Carol Rees Parrish, R.D., M.S., Series Editor

# Dumping Syndrome

Andrew Ukleja

**Dumping syndrome is a constellation of gastrointestinal and vasomotor symptoms resulting from changes in the anatomy and physiology of the stomach created by gastric surgery. Dumping syndrome is frequently attributed to the rapid emptying of gastric content into the small bowel. However, the etiology of dumping syndrome is multifactorial. Severe dumping can be complicated by malnutrition and it can be associated with poor quality of life. Most patients with dumping syndrome can be treated conservatively with dietary modifications. Octreotide is the most effective drug therapy for patients with incapacitating symptoms. Those patients who failed medical therapy may be considered as surgical candidates. The aim of this article is to review the clinical features and pathophysiology of dumping syndrome in addition to providing guidelines for its management.**

## INTRODUCTION

Operations on the stomach can lead to a variety of undesirable and chronic sequelea. The dumping syndrome refers to gastrointestinal (GI) and vasomotor symptoms that occur following ingestion of a meal in individuals after gastric surgery. The association between postprandial symptoms and rapid drainage of the stomach after gastroenterostomy was first described by Hertz in 1913 (1). The term "dumping" was introduced by Andrews and Mix in 1920, who reported a radiographic observation of rapid gastric emptying of contrast in patients with typical dumping symptoms after gastrectomy (2).

Andrew Ukleja, M.D., Assistant Professor of Medicine, Department of Gastroenterology, Cleveland Clinic, Weston, Florida.

The incidence and severity of the symptoms associated with dumping correlate with the type of gastric surgery. Dumping occurs in approximately 15%–20% of patients after partial gastrectomy (3). Significant dumping has been reported in 6%–14% of patients who have undergone truncal vagotomy with drainage. A lower incidence of dumping has been observed after proximal gastric vagotomy without drainage procedure. After Roux-en-Y gastric bypass, 50% to 70% of patients experience dumping syndrome in the early post-operative period (4). However, symptoms of dumping subside after 15–18 months from gastric bypass. In children, dumping syndrome has been reported almost exclusively after fundoplication (5). Only a minority (1%–5%) of patients with dumping syndrome suffer from severe, disabling symptoms.

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## CLINICAL FEATURES OF DUMPING SYNDROME

The clinical manifestations of dumping include GI and vasomotor symptoms. Dumping syndrome can be divided into early and late dumping depending on the relation of symptoms to the time elapsed after a meal (Table 1). The severity of symptoms varies between individuals. Symptoms of early dumping occur within 10–30 minutes after meals. They result from accelerated gastric emptying of hyperosmolar content into the duodenum or small bowel, followed by fluid shifts from the intravascular compartment into the intestinal lumen. This leads to small bowel distention and increased intestine contractility, both, believed to be responsible for GI symptoms such as nausea, bloating, abdominal cramps, and explosive diarrhea (6). The majority of patients have early dumping and they suffer from both GI and vasomotor symptoms.

Late dumping occurs 1–3 hours after a meal, and it is characterized predominantly by systemic vascular symptoms including flushing, dizziness, palpitations, and an intense desire to lie down. Physical exam of these patients may reveal profound orthostatic changes including drop in blood pressure and increased heart rate. Late dumping occurs in approximately 25% of patients with dumping syndrome. Those patients with late dumping have mostly vasomotor symptoms. Late dumping is a consequence of reactive hypoglycemia from an exaggerated release of insulin (7).

Uncontrolled severe dumping can result in sitophobia (fear of food or eating) and weight loss leading to under- and malnutrition. Weight loss of up to 30% from preoperative weight has been reported in patients with severe dumping (8).

