Nutritional Consequences of Surgical Resection of the Gastrointestinal Tract for Cancer

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Summary

The nutritional sequelae of major resection of the "hollow organs" of the gastrointestinal tract for cancer are reviewed. Radical surgery of the head and neck region may lead to significant nutritional problems due to the mechanical effects of surgery that limit normal methods for nutritional intake, but these problems can be overcome by tube feeding. Resection of the thoracic esophagus or stomach for cancer may produce varied forms of malabsorption, particularly of ingested fat, but the reduction of caloric intake that often will accompany these procedures plays the primary role in each instance where malnutrition is produced by the operation. These nutritional difficulties are corrected by assuring adequate caloric intake and reducing the symptoms that inhibit this. Small bowel resection rarely produces nutritional problems unless the resection is massive, a situation wherein malabsorption becomes a major problem in nutritional management. Fortunately, massive small bowel resection is rarely indicated for the treatment of cancer. Radical colon surgery of any extent is well tolerated from the nutritional standpoint. An understanding of the causes of malnutrition in patients undergoing surgical resection of the gastrointestinal tract for cancer should lead to effective management of any problems that do occur.

The most common form of cancer in the United States is cancer of the gastrointestinal tract, and surgical resection is, currently, the primary approach to patient management. Since the gastrointestinal tube is our major source of nutrients under normal circumstances, a nutritional impact resulting from these operative manipulations is unexpected. This report is not meant to be an exhaustive review of the literature on this subject, but an attempt will be made to summarize observations that have been made from a clinical perspective. These nutritional sequelae of resection are summarized in Table 1.

Resection of Head and Neck Cancer

Radical resection of cancer arising from the oral cavity and pharynx is often required in subjects who present with preexisting nutritional difficulties. Prior to resection, there are varying degrees of disability from restrictions in both mastication and deglutition. Surgical resection of these cancers often produces the same disabilities or increases the preoperative deficiency if nutritional intake has been hampered by these intake problems.

Since the nutritional sequelae of the resection of anatomical structures in the head and neck are on a mechanical rather than a metabolic basis, the clinical solutions are likewise mechanical ones. For short-term nutritional maintenance, both before and after operation, the patient can be fed by a nasopharyngeal or nasogastric tube. A frequent lack of emphasis on preoperative nutritional support is certainly a partial explanation for the postoperative wound problems that often plague the postoperative period. Adequate alimentation can be accomplished for both the replacement of preexisting nutritional defects, and postoperative maintenance, by using appropriately prepared high calorie liquid diets. For patients requiring long-term tube feeding due to permanent intake disability or protracted reconstructive procedures in the post-resection interval, a cervical tube esophagostomy is often preferable to the nasal tube. This procedure is easily accomplished either at the completion of the resection or at a later time.

Resection of Cancer of the Thoracic Esophagus

Careful appraisal of patients undergoing total or partial resection of the thoracic esophagus for cancer led to the observation that patients had varying degrees of clinical steatorrhea and diarrhea following these procedures. Detailed metabolic studies of a series of patients by Shils and Gilat (66) led to clear-cut evidence of laboratory steatorrhea in all patients when quantitative stool fat excretion measurements were made (Table 2) but carbohydrate, nitrogen, vitamin B12, and electrolyte absorption measurements were virtually normal. These and other studies (52, 54, 64) demonstrated data similar to those obtained in patients subjected to truncal vagotomy and a gastric drainage procedure, although the effect on fat absorption was generally greater in patients undergoing thoracic esophageal resection. These reports are convincing in regard to the major role played by vagotomy in the production of the observed malabsorption and the diarrhea, but the mechanisms are not completely clear.

Attempts to correct both this mild diarrhea and malabsorption after esophagectomy by feeding pancreatic and biliary supplements or the use of a gluten-free diet were unsuccessful. The substitution of medium-chain triglycerides for the longer-chain fatty acids did reduce fecal fat excretion in patients with malabsorption (64). Another potential solution to this metabolic defect observed in some patients after partial or total resection of the thoracic esophagus might well be preservation of the vagus innerva-
tion, if this were feasible. When the resection is for cancer, however, this approach is untenable, as the resection must include the vagus nerves, for obvious anatomical reasons.

