldosterone from the adrenal cortex also are released to aid in restoring plasma volume. These actions and responses result in the "early dumping" seen in the immediate post-prandial period.

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11. Harrison, T.R. Principles of Internal Medicine, New York: McGraw-Hill Book Co., 1966 p. 984

12. Wells and Johnson, Op. Cit., p. 483.

13. Lake, N.C. "The Aftermath of Gastrectomy." British Medical Journal, 2:265, 1951

"Late dumping" consists of palpitations, lightheadedness and possible fainting in the period from one to two hours following a meal. This particular entity has been shown to result from a reactive hyper-secretion of insulin. The absorption of glucose precedes at a rapid rate in the immediate post-prandial period within the proximal jejunum, particularly if the meal has been rich in carbohydrate. A large amount of food "dumped" into the jejunum results in rapid absorption of glucose and hyperglycemia. The pancreas reacts with increased insulin production which may "over-shoot" with resultant hypoglycemia creating the "late dumping" entity.[12]

Treatment for the "efferent loop" syndromes is non-operative. Small, frequent feedings low in carbohydrates have been used successfully.

Another post-gastrectomy syndrome, the "small stomach syndrome," is characterized by early satiety, a feeling of fullness, lethargy and mild discomfort following meals. This is rarely severe or incapacitating and is relieved by resting after meals. It is described by gastrectomy patients as being exactly like the feeling normal persons have following a large meal. The feelings are simply due to the decreased stomach capacity plus mild distention.[15, 16]

Various malabsorption states have been described in post-gastrectomy patients. These include anemias, steatorrhea and undernutrition. The usual post-gastrectomy patient will adjust to the altered physiology in time to prevent such secondary developments.[17]

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12. Wells and Johnson, Op. Cit., p. 483.

15. Capper, W.M., Welbourn, J. "Early Post-Cibal symptoms following Gastrectomy." British Journal of Surgery 43:406, 1955.
16. Lake, N.C., Op. Cit., p. 267
17. Ibid., p. 269.

The afferent loop syndrome was first recognized and described as a distinct clinical entity in the post-gastrectomy patient in the early 1950's by the British surgeons, Wells and Welbourn.[18] Their work, along with subsequent studies by others, has clearly delineated the afferent loop syndrome or postgastrectomy bilious vomiting from the other post-gastrectomy complications. This distinction has not always been so clear-cut. Other authors, prior to 1951, have been reluctant to make this distinction.

Nearly all of the clinical work and interest in the "afferent loop syndrome" is recorded in the British literature. American surgeons have shown little interest in this particular problem. This disinterest may be related to failure to recognize the syndrome as a distinct entity or it may be such an infrequent complication that individual experience with it is limited. At any rate, a smaller incidence is represented in the American literature.[21]

The symptoms of a patient suffering from the afferent loop syndrome are generally described in quite similar terms by different authors. The characteristic attack begins after the ingestion of a meal, from a few minutes to one hour. Acute onset of epigastric pain often described as a bursting, pressing or cramp-like sensation is noted. Not infrequently, the pain radiates to the back and scapular region bilaterally. This distress usually lasts no more than a few minutes and is suddenly and completely relieved by vomiting.[22] The vomitus is always des-

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18. Wells and Welbourne, Op. Cit. P. 548

21. Herrington, J.L. "Experiences With the Surgical Management of the Afferent Loop Syndrome." Annals of Surgery, 164:799, Dec. 1964

22. Wells, C.A. and Macplee, I.W. "The Afferent Loop Syndrome" Lancet, Dec. 20, 1952 p. 1190

cribed as bile-stained fluid, unmixed with the previously ingested meal.[23,24,25] The act of vomiting relieves the pain and leaves the patient with a feeling of well being and hunger. The amount of fluid vomited may be copious, from four-hundred up to one-thousand cc of bile-stained fluid consisting of bile, pancreatic secretions and duodenal secretions. The amount of these secretions is approximately twelve-hundred to fifteen-hundred cc per twenty-four hours.[26] No relationship has been noted between the severity of the attack and any particular type of food. However, the amount of food ingested sometimes is associated with severity. Small feedings tend to decrease the severity of symptoms. The patient may sometimes suffer from post-prandial diarrhea, but no other manifestations of the "dumping syndrome" are noted in uncomplicated cases.[27]