**Table 1**
**Symptoms of the dumping syndrome**

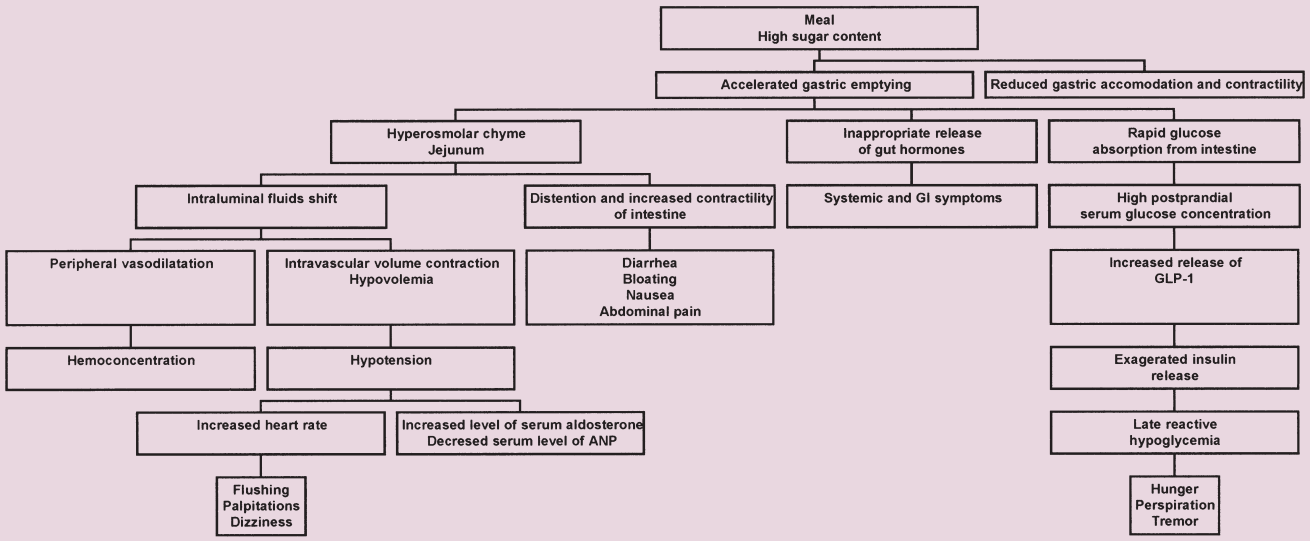
| ***Abdominal*** | ***Vasomotor/Systemic*** | |
|---|---|---|
| *Early dumping* | | *Late dumping* |
| Epigastric fullness | Diaphoresis | Difficulty with concentration |
| Nausea | Desire to lie down | Decreased consciousness |
| Diarrhea | Headache | Hunger |
| Vomiting | Flushing | Perspiration |
| Abdominal cramps | Fatigue | Tremor |
| Borborygmi | Lightheadedness | |
| Bloating | Pallor | |
| | Palpitations | |
| | Syncope | |

## PATHOPHYSIOLOGY OF DUMPING SYNDROME

The pathogenesis of a dumping syndrome is poorly understood and it is likely to be multifactorial (Table 2). The alterations of gastric anatomy by surgery, including resection or bypass of the pylorus, and interference with gastric innervation, have a profound effect on the rate of gastric emptying. The accommodation and the cyclic contractility of the stomach in response to distention are abolished after partial gastrectomy, allowing immediate dumping of gastric contents into the jejunum (9). This accelerated gastric emptying of liquids is a critical step in the pathogenesis of dumping syndrome. Rapid delivery of large amounts of hyperosmolar chyme into the upper small intestine leads to bowel distention and intestinal hypermotility. However, no difference in rate of gastric emptying was found between patients with and without dumping symptoms after surgery in a few studies suggesting other mechanisms may be involved in the etiology of dumping (10).