To put the nutritional problem of esophagectomy in perspective, it should be stressed that the degree of postoperative nutritional disability observed is quite limited and is of greater physiological interest than of practical importance. The fecal fat losses that may occur can be easily compensated for by a modest increase in caloric intake and/or the exchange of medium-chain triglycerides for long-chain fatty acids in the diet.

Gastrectomy for Cancer

Nutritional disability is more frequently observed after either partial or total gastrectomy for cancer than after esophagectomy. This impairment includes some metabolic defects due to malabsorption, as well as a host of symptom problems that may limit total intake. The normal function of the stomach is to serve as a reservoir to receive and retain food, alter the food to some degree by digestive processes, and slowly discharge the altered nutriments into the small intestine at a rate that is the most efficient for the digestive and absorptive functions of the gut. It is not surprising that major mechanical alterations in the size of the stomach, or its emptying function, may have a major effect on nutritional capabilities of the patient. The limited number of patients who have nutritional problems following gastrectomy is probably even more surprising. Resection of the stomach for cancer may require partial or total resection, but the extent of resection is usually greater than that performed for benign disease, and the nutritional problems observed after gastric resection generally increase proportionately with the extent of the resection that is performed.

Malabsorption. There is an extensive literature documenting absorptive defects following gastrectomy, particularly total gastrectomy. Absorption of one of the major nutrients, carbohydrate, is not quantitatively impaired by gastrectomy, and an increase in fecal excretion of the nitrogen, as a measure of protein malabsorption, is responsible for little if any nutritional impairment in the gastrectomized patient (17, 45, 61, 74). The primary defect in absorption that may play some role in chronic difficulties with weight maintenance and nutrition in the postgastrectomy patient is impairment of fat absorption (8, 38, 45, 50, 56, 62, 67, 70, 74). Associated problems in absorption of iron and vitamins may occur also.

Fat Absorption (Chart 1). There are 2 aspects of the steatorrhea problem that must be kept in mind from a practical clinical standpoint. The 1st is the relatively poor correlation noted between the ability of the patient to maintain body weight after gastrectomy and the degree of steatorrhea that has been observed on metabolic balance studies (38, 45, 56). Although these quantitative laboratory studies demonstrate minor degrees of steatorrhea after partial gastrectomy, many patients, after partial resection, have fat excretion levels in the normal range (73). Mean values for fat absorption after total gastrectomy (approximately 80% of ingested fat) are well below the normal range (92 to 100%), but there is much variation between individual patients, and these data bear little relationship to weight maintenance (38, 45) (Chart 2). The 2nd factor that should be appreciated is that the degree of steatorrhea noted in these gastrointestinal patients is mild, compared with that noted after massive small bowel resection, and that of patients with sprue syndromes or who are subjected to major pancreatic resection. Although the steatorrhea observed after total gastrectomy is of great interest from both the etiological and physiological viewpoints, the role of this steatorrhea in nutritional disability after gastrectomy appears to be a relatively minor one.

Nutritional consequences of "radical" resection

<table>
<thead>
<tr>
<th>Organs resected</th>
<th>Nutritional sequellae</th>
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<tbody>
<tr>
<td>Oral cavity and pharynx</td>
<td>Dependency on tube feedings</td>
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<tr>
<td>Thoracic esophagus</td>
<td>Gastric stasis (secondary to vagotomy)</td>
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<tr>
<td></td>
<td>Fat malabsorption</td>
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<td>Gastrostomy feedings in patients without reconstruction</td>
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<tr>
<td>Stomach</td>
<td>Dumping syndrome</td>
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<td></td>
<td>Fat absorption</td>
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<tr>
<td>Small intestine</td>
<td>Pancreatobiliary deficiency with fat malabsorption</td>
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<tr>
<td>Duodenum</td>
<td>Decrease in efficiency of absorption (general)</td>
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<tr>
<td>Jejunum</td>
<td>Vitamin B12 and bile salt malabsorption</td>
</tr>
<tr>
<td>Ileum</td>
<td>Fat malabsorption and diarrhea; vitamin B12 malabsorption; gastric hypersecretion</td>
</tr>
<tr>
<td>Massive (&gt;75%)</td>
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<tr>
<td>Colon (total or subtotal)</td>
<td>Water and electrolyte loss</td>
</tr>
</tbody>
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Fat malabsorption after esophagectomy (distal or near total)

<table>
<thead>
<tr>
<th>Range of fat absorption (%)</th>
<th>Mean (%)</th>
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<tbody>
<tr>
<td>Before</td>
<td>92-98</td>
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<td>After</td>
<td>65-84</td>
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<tr>
<td></td>
<td>96</td>
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<td></td>
<td>74</td>
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Chart 1. Fat absorption after total gastrectomy [from Lawrence et al. (45)].