Bilious vomiting of chronic, persistent nature results in a gradual decline with increasing disability over a period of months or years. The mechanism was thought to be purely obstructive in the early days. Mechanical obstruction, of an intermittent and reversible nature, seemed to explain adequately the observed symptoms. Bilious vomiting is virtually unknown following a gastroduodenostomy. This fact, in itself, indicated mechanical obstruction as only the gastrojejunostomy procedure leaves the so-called blind afferent loop. In general, then,

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23. Wells and Johnson, Op, Cit. p. 482
24. Tanner, N.C., Op. Cit., p. 152
25. Jordan, G.L. Jr., "The Afferent Loop Syndrome." Surgery 38:1029, 1955
26. McNealy, Op. Cit., p. 211
27. Woodward, Op. Cit. p. 59

a clear-cut obstructive cause has been found at the time of correction, but this has not always been the case.[28,29]

Many different explanations have been offered. If the afferent loop is excessively long, twisting and volvulus of the segment, at the anastomosis, are more likely. The complication of bilious vomiting is more frequent when the anastomosis has been made in the ante-colic as opposed to the retro-colic manner.[30] If the ante-colic loop is too short, tension at the juncture may cause narrowing to compromise the jejunal stoma.[31] Traction and kinking by the mesentery have been felt to be responsible for a sharp angulation at the anastomotic site in some cases.[32] Intussusception of the afferent loop into the gastro-jejunal stoma, or into the efferent loop itself, with subsequent compromise of the vascular suppy and infarction has been reported in acute cases.[33]

In chronic bilious vomiting, the mechanism is not always clear-cut at laparotomy. Duodenal and pancreatic secretions and bile may accumulate in a long afferent loop and by sheer weight cause kinking and increased angulation at the stomal site. Relief by vomiting would decompress this loop.[34]

Another interesting possibility has been proposed in more recent years. Some observers have witnessed a significant number of cases in which no anatomic

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28. Ibid., p. 61
29. Bartlett, M.K. and Burrington, J.D. "Bilious Vomiting After Gastric Surgery," Archives of Surgery, July, 1968 P. 37
30. West, J.P. "Obstruction of the Proximal Jejunal Loop Following Gastrectomy." American Journal of Surgery 1956, p.99
31. Wells and MacPhee, Op.Cit. p.1192
32. Simon, M.M. "Obstruction of the Proximal Jejunal Loop Following Gastrectomy." American Journal of Surgery 91:423, 1956
33. Hoffman, W.A. and Spiro, H.M. "Afferent Loop Problems" Gastroenterology 40:203, 1966
34. Adams, D.D. and Renstein, M.D. "Acute Obstruction of the Afferent Loop Following a Billroth II Gastrectomy." The American Surgeon 31:505, Aug, 1965

cause for stasis could be discovered at the time of revision. Dialation of the gastric remnant itself upon ingestion of food to produce a transient angulation or torsion of the stoma has been indicted. The possibility of some functional disturbance of motility and retention of secretions in the afferent loop has also been suggested. The mounting pressure of secretions in the afferent loop, stimulated by the presence of food, soon builds to a point where the secretions are injected into the gastric remnant. In the meantime, the ingested material would have moved on into the efferent loop. This proposed sequence would account for the fact that the vomitus is unmixed with food. Assuming, then, that these alkaline secretions are irritating to the gastric mucosa, vomiting would follow, pressure would be relieved and symptoms would cease.[35]

As noted previously, mechanical obstruction though offering a plausible explanation for the afferent loop syndrome, is not always observed.[36,37] Williams and Toye[38] described a case which provided a unique opportunity to study the etiology and dynamics of bilious vomiting. The investigation was prompted by the observation of these workers that relief of symptoms did not always follow the normally successful conversion operations.