Gastric emptying is under control by fundic tone, antropyloric mechanism, and duodenal feedback, regulated by the enteric nervous system and circulating GI hormones. Relative intravascular volume contraction and hemoconcentration occur as a consequence of osmotic shift of fluids from the intravascular compartment into the gut lumen. Rapid heart rate, elevated hematocrit and drop in plasma volume have been observed in patients in response to oral hyperosmolar glucose with early dumping (11). This leads to release of vasoactive GI hormones responsible for peripheral and splanchnic vasodilatation and vasomotor symptoms such as flushing, tachycardia and dizziness. Hinshaw, et al first reported peripheral vasodilatation in patients with dumping syndrome, despite a volume-contracted state (12). However, vasodilatation has not been

**Table 2**
**Mechanisms responsible for ealy and late dumping**




confirmed by other investigators (13). Low plasma levels of atrial natriuretic peptide (ANP) and elevated levels of aldosterone, activation of the rennin-aldosterone axis, have been reported in early dumping in response to hypovolemia (14).

A role of hormones in the etiology of the syndrome has been confirmed in animal study by induction of dumping symptoms in a healthy dog after a blood transfusion from portal vein of another dog with dumping syndrome (15). Higher postprandial levels of gut hormones such as pancreatic polypeptide, enteroglucagon, peptide YY (PYY), vasoactive intestinal polypeptide (VIP), neurotensin and glucagon-like peptide (GLP) have been documented in patients with a dumping syndrome (16,17). Neurotensin, VIP, and PYY delay motility of the upper GI tract and reduce gastric and intestinal secretions. In response to rapid delivery of a meal to the small intestine, high concentration of carbohydrates is seen in the proximal small bowel followed by rapid absorption of glucose into the circulation. Hyperglycemia stimulates rapid insulin secretion followed by reactive hypoglycemia. Glucose-dependent insulinotropic peptide and GLP-1 produced in the small bowel and colon, respectively, are believed to be mediators of late dumping (18). Exaggerated release of GLP-1 induces hyperinsulinemic response and subsequent late hypoglycemia (19). However, it is not clear why only some patients develop dumping symptom while others are asymptomatic after surgery.

## THE DIAGNOSTIC DILEMMA

Dumping syndrome is diagnosed based on constellation of characteristic symptoms after meals in a patient who has undergone gastric surgery or by a dumping provocation test. Laboratory studies are rarely helpful in establishing the diagnosis. In severely malnourished patients, anemia and hypoalbuminemia may be found.

A diagnostic scoring system has been developed by Sigstad (20) (Table 3). The score index is very helpful in assessing a response to therapy. It is based on weighing factors assigned to symptoms of dumping. A score index higher than 7 points is suggestive of dumping syndrome.

Oral glucose provocation and hydrogen breath tests are useful when the diagnosis is in doubt. Symptoms of early dumping can be elicited by an oral glucose challenge. A rise in heart rate by 10 beats per minute or more in the first hour after an oral glucose challenge (with 50 g of glucose), following 10-hour fasting is diagnostic. This test was found to be highly sensitive and specific, 100% and 92%, respectively

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**Table 3**
**Dumping symptoms according to the Sigstad's scoring system**

| | |
|---|---|
| Shock | +5 |
| Fainting, syncope, unconsciousness | +4 |
| Desire to lie or sit down | +4 |
| Breathlessness, dyspnea | +3 |
| Weakness, exhaustion | +3 |
| Sleepiness, drowsiness, apathy, falling asleep | +3 |
| Palpitation | +3 |
| Restlessness | +2 |
| Dizziness | +2 |
| Headaches | +1 |
| Feeling of warmth, sweating, pallor, clammy skin | +1 |
| Nausea | +1 |
| Abdominal fullness, meteorism | +1 |
| Borborygmus | +1 |
| Eructation | –1 |
| Vomiting | –4 |

(21). A challenge test with higher amounts of glucose should be avoided, because it can provoke symptoms of dumping in non-dumpers. A positive hydrogen breath test after glucose ingestion has been reported to be 100% sensitive for early dumping. A diagnosis of late dumping can often be confirmed by frequent blood sampling after provocation with oral glucose. In response to this test, elevated plasma levels of glucose during the first 60 minutes and reduced plasma glucose levels 1–2 hours later are expected. However, induction of symptoms after glucose provocation is more accurate for diagnosis of late dumping. Evaluation of the upper GI tract anatomy and function is an important step in confirmation of dumping because symptoms can mimic other postgastrectomy syndromes. Gastric emptying scintigraphy can reveal rapid rate of gastric emptying. An upper endoscopy or barium study can help to exclude ulcer or obstruction.