Chart 2. Fat malabsorption after esophagectomy (distal or near total)
Data on factors affecting steatorrhea in the gastrectomized patient are of some interest from the standpoint of both the etiology of this defect in absorption and its possible correction. One mechanism might be a decrease in digestive enzymes normally initiated by gastric acid. In this regard, it is of interest that hydrochloric acid supplements prior to meals in totally gastrectomized patients produced a significant improvement in the measured fat absorption in these same patients (70). This effect of hydrochloric acid might be interpreted as a secretin stimulus prior to the arrival of food stuff in the small bowel, thereby alleviating the "pancreatico-cibal asynchrony" that is produced by the anatomical loss of the stomach. A decrease in fat absorption that has been observed with an increase in carbohydrate intake in the diet of the gastrectomized patient (70) correlates well with this concept of insufficient pancreatic and biliary secretion in these patients (Chart 3). Hypertonic glucose feedings in dogs also suppress pancreatic secretion (41). It is difficult to correlate the degree of the steatorrhea with bowel hypermotility, but the increase in fecal fat during high carbohydrate intake suggests a significant role for motility, since this dietary alteration does increase "intestinal hurry."

A study of factors that do not effect fat absorption in the gastrectomized patient are of practical importance in clinical management. Dietary adjuvants such as pancreatin, Tween 80, and lipase failed to show a significant affect on steatorrhea (63), nor did variations in anatomic reconstruction of the intestinal tract after total gastric resection (8). On the other hand, all of the measurable fecal fat losses after gastrectomy could be easily balanced by a small increase in fat intake due to the interesting observation that the percentage absorption of fat remains constant over a wide range of intake (58, 62) (Chart 4). These observations support the concept that difficulty in maintenance of body weight and malnutrition after gastrectomy is due to fat absorption to only a minimal degree. Other factors affecting total intake are of greater importance.

**Protein Absorption and Metabolism (Chart 5).** Since optimal nutrition and maintenance of lean tissue mass are partially governed by absorption of protein, as well as the utilization of this foodstuff, the metabolism of nitrogen in gastrectomized patients is of great interest. Although the fecal excretion of nitrogen is at a slightly higher level than normal in patients after extensive gastrectomy (45, 61, 74), this malabsorption proved to be less striking than that noted for fat. Whether the increase in nitrogen requirement for equilibrium in totally gastrectomized patients, compared with normal patients, is due only to diminution of intestinal absorption of nitrogen, or to some unexplained increase in metabolic requirements, was evaluated by comparisons of fecal nitrogen excretion at several levels of nitrogen intake. These metabolic data revealed no increase in nitrogen excretion in postgastrectomy patients that could not be attributed to the small excess of fecal nitrogen alone (45). This appears to rule out any increased metabolic requirements for nitrogen in this group of patients. Since the absorptive defect is relatively small, a slight increase in protein intake can easily offset any defect in absorption that does occur.

**Caloric Intake.** The relatively minor defects in absorption described have proved of great interest to investigators. However, it is clear from a number of observations that a limitation of total caloric intake after gastrectomy is the major reason for observed instances of nutritional depletion.
and poor weight maintenance (7, 38, 45, 58). This is supported by the metabolic data presented, the poor correlation between the absorption data and weight maintenance in patients, careful dietary histories in gastrectomized patients who do have nutritional difficulties, and the excellent correlation between the state of nutrition and the presence or absence of disturbing postprandial symptoms that limit intake.