The subject was a fifty-six year old white man who had suffered from bilious vomiting for eight years. He had been previously treated, for peptic ulcerations of the duodenum, with a gastro-jejunostomy. The bilious vomiting had become gradually more severe and debilitating over the eighteen months before he pre-

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35. Woodward, Op. Cit. p. 62

36. Ibid., p. 64
37. Bartlett and Burrington, Op. Cit., p. 36
38. Toye, D.K.M. and Williams, J.A. "Post-Gastrectomy Bile Vomiting" Lancet II, Sept. 1965 p. 526-527

sented himself for treatment. His presenting complaint was right upper quadrant pain with typical cholelithiasis symptoms. Oral cholecystogram showed a gallbladder full of stones. The patient agreed to allow the surgeons to study his bilious vomiting problem by leaving a drainage tube in the duodenal stump at the time of cholecystectomy.

The investigators found that the amount of duodenal drainage collected after each meal amounted to approximately two-hundred cc per hour. The patient experienced no bilious vomiting while the tube was draining freely. They then tested the patient's reactions upon infusion of various test solutions into the afferent loop and stomach. The solutions were infused at body temperature at a volume of no more than four-hundred cc at any given time. By infusion of the patient's own duodenal secretions, with the infusion apparatus out of the patient's view, they were able to produce typical symptoms by infusion of two-hundred to three-hundred cc. The rate of infusion and positioning of the patient (supine or upright) had no effect. Infusion of four-hundred cc of saline solution produced only a full feeling without nausea or pain.

The maneuvers were then repeated with 2:1 mistures of duodenal secretions with saline containing gastrographin for X-ray studies of the system. Upon the infusion of the saline mixture into the duodenum, fluoroscopic examination revealed rapid emptying of the afferent loop into the gastric remnant as well as the efferent loop. Once again, the patient felt no pain or nausea, only a subjective feeling of gastric fullness. When the gastrographin-duodenal secretion mixture was injected, the afferent loop emptied into the stomach and efferent loop as before. Symptoms occured when about three-hundred cc of dye had been infused, and at this point, the efferent loop emptying had stopped. Vo-

miting of the material from the stomach brought about immediate relief of symptoms.[38]

In the above study, the factor in symptom production seemed to be the contact of the bilious material with the gastric mucosa, rather than simple stasis in the afferent loop. This observation is in agreement with others made at gastroscopy in patients suffering from bilious vomiting. Reddened irritated gastric mucosa compatible with long-term gastritis has been observed in such patients.[39] It seems probable then, that in the chronic bilious vomiting syndrome, gastric hypersensitivity and mucosal irritation are contributory, if not causative, in many cases.

Chronic, intermittent obstruction of the afferent loop and bilious vomiting in which there is no anatomic or mechanical cause do not seem to be distinguishable on the basis of symptomatology. In fact, no great advantage would be gained from such a distinction in light of present knowledge. Any corrective procedure should be designed to prevent reflux of the bile and duodenal secretions into the gastric remnant thus preventing the "alkaline gastritis."[40] Any corrective procedure would also be aimed at eliminating causes of mechanical obstruction as well. However, in the earlier literature, tests were devised to demonstrate obstruction. The so-called Jordan test is one of these. A Levine tube, weighted with mercury, is passed six to twelve inches beyond the gastrojejunal stoma and it's position is confirmed by X-ray. A sample is aspirated and this is followed by a test meal of sixty cc of an oil-fat emulsion, two hundred forty cc of cow's milk and one hundred eighty cc of a forty percent glucose solution. Aspirations are then made at one minute and

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38. Toye, D.K.M. and Williams, J.A., "Post-Gastrectomy Bile Vomiting" Lancet II Sept, 1965, p. 526-527
39. Woodward, Op. Cit., p. 57
40. Jordan, G.L., Op. Cit. p. 1031

following at five minute intervals until good mixing with the bilious secretions is seen. If obstruction of the afferent loop is present, one will observe the absence of bile in the aspirant while symptoms are present. The presence of bile in the aspirant indicates no obstruction, or, if symptoms are present and then relieved and bile noted with relief, the bile has passed the obstruction point.[40]