## MANAGEMENT

### Diet

Dietary modifications are the mainstay of therapy in dumping syndrome. Fluid intake during meals should be restricted. Patients should be instructed to avoid liquids for at least 30 minutes after a solid meal. Daily food intake should be divided into at least six meals. Carbohydrate intake should be reduced, with preference for complex, rather than simple carbohydrates (Table 4). Milk and dairy products are not well tolerated and they should be avoided (22). Increased intake of protein and fat is recommended to meet daily energy needs. Supplementation of dietary fibers (bran, citrucel) has been shown to be beneficial in the treatment of late hypoglycemia. Pectins, guar gum, and glucomannan are effective by delaying glucose absorption and prolongation of small bowel transit time (23).

Most patients with mild symptoms respond well to dietary changes. Therefore, a proper patient education about dietary restrictions is very important. In cases of severe vasomotor symptoms, lying supine for 30 minutes after meals may reduce the chance of syncope by slowing the rate of gastric emptying and improving venous return.

### Drug Therapy

Medical therapy plays an important role in patients who fail dietary modifications. Several drugs have been described as beneficial in symptom control, without consistent success, in small studies and case reports, including tolbutamide, propranolol, cyproheptadine, methysergide, and verapamil (Table 5). Two other drugs, acarbose and octreotide, have been studied more extensively and will be discussed in more details.

### Acarbose

Acarbose is a potent competitive inhibitor of alpha-glycoside hydrolase, which interferes with carbohydrate absorption. Efficacy of acarbose in late dumping is related to delayed carbohydrate digestion by slowing conversion of starch and sucrose to monosaccharides and blunting the postprandial rise of serum glucose and insulin (24). Acarbose at a dose of 50 mg has been shown to reduce symptoms of postprandial hypoglycemia in patients after gastric surgery (25). A complete disappearance of late symptoms, palpitation and dizziness, has been reported with acarbose (50/100 mg t.i.d) in patients with dumping and non-insulin dependant diabetes mellitus (26). In contrast, Lyons, et al

**Table 4**
**Dietary modifications in dumping syndrome**

| *Preferred Carbohydrate Foods* | *Simple Carbohydrates to Avoid* |
|---|---|
| Unsweetened cereals | Sweetened cereals |
| Bread, pasta | Sweet rolls |
| Rice, potatoes | Pancakes with syrup |
| Crackers | |
| Fresh fruit | Canned fruit in heavy syrup |
| Unsweetened frozen fruit | Sweetened juice |
| | Candied fruit |
| Plain yogurt | Milkshakes |
| Skim milk | Sweetened yogurt |
| Sugar-free pudding/candies | Cakes, ice cream, honey, jelly |
| Sugar free beverages (coffee, tea) | Sweetened drinks (regular soda) |

Patient education materials on dumping diet are available on the website of University of Pittsburgh Medical Center at http://patienteducation.upmc.com or, from the University of Virginia Health Center of Excellence at http://www.healthsystem.virginia.edu/internet/digestive-health/nutrition/patientedu.cfm

showed negative outcome with acarbose in subjects with dumping syndrome (27). Despite attenuation of hyperglycemia and reduced rise in plasma insulin with acarbose at 50 mg, no statistically significant improvement was seen in the dumping score, including those patients who continued a longer trial. Use of acarbose may be limited by the occurrence of diarrhea and flatulence secondary to fermentation of unabsorbed monosaccharides, but its adverse effects subside over time. Its role in the therapy of dumping has yet to be clarified.