Other Metabolic Deficiencies Associated with Gastrectomy. Some vitamin and mineral deficiencies may result from radical gastrectomy for cancer. These include malabsorption of iron, vitamin B12, and calcium. The stores of vitamin B12 are slowly depleted after total gastrectomy, but the development of megaloblastic anemia is delayed due to the large store normally present in the liver that delays the appearance of the clinical effects of vitamin B12 deficiency (22). This delay in development of megaloblastic anemia may extend from 6 months to 4 years, and both the anemia and the typical neurological symptoms respond effectively to parenteral vitamin B12 administration. Probably all patients surviving total gastrectomy for more than 2 years should receive prophylactic B12. It is of interest that folic acid deficiency has been identified as a cause of megaloblastic anemia after gastrectomy as insufficient vitamin B12 (48). In these patients, the intake of folic acid alone will prevent the anemia resulting from vitamin B12 deficiency after gastrectomy as well, but it will not control the neurological problems from insufficient vitamin B12 (48). In these patients, the intake of iron is often low, and this compounds the mild defect of iron absorption. Absorption of iron may be impaired after gastrectomy, although the presence of acid and the presence of the duodenum are not essential to iron absorption (7, 21, 27, 68, 71). Iron deficiency anemia has been demonstrated to be a problem in approximately 50% of patients undergoing total gastrectomy, but it is easily overcome by increased intake.

Inadequate absorption of vitamin D after gastrectomy due to steatorrhea may contribute to osteomalacia at a later time in some patients (13, 16) but the metabolism of calcium, phosphorus, magnesium, and potassium generally does not differ from that of normal persons (62). Other vitamins, particularly vitamins B and A, may be less efficiently absorbed after gastrectomy, but deficiency of these vitamins can be prevented by oral supplements (2).

Postprandial Symptoms and Nutrition. Postprandial symptoms after radical gastrectomy undoubtedly play the major role in malnutrition by producing a self-limited intake of foodstuff (9, 18, 58). Despite some variation in their manifestations, the most frequent symptom complaints are usually described as “dumping syndrome” (12, 26). It is now generally accepted that this group of symptoms is the sequela to the loss of pyloric function, although a more extensive resection of the stomach is more often the offender than limited gastric resection. The incidence of this disturbing problem varies somewhat, depending on the interest of the observer, since so much of the symptomatology is subjective. After subtotal gastric resection, these dumping symptoms in the early postprandial period probably occur in 10 to 25% of patients, and the incidence is greater than this after total gastrectomy. Less common postgastrectomy symptoms affecting dietary intake are the so-called “afferent loop syndrome” (35) and reflux gastritis. The symptoms of the dumping syndrome include epigastric fullness, hyperperistalsis, borborygmi, and cramps with occasional nausea, vomiting, or diarrhea. Many of these symptoms are adequately described by the term, intestinal hurry. In addition of these abdominal complaints, a more striking series of postprandial symptoms suggesting sympathetic discharge are also observed. These include unpleasant warmth, tachycardia, sweating, weakness, and dizziness, and are accompanied by electrocardiographic findings and measurable alterations in cardiac output and regional blood flows (49, 50, 60). All of these early symptoms are most marked approximately 15 min after ingestion of foodstuff in the symptomatic gastrectomized patient. This complex of symptoms can occur with all degrees of severity, and symptoms in some patients are elicited only by an abnormal challenge with hypertonic foodstuff. Others experience disabling symptoms with most of their normal meals. This individual susceptibility can even be detected in the patient who has not had a gastrectomy by instilling test meals of hypertonic foodstuff into the small intestine through an intestinal tube (29). There is a relatively close correlation between postgastrectomy malnutrition and the severity of these symptoms, since reducing total dietary intake is one solution the patient finds effective for controlling these unpleasant symptoms.

