Reinfusion of Intestinal Secretions: A Viable Option for Select Patients

Fifteen to 20% of patients with pancreatitis will go on to develop a severe or complicated course requiring nutrition support. A small percentage of these patients will have an ongoing gastric outlet obstruction from duodenal inflammation and compression requiring gastric decompression; a few might even need an external biliary drain for a time. Enteral jejunal feeding has been demonstrated to be superior to parenteral nutrition in this patient population. For those patients with pseudocyst development needing extended nutrition support (>30 days), enteral nutrition can be achieved by way of jejunal access as nasojejunal, PEG/J, or separate PEG and surgical jejunostomy tubes. This article focuses on the hydration aspects of these patients and shares one institution’s experience of reinfusing gastric and/or pancreatobiliary secretions to maintain absorption, hydration and electrolyte status in the patient’s own home environment.

CASE

A 57-year-old female was admitted with nausea and vomiting due to gastric outlet obstruction as a result of duodenal compression from pancreatitis. A large pseudocyst was noted on CT exam. She was to undergo ERCP for common bile duct stone removal with placement of percutaneous endoscopic gastrostomy with a jejunal arm (PEG/J) for nutrition support while the pseudocyst resolved. She was 5’7” and weighed 118#, down from her usual body weight of 145#. She was considered severely malnourished and at refeeding risk. After her PEG/J placement she tolerated tube feeding well and was discharged home 4 days later on a 1.5 cal/mL, non-fiber containing enteral product, full strength, at 100mL per hour over 12 hours at night via her j-port. She initially required venting of her gastric secretions, but was able to clamp her g-port prior to discharge. She was readmitted 3 weeks later with the following laboratory results:

- Na—107 mmol/L
- K—1.9 mmol/L
- Cl—74 mmol/L
- CO₂—34 mmol/L
- BUN—11 mg/dl
- Cr—0.5 mg/dl
- Glucose—133 mg/dl

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The patient had been placed on a liquid proton pump inhibitor prior to discharge, but did not take it regularly after she returned home. In addition, the patient began venting off her gastric secretions due to nausea and occasional vomiting once she was at home. When queried by the GI nurse coordinator, her husband reported venting and discarding, “maybe 1–2 cups of gastric juice every day.” (Nutrition Editors note: This reminds me of Bill Cosby’s, “I know what I mean when I say oops, but what do you mean when you say oops?!”—Not to generalize, but I have noticed over the years that men have a different concept of what “a cup” is—it usually runs anywhere from 10–32 oz. I keep kitchen measuring cups in our GI nutrition clinic so patients/caregivers can show me what they mean by the portions that they eat or drink). The patient was, in fact, venting off 1–2, 16 oz. “cups” (480–960 mL) of fluid from her gastric port each day with occasional vomiting also.

She ultimately required a percutaneous external biliary drainage tube due to an infected gallbladder and the surgeons wanted to wait until she was better nourished before she went to the OR for a cholecystectomy. Her biliary drainage tube was putting out an average of 600 mL/day.

INTRODUCTION

Pancreatitis is not only a very painful process; it can also significantly ravage one’s nutritional status (see Table 1).

Nutrition support is needed in approximately 15–20% of patients enduring a severe course of pancreatitis. Enteral nutrition (EN) support has been clearly shown to be superior to parenteral nutrition (PN) with reductions in mortality, multiple organ failure, systemic infections and the need for surgical interventions (2,3), with the majority favoring the nasojejunal route. If the patient has ongoing emesis, gastric decompression will be required to relieve the patient of nausea and vomiting. Vigorous fluid resuscitation is the mainstay of supportive therapy in acute pancreatitis to prevent or minimize pancreatic necrosis (4). Hyperglycemia, if not well-controlled, can not only thwart efforts at nutrition support, but also accelerate dehydration. In the hospital because of problems caused by excess fluid loss from gastric decompression.

Our case demonstrates the metabolic complications that can arise when patients have excessive loss

<table>
<thead>
<tr>
<th>Table 1</th>
<th>Factors Associated with Nutritional Deficits in Pancreatitis</th>
</tr>
</thead>
<tbody>
<tr>
<td>• Functional</td>
<td>- Inability to eat as a result of pain with food ingestion</td>
</tr>
<tr>
<td>• Functional</td>
<td>- Gastric outlet obstruction due to duodenal compression from swollen pancreas</td>
</tr>
<tr>
<td>• Functional</td>
<td>- Anatomical changes if surgery required (Whipple, etc.)</td>
</tr>
<tr>
<td>• Iatrogenic</td>
<td>- NPO for procedures/surgery</td>
</tr>
<tr>
<td>• Iatrogenic</td>
<td>- Hospital Food</td>
</tr>
<tr>
<td>• Iatrogenic</td>
<td>- Therapeutic hospital diets (insult to injury)</td>
</tr>
<tr>
<td>• Endocrine</td>
<td>- Poorly controlled hyperglycemia</td>
</tr>
<tr>
<td>• Exocrine</td>
<td>- Pancreatic insufficiency</td>
</tr>
<tr>
<td>• Exocrine</td>
<td>- Malabsorption</td>
</tr>
<tr>
<td>• Exocrine</td>
<td>- Steatorrhea</td>
</tr>
<tr>
<td>• Exocrine</td>
<td>- Fat soluble vitamin loss</td>
</tr>
<tr>
<td>• Exocrine</td>
<td>- Osteoporosis</td>
</tr>
<tr>
<td>• Exocrine</td>
<td>- Decrease in bicarbonate secretion</td>
</tr>
<tr>
<td>• Exocrine</td>
<td>- Decrease normal pancreatic enzyme function due to inadequate rise in pH</td>
</tr>
<tr>
<td>• Exocrine</td>
<td>- Decrease efficacy of exogenous enzymes—enteric coated needs bicarbonate to be released</td>
</tr>
<tr>
<td>• Exocrine</td>
<td>- Decrease efficacy of bile salts due to more acidic environment</td>
</tr>
<tr>
<td>• Medication Induced</td>
<td>- Acid reduction and Narcotics</td>
</tr>
<tr>
<td>• Medication Induced</td>
<td>- Predispose to small bowel bacterial overgrowth</td>
</tr>
<tr>
<td>• Medication Induced</td>
<td>- Constipation</td>
</tr>
</tbody>
</table>

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<table>
<thead>
<tr>
<th>Table 2</th>
<th>Secretion of Fluid within the Gastrointestinal Tract</th>
</tr>
</thead>
<tbody>
<tr>
<td>Source</td>
<td>mL/24 hours</td>
</tr>
<tr>
<td>Saliva</td>
<td>500–1500</td>
</tr>
<tr>
<td>Stomach</td>
<td>2000–3000</td>
</tr>
<tr>
<td>Bile</td>
<td>1000</td>
</tr>
<tr>
<td>Pancreas</td>
<td>1000</td>
</tr>
<tr>
<td>Intestine</td>
<td>1000</td>
</tr>
</tbody>
</table>

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Reinfusion of Intestinal Secretions

of upper GI tract secretions that are not adequately replaced. With the placement of a percutaneous endoscopic gastrostomy with jejunal arm (PEG/J), malabsorption can be averted, medication delivery can be preserved, and patients can maintain their hydration and nutritional needs without the use and risks of a central line. Successful jejunal feeding in this patient population has been described elsewhere (5,6). Devising an effective plan that allows these patients to return to the comfort of their home is the focus of this article. While the article presents our experience with patients with severe pancreatitis complicated by pseudocysts, the same process can be used for patients with refractory gastroparesis, obstructing