Another confirmatory test, described by Dahlgren (Dahlgren test), is believed by Herrington to be a more reliable indication of afferent loop obstruction.[41] This procedure consists of the injection of secretin intravenously. Secretin, a polypeptide hormone present in the mucosa of the upper small intestine, is released when chyme from the stomach enters the small bowel. Secretin causes the pancreas to secrete large quantities of a fluid high in bicarbonate ion (ius meq/l) called hydrelatic secretion. The fluid is composed principally of a watery solution containing almost no enzymes. This fills the afferent loop. If reversible obstruction is present, typical symptoms should follow secretin injection within approximately fifteen minutes. The symptoms are then relieved by bilious vomiting after the pressure in the afferent loop has become sufficiently high to relieve obstruction.[42,43]

The tests described above were found to be of interest in explaining the afferent loop syndrome in the earlier days of investigation of this entity. However, with the more recent observations and concept of sensitivity of the gastric mucosa

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40. Jordan, G.L., Op.Cit. p. 1031

41. Herrington, Op.Cit., p.806

42. Woodward, Op. Cit., p. 61

43. Herrington, Op. Cit. p. 801

to the alkaline secretions, it is apparent that their usefulness if limited. Cases have been reported in which a mechanical cause was found and corrected only to have symptoms continue or relapse again after transitory improvement.[45,46] It would seem, then, that both obstructive and irritative factors are at work in different cases of bilious vomiting. In some instances, both may be a factor in a single case. Reflux and obstruction must both be corrected to bring about a cure.

It is obvious that the type of operation performed initially bears a great deal of importance in the development of bilious vomiting. This has not been encountered after gastroduodenostomy. The rate of minor differences in operative technique and of special conditions encountered in the abdomen at the time of operation (adhesions, anomalies) has been difficult to assess. At least one report has indicated that the problem is more common in patients in whom a good deal of scarring and adhesions around the intestine are encountered at the initial operation.

The conditions mentioned in the preceding paragraph will obviously introduce many variables and complicate the determination of the actual incidence of bilious vomiting. A wide variation in incidence, along with a relative paucity of reports is encountered in the literature. Bilious vomiting of the chronic type varies in reported incidence from 53%[48] over-all to 5-10%.[49] The incidence re-

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45. Conyers, Hall, Lang, Op.Cit. p. 535

46. Tanner, N.C. "The Surgical Treatment of Peptic Ulcer," British Journal of Surgery, Vol. 15, 1964, p. 51

47. West, Op. Cit., p. 103

48. Wells, and MacPhee, Op. Cit. p. 510

ported by American authors is much less (.99% - .1%)[50,51] than the British incidence.

Treatment of the "afferent loop syndrome" is surgical. A number of different procedures to relieve chronic bilious vomiting have been devised and applied with varying results. It is generally agreed that any attempt at correction in the chronic state should be delayed for at least eighteen months after the onset of symptoms.[52] Symptoms have been observed to diminish in severity and even disappear without treatment in some cases.[53]

Conversion of a gastrojejunostomy to a gastroduodenostomy is one of the simplest and most effective procedures used for the correction of bilious vomiting.[54] This serves the dual purpose of eliminating stasis due to kinking of transitory compromise of the stoma and minimizing the possibility of alkaline reflux to the gastric mucosa. This procedure has also proven useful in the treatment of the "dumping syndrome". Ulceration at the site of re-anastomosis is the major complication of this conversion if vagotomy is not added. The extent of gastric resection also influences the incidence of peptic ulceration.[55]

Other procedures have been introduced with good success. A procedure which has received wide attention is conversion to a "Y" anastomosis after the method of Roux (Roux-en-Y procedure).[56] Complete relief of bilious vomiting was reported in thirty-two of forty patients with varying degrees of relief in the remaining eight