For many years, various mechanical explanations were given for these symptoms, but none were well substantiated. Machella (47) established the fact that a large volume shift into the bowel occurred after intraluminal introduction of a hyperosmolar meal (Chart 6). This observation correlates quite well with the fact that it is a uniform observation in gastrectomized patients that high-carbohydrate meals, which are more likely to become hyperosmolar, intensify symptoms (36, 46, 51). It has been shown experimentally that this large shift of fluid into the gut occurring 15 to 30 min after ingestion of test meals is associated with a water loss from the plasma as well as electrocardiogram and cardiac output alterations and a redistribution of blood flow (43, 44, 50, 60). This redistribution affects the renal blood flow, the digital blood flow, and flow in the intestinal circuit (28, 49).
Subsequent investigations support the release of a humoral agent or agents in response to the hyperosmolar meals, and this appears to be a reasonable explanation for both the autonomic symptomatology and these experimental findings (28, 34). It has been suggested that the primary humoral agent producing these associated symptoms and physiological changes is serotonin (41, 32, 53), and some relief of the symptoms has been achieved by pharmacological means on this basis (34). Although the release of other vasoactive polypeptides from small bowel mucosa undoubtedly occurs in response to the hyperosmolar challenge, the practical means for dealing with this symptom problem are dietary alterations and the attention to methods of reconstruction after gastrectomy that will minimize these physiological responses. From the nutritional standpoint, the key to maintaining adequate nutritional state after gastrectomy is the diminution of this symptom complex in those patients who are the most susceptible.

Management of Malnutrition Observed after Gastrectomy. Since these accumulated data demonstrate that the control of postprandial symptoms after gastrectomy is the major factor in adequate weight maintenance, dietary alterations to minimize symptoms have proven to be the primary therapeutic approach (1, 58, 60). A high-protein, high-fat, low-carbohydrate, frequent-feeding diet has achieved this objective in most patients by eliminating the primary symptom challenge (the high-carbohydrate insult to the small bowel), by reducing the sudden influx of foodstuff in large quantity into the small bowel (by using small frequent feedings), and by providing adequate calories in as small a volume as possible (high-fat diet). Not only is there higher caloric value in fat/g of food stuff, but both protein and fat have the additional advantage of slower enzymatic breakdown in the bowel than carbohydrate, thereby avoiding the rapid development of a hyperosmolar solution in the bowel lumen. Other measures include reclining after meals and the maintenance of a normal blood volume, since the described postprandial shifts in blood flow are more symptomatic in patients with contracted blood volumes. An increase in iron intake and vitamin supplements, particularly vitamin B₁₂, are also important in the dietary management of these patients.

In spite of dietary measures, some patients continue to have disabling postprandial symptoms after radical gastrectomy and thereby suffer secondary nutritional deficiencies. Remedial operations have been designed to counteract these symptoms by either creating a reservoir to substitute for the missing stomach (25, 33, 42, 75) or transposing intestinal segments with or without a restriction to outflow to retard the rapid intestinal transit of foodstuff that serves to accentuate the offending symptomatology (3, 4, 23, 24, 57). There is a wide range of such procedures that have proved applicable to a number of differing anatomical situations present after radical subtotal gastrectomy or total gastrectomy (25, 59). These procedures are of some value in well-selected patients that are not suitably managed by the dietary means described. These anatomical alterations are particularly relevant to the patient who has had total gastrectomy, but experience in recent years with the construction of a substitute pouch at the time of primary resection has markedly reduced need for these secondary operations. It is impossible to construct a substitute stomach after gastrectomy that equals the function of the original organ, but these methods frequently can produce a functional system that allows symptoms to be controlled adequately by the dietary alterations described. Body weight is usually stabilized at a level that is lower than the preoperative weight, but the overall nutritional status is satisfactory in most patients.

Intestinal Resection for Cancer

In contrast to the more proximal portions of the gut, major resection of the intestinal tract distal to the pylorus is more often required for noncancerous disorders, but the nutritional sequela of intestinal resection are directly related to the extent of resection in each instance.