**Table 5**
**Drugs used in reduction of dumping symptoms**

| *Drug* | *Dose* | *Effect* |
|---|---|---|
| Tolbutamide (38) | 0.25–0.75 g, t.i.d. | Subjective improvement |
| Propranolol (39) | 10 mg, q.i.d. | Reduced early dumping |
| Cyproheptadine (40) | 4–8 mg, t.i.d. | Preventing vasomotor symptoms |
| Methysergide maleate (41) | 4–8 mg, b.i.d. | Reduced vasomotor symptoms |
| Verapamil (42) | 120–240 mg, q.d | Reduced vasomotor symptoms |
| Acarbose (26) | 50–100 mg, t.i.d | Reduced late dumping |
| Octreotide (43) | 25–100 mcg, t.i.d | Reduced vasomotor symptoms |

## Octreotide

The beneficial role of somatostatin and its synthetic analogue octreotide (Sandostatin) in the treatment of dumping has been well established. Octreotide has a strong inhibitory effect on the release of insulin and several gut-derived hormones. It prevents late hypoglycemia by delaying the maximal rise in plasma glucose level and by reducing peak insulin concentration (28). The other beneficial mechanisms of action in dumping include slowing the rate of gastric emptying and small intestine transit time, inhibition of postprandial vasodilation and splanchnic vasoconstriction, and increase in intestinal absorption of water and sodium (29). Octreotide has been shown to decrease the Sigstad's index score, pulse rate, and plasma insulin levels, and to minimize changes in orthostatic blood pressure, packed cell volume and plasma osmolarity in subjects with dumping when compared to placebo (30). The effectiveness of octreotide in ameliorating symptoms of dumping is summarized in Table 6. The initial recommended dose of octreotide is 25–50 µg administered subcutaneously, 2–3 times daily, 15–30 minutes before meals. The dose can be increased to 100–200 µg if the smaller dose is not effective.

In short-term studies relief of symptoms has been reported in near 100% of patients. Octreotide has been shown to maintain its efficacy long-term in patients with refractory dumping (31). Improvement in quality of life has been seen with long-term treatment with some patients able to return to work. In the largest study up to date, Vecht, et al reported long-term outcome of octreotide

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**Table 6**
**Randomized controlled trials of octreotide in patients with severe dumping**

| *Author (%)* | *Number of Patients* | *Dose (µg)* | *Efficacy of therapy (%)* | |
|---|---|---|---|---|
| **Short-term** | | | | |
| Hopman (44) | 12 | 50 | 100 | |
| Tulassay (45) | 8 | 50 | 100 | |
| Primrose (46) | 10 | 50–100 | 90 | |
| Richards (47) | 6 | 100 | 100 | |
| Geer (31) | 10 | 100 | 100 | |
| Gray (48) | 9 | 50 | 100 | |
| Hasler (49) | 8 | 50 | 100 | |
| **Long-term** | | *Dose/frequency* | | *Duration of therapy (months)* |
| Geer (31) | 10 | 100, t.i.d | 90 | 3–15 |
| Primrose (50) | 5 | 50, b.i.d | 50 | 48 |
| Mackie (51) | 14 | 50, b.i.d | 75 | 3 |
| Vecht (32) | 20 | 25–100, t.i.d | 55 | 37 |

b.i.d., ×2 daily, t.i.d., ×3 daily

therapy at doses of 50–200 µg/day in 20 patients with severe dumping and mean follow-up of 3 years (32). The initial relief of symptoms was achieved in all subjects with further symptom control in 80% of patients after three months of therapy. Treatment was discontinued due to lack of improvement or side effects. Major adverse effects of octreotide therapy, painful injections and severe diarrhea, are infrequent, but can result in discontinuation of treatment. Significant steatorrhea has been found with octreotide use. An early morning diarrhea or steatorrhea associated with long-term therapy can be controlled with an extra dose of octreotide before bedtime or pancreatic enzyme replacement.