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50. Adams and Renstein, Op. Cit. p. 506
51. Jordan, Op. Cit., p. 1023
52. Capper and Welbourn, Op. Cit. P. 409
53. Wells and MacPhee, Op. Cit. p. 1192
54. Tanner, N.C., Op. Cit., 1959, p. 155
55. Woodward, M.D., Op. Cit., P. 59
56. Schofield, R. "Treatment of Post-Gastrectomy Bilious Vomiting." Lancet Vol. 1, 1954, p. 519

reported by Tanner.[57] This procedure involves transection of the afferent loop and performing a re-anastomosis into the efferent loop in a circular fashion, with the distal end of the transected loop. The proximal portion of the afferent loop containing the duodenal stump and bile and pancreatic ducts is anastomosed in "Y" fashion to the jejunum at a point distal to the first anastomotic site. It is recommended that the site at which the bile enters the small intestine be at least fifty cm. below the gastrojejunostomy. With this procedure, complete cure has been reported in five patients by Bartlett and Burrington.[58] Prevention of alkaline secretion reflux into the gastric remnant is the prime objective.

This procedure is recommended only in those patients who are not prone to recurrent ulceration of the stoma secondary to the shunting of the alkaline material away from the gastrojejunostomy. (i.e. those with vagotomy or near total gastric resection.)

A modification of the above technique has been used with excellent results in a small series of thirteen patients by Wells and Johnson.[59] The afferent loop is transected near the stomach and the distal end closed. The proximal end is then anastomosed at least 25 cm. below the stomach into the jejunum by a "Y" type of anastomosis. This procedure involves only one jejuno-jejuno anastomosis and the circular loop of jejunum under the gastro-jejunal stoma is not created. It is still open to question which of the modifications described is superior, due to the limited numbers in both series.

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57. Tanner, N.C., Op. Cit., 1959, p. 157

58. Bartlett and Burrington, Op. Cit. p. 39

59. Wells and Johnson, Op. Cit. p. 482

In the past, techniques were devised for treatment of bilious vomiting which relieved obstruction only, without preventing alkaline reflux into the gastric mucosa. In cases in which obstructive processes were the initiating problem, improvement was achieved, though recurrence of the problem was apt to occur. The "pantaloon" or "double barreled" jejunal anastomosis of Steinberg was of value in treatment of bilious vomiting.[60] The procedure involves creating a jejuno-jejuno anastomosis just below the gastrojejunal stoma. The resulting configuration resembles the old-fashioned "pantaloon" undergarment.[61] This procedure, while limiting the possibilities of mechanical obstruction and serves to increase reservoir capacity. Reflux of the alkaline secretions into the gastric remnant is still a problem here and the operation is not often used to treat bilious vomiting today.

Other operations that have been used and largely discarded include suspension of the afferent loop to surrounding structures, excision of the greater omentum in such a way as to prevent kinking or extrinsic pressure and buttressing the afferent loop to the gastric pouch with sutures. More radical procedures involving jejunal or colonic transection and the interposing of a segment of the jejunum or colon between the gastric remnant and duodenum have been attempted. These procedures obviously involve a great deal more injury to the patient and their resultant high mortality and morbidity have largely precluded their use.[62]

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60. Tanner, N.C., Op. Cit., p. 149

61. Steinberg, M.D. "A Double Jejunal Lumen Gastro-jejunal Anastomosis." Journal of Obstetrics and Gynecology, Vol 88, 1949, p. 255

62. Capper and Welbourn, Op. Cit., p. 409

At the present time then, it would seem that the two operations used for treatment of post-gastrectomy bilious vomiting are the Roux-en-Y anastomosis[63] and conversion to gastroduodenostomy.[64]