Small Intestine. The effect of small-bowel resection is related to the individual functions of the various segments, as indicated in Table 1. The nutritional sequela of duodenal resection relate more to the anatomical alteration of the pancreatic and biliary secretions than to the mechanical and absorptive functions of the duodenum itself (for a detailed discussion, see Ref. 65). Clinical studies in normal subjects and those with ablation of portions of the jejunum indicate that all of the various nutrients, with the exception of vitamin B₁₂, are most efficiently absorbed in this segment of the intestine, but the reserve absorptive capacity of the ileum can accommodate for any functional change produced by loss of the more proximal bowel (5, 6, 15, 69, 76). The distal ileum is responsible for absorption of vitamin B₁₂ and conjugated bowel salts, as well as serving as a back-up absorptive site for the nutrients normally absorbed in the jejunum (Chart 7). The adaptability of various segments of the small intestine to increase their absorptive capabilities prevents major clinical problems after small bowel resection, except for those patients who have massive bowel resection in the range of 75% of the total small bowel. The fecal fat losses can be much greater in this circumstance.
require resection of more than 2 ft of ileum and ileocecal valve, there may be a measurable increase in fecal fat, but this can be controlled quite easily with a high-protein low-fat diet (37, 55). Vitamin B₁₂ supplements are required to correct the deficiency in this vitamin that is produced by resection of this bowel segment. Resection up to 8 ft of the jejunum fails to interfere with the absorption of glucose, fat, protein, folic acid, vitamin B₁₂, or other vitamins, but resection of both the jejunum and ileum is accompanied by an increase in absorptive problems from the standpoint of all nutrients, with the severity of these problems depending on the extent of the resection. Calcium supplements and vitamin D may be required, also, with these more extensive small bowel resections, with their resulting fecal losses. The diarrhea and the increased fecal calcium, magnesium, and other electrolyte losses occurring in some patients with massive resection may be reduced by reducing the intake of long-chain fatty acids in the diet. Medium-chain triglycerides have proven to be a useful approach but, when used in significant amounts, the development of ketosis may lead to the need for supplementary sodium bicarbonate. It is often necessary to supplement the p.o. feedings with i.v. hyperalimentation in the early postoperative period to avoid cachexia at this critical stage. Another approach to the diminution of diarrhea in patients with massive bowel resection is the administration of cholestyramine (30), since this will chelate the increased volume of bile salts reaching the large bowel secondary to their reduced absorption after resection of the terminal ileum.

The nutritional management of the patient with massive small-bowel resection can be quite challenging for the clinician but, fortunately, this is an uncommon problem for patients undergoing gastrointestinal surgery for cancer. Blind-Loop Syndrome. There are a number of conditions following surgery of the small intestine for cancer which may give rise to this symptom complex. These include blind loops of the small bowel resulting from atypical intestinal anastomosis, stricture of the intestine, or other mechanical reasons for blind-pouch formation, all of which have in common intestinal stasis and subsequent intraluminal infection. This symptom complex is characterized by diarrhea, steatorrhea, anemia, loss of weight, and multiple vitamin deficiencies (1, 14, 39). The actual frequency of this blind-loop syndrome is uncommon, since the surgeon carrying out a small-bowel resection ordinarily avoids any form of short circuit or blind-pouch formation, but identification of these phenomena in the affected patient is usually delayed. The diagnosis is often suspected from the hematological aspects of the problem, particularly vitamin B₁₂ deficiency, but there may be steatorrhea and symptoms of other vitamin deficiencies as well. This phenomenon has engendered much interest from the standpoint of mechanism, but it is clear that the principal cause is the abnormal bacterial growth that occurs in these unusual anatomical situations. Treatment obviously requires appropriate antibiotic therapy and subsequent operative correction of the underlying mechanical cause of the stasis in the small bowel.

Surgical Correction of Nutritional Deficiencies after Massive Small Bowel Resection. A mechanical approach to the management of patients with massive small bowel resection is the operative transposition of a short antiperi-
otalic segment of bowel to replace the function of the absent ileocecal valve. This is not generally utilized early in the clinical course after resection, due to the marked adaptation that may occur in some patients and concern regarding the possible loss of absorptive surface by operation in a patient who already has a reduced intestinal length. This is an approach that is used after several months in unusual circumstances in which the ileocecal valve has been resected along with extensive, small bowel resection, and clinical nutritional management is incapable of dealing with the problem (76).

Colon

Operations for cancer requiring resection of the ileocecal valve, ileum, and right colon rarely produce nutritional deficiency, unless the extent of ileal resection is large, and this is not the usual circumstance for patients with colon cancer. Major resection of the left colon is well tolerated also, even with extensive resection, and nutritional deficiencies do not occur as a result of the resection itself. However, subtotal resection of the colon may produce significant water and electrolyte loss due to diarrhea if the ileum is anastomosed to the proximal rectum. A similar situation occurs in the patient with total colectomy that requires an ileostomy, as this may be followed by large water and electrolyte losses in the early postoperative period. Fortunately, these fluid losses decrease quite rapidly soon after surgical resection in both circumstances, and these patients fail to develop nutritional problems of clinical significance.

References


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