A long acting form of octreotide is available and its use may improve compliance with treatment and reduce its side effects. In a recent study, the efficacy of depot long-acting release octreotide, Sandostatin LAR (Novartis Pharmaceuticals, East Hanover, NJ) intramuscular (i.m.) injection was compared to its subcutaneous form (s.c.) in twelve patients with severe dumping (33). In this open study octreotide, s.c., was switched to Sandostatin LAR 10 mg i.m. injection every 4 weeks for 6 months. Sandostatin-LAR was found to be as effective as octreotide in ameliorating dumping symptoms and it was more effective than octreotide in increasing body weight and improving quality of life. In summary, therapy with octreotide is safe and it should be offered to patients with severe dumping after other medical treatments have failed.

## SURGERY

Conservative management is always preferred because most patients may expect improvement in dumping symptoms over time. It is suggested to follow medical measures, including diet, and behavioral and drug therapy for at least one year before consideration of a corrective surgery.

Several surgical procedures have been developed to abate the symptoms of dumping with the goal of most of them to slow down gastric emptying (Table 7). A proper selection of the surgical intervention is very important. Pyloric reconstruction is a first choice surgery in patients with severe dumping after pyloroplasty. A stomal revision has been abandoned because of a higher risk of complications especially stomal stricture. Roux-en-Y reconstruction is a preferred cura-

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**Table 7**
**Surgical procedures to correct dumping syndrome**

| *Procedure* | *Mechanism* | *Complications* |
|---|---|---|
| Stoma revision | Narrowing of the gastrojejunal stoma | Stomal strictures<br>Gastric outlet obstruction (*abandoned) |
| Pyloric reconstruction | Modification of pyloroplasty by cutting the pyloroplasty incision and its longitudinal closure | A low-risk procedure |
| Conversion of Billroth II to Billroth I anastomosis | Restoration of physiologic delivery of the meal to the duodenum | A low-risk procedure |
| Jejunal Interposition | Creation of a long iso- or antiperistaltic limb between stomach and jejunum | Ulceration and stenosis of the interposed segment |
| Roux limb conversion to Roux-en-Y gastrojejunostomy | Slowing rate of gastric emptying and chyme transit via the Roux limb | Roux stasis |
| Intestinal retrograde electrical pacing | Experimental procedure (*No human studies performed) | |

tive operation for patients with Billroth I and Billroth II gastrectomies. A favorable outcome has been reported in 85%–90% of patients with Billroth I and II gastrectomies after this surgical conversion (34).

In patients with Billroth II gastrectomy, a conversion to Billroth I anatomy allows restoration of the gastric content delivery into the duodenum. Symptomatic improvement in dumping with this type of surgery has been reported in up to 75% of patients (35). For those patients who already had a Roux-en-Y reconstruction, an antiperistaltic jejunal loop interposition should be considered. Excellent results with an interposed jejunal segment has been reported (36). The efficacy of jejunal interposition is related to slowing of gastric emptying by the creation of a long iso- or antiperistaltic limb between the stomach and jejunum. The length of an interposed segment has influence on the surgical outcome.

Overall, surgery has a very limited role in the treatment of dumping symptoms and may not always be curative. Therefore, it is most important to prevent development of dumping syndrome by selecting a gastric procedure associated with less dumping symptoms and minimal impairment of gastric emptying. A proximal gastric vagotomy is a preferred surgery for the management of refractory peptic ulcer disease despite higher rate of recurrent ulcer (37). Gastric resection is also preferable to a Roux-en-Y gastrojejunostomy because of the lower rate of dumping when compared to pyloroplasty or loop gastrojejunostomy.

## SUMMARY

Dumping syndrome is a common complication and important to recognize after gastric surgery. The diagnosis of dumping is based on clinical presentation or a glucose provocation test in difficult cases. Severe dumping can be associated with considerable morbidity. A differentiation from other postgastrectomy syndromes is critical to initiate an appropriate therapy. The majority of patients respond well to dietary modifications. Proper education of the patient is a key to assure compliance with the diet. A trial of acarbose and octreotide should be given as an effective alternative before consideration of a surgical intervention. ■

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