Acute obstruction of the afferent loop presents another facet in the study of post-gastrectomy syndromes. This presents as an abdominal emergency and diagnosis may be quite difficult.[65] There is a sudden, severe, onset of a cramping, colicky pain which may radiate to the back. Often, symptoms begin following a meal. Retching of the gastric and jejunal efferent loop contents follows. The abdomen becomes rigid; there may be a palpable epigastric mass. Fever, leukocytosis and a rising serum amylase are also seen. Radiologic exam may reveal diminished filling of the afferent loop or no filling at all. In either case, radiologic findings are not really diagnostic.[66]

The differential diagnosis, in this situation, includes acute appendicitis, acute cholecystitis and coronary occlusion. Perforated peptic ulcer, intestinal obstruction, mesenteric thrombosis, acute pancreatitis, are difficult to distinguish from afferent loop obstruction of the acute type because they will produce a rise in serum amylase also. Acute pancreatitis is most often mis-diagnosed as afferent loop obstruction in the post-gastrectomy patient. In this case, serum amylase levels in serial may be useful. A high amylase which continues to rise is characteristic of obstruction, while an initially high level which falls

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63. Bartlett and Burrington, Op. Cit. p. 39.
64. Ibid., p. 410
65. Stammers, F.A.R., "Small Bowel Obstruction Following Antecolic Gastrectomy." British Journal of Surgery, Vol. 20, 1952, p. 59
66. Herrington, J.L. "The Afferent Loop Syndrome: Additional Experience with it's Surgical Management." The American Surgeon Vol 34, 1968, p. 321

slowly indicates pancreatitis.[66]

Treatment is immediate laparotomy in a patient with typical history and symptoms, even though the diagnosis may not be made pre-operatively on the basis of observations and laboratory findings.[68] If operation is delayed, death from shock often occurs within forty-eight hours. On occasion, the blind loop may rupture through the duodenal stump with subsequent peritonitis and gram negative septic shock. In Adam's series, all patients who survived the obstructive and operative episodes were operated within twelve hours after the onset of symptoms.[69] The procedure is aimed at relief of acute obstruction by whatever means necessary.

Incidence of acute afferent loop obstruction following is reportedly small. Two different series showed incidences of 1.0%[70] and 2.3%.[71] The incidence is much higher in antecolic as opposed to retrocolic anastomoses.[72]

In summary, afferent loop stasis or bilious vomiting in the post-gastrectomy patient is an infrequent but potentially serious problem following gastrojejunal anastomosis. It is a clinical entity, distinct from the other post-gastrectomy syndromes. Originally, the symptoms were thought to be caused by simple stasis of pancreatic, duodenal and bile secretions in the afferent loop secondary to mechanical blockage at the anastomosis site. Recent investigations have shown

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66. Herrington, J.L. "The Afferent Loop Syndrome: Additional Experience with Its Surgical Management." The American Surgeon Vol. 34, 1968, p. 321.
68. Levin, J.M. "The Afferent Loop Syndrome." The American Surgeon Vol. 34, 1966, p. 393.
69. Adams and Renstein, Op. Cit. p. 510
70. Ibid, p. 507
71. Mimpriss, T.W. and Birt, J.M.C. "Results of Partial Gastrectomy for Peptic Ulcer Disease." British Medical Journal, Vol. 2, 1948, p. 1095
72. Adams and Renstein, Op. Cit., p. 510

mechanical causes of stasis are not always present. A more acceptable explanation is the reflux of alkaline secretions into the gastric remnant and resultant gastritis. It is conceivable that both factors may be present in any given case. Surgical management is aimed at the relief of any obstruction and prevention of alkaline secretions entering the gastric remnant.

BIBLIOGRAPHY

Adams, D.D., Renstein, M.D. "Acute Obstruction of the Afferent Loop Following Billroth II Gastrectomy" The American Surgeon, 31:503-10 Aug., 1965

Bartlett, M.K., Burrington, J.D. "Bilious Vomiting After Gastric Surgery." Archives of Surgery July, 1968, pp. 35-39

Butler, T.J., Capper, W.M. "A Clinical Study of Post-Gastrectomy Syndromes" British Medical Journal, 2:265, 1951

Capper, W.M., Welbourn, J., "Early Post-Cibal Symptoms Following Gastrectomy" British Journal of Surgery 43-406, 1955-56

Conyers, J.H., Hall, R.R., Luing, D., Pulvertaft, C.N., Willson-Pepper, J.K., "The Use and Risks of Roux-En-Y Anastomosis in Post-Gastrectomy Syndromes" British Journal of Surgery, 47-533, 1959

Davenport, H.W., "Physiology of the Digestive Tract" Chicago: Year Book Medical Publishers, 1966, pp. 117-18

Guyton, A.C. Textbook of Medical Physiology Philadelphia: W.B. Saunders Company, 1966, p. 882

Harrison, T.R. Principles of Internal Medicine New York: McGraw-Hill Book Company 1966, p. 984

Herrington, J.L. "Experiences With the Surgical Management of the Afferent Loop Syndrome." Annals of Surgery, 164: 797-809, Dec. 1964

Herrington, J.L., "The Afferent Loop Syndrome: Additional Experience With It's Surgical Management." The American Surgeon, 34-321, May 1968

Hertz, A.F. "The Cause and Treatment of Certain Unfavorable Effects of Gastrectomy." Annals of Surgery, 58-466, 1913

Hibner, R., Richards, V. "Stomal or Small Bowel Obstruction Following Partial Gastrectomy." American Journal of Surgery 96-309, 1958

Hoffman, W.A., Spiro, H.M., "Afferent Loop Problems." Gastroenterology, 40:201, 1966

Jordan, G.L. Jr., "The Afferent Loop Syndrome." Surgery 38:1027, 1955

Levin, J.M., "The Afferent Loop Syndrome" The American Surgeon, 34: 391, June, 1966

Luke, N.C., "The Aftermath of Gastrectomy." British Medical Journal, 1:285, 1948

McNally, R.W., "Problems With Duodenal Stump in Gastric Resections." Surgery, 12:207-215, 1942

Mimpriss, T.W., Birt, J.M.C., "Results of Partial Gastrectomy for Peptic Ulcer Disease." British Medical Journal, 2:1095, 1948

Muir, A. "Postgastrectomy Syndromes, British Journal of Surgery, 37-165, 1949-50

Pulvertaft, C.N. "Post-Gastrectomy Bilious Vomiting." Lancet 1:225, 1952

Schofield, R., "Treatment of Post-Gastrectomy Bilious Vomiting." Lancet 1:519, 1954

Simon, M.M. "Obstruction of the Proximal Jejunal Loop Following Gastrectomy." American Journal of Surgery 91: 423, 1956

Stammers, F.A.R., "Small Bowel Obstruction Following Antecolic Gastrectomy." British Journal of Surgery 20:58, 1957

Steinberg, M.E., "A Double Jejunal Lumen Gastrojejunal Anastomosis." Journal of Obstetrics and Gynecology, 88: 253, 1949

Tanner, N.C., "Operations for Post-Gastrectomy Symptoms." Gastroenterologia, 92: 146-156, 1959

Tanner, N.C., "The Surgical Treatment of Peptic Ulcer." British Journal of Surgery, 51: 5-23, 1964

Toye, D.K.M., Williams, J.A. "Post-Gastrectomy Bile Vomiting." Lancet, Vol. II, Sept. 11, 1965, P. 525

Wells, C., Johnson, J.H. "Revision to the Roux-En-Y Anastomosis for Post-Gastrectomy Syndromes." Lancet, 2:479, 1956

Wells, C.A., MacPhee, I.W., "The Afferent Loop Syndrome." Lancet Dec. 20, 1952, p. 1189-1193

Wells, C., Welbourn, R., "Post-Gastrectomy Syndromes," British Medical Journal, March 17, 1951, 1:546

West, J.P., "Obstruction of the Proximal Jejunum Following Gastric Resection and Antecolic Anastomosis." Surgery, 37: 98, 1953

Woodward, M.D., The PostGastrectomy Syndromes, Springfield, Ill: Charles C. Thomas Publishers, 1963, p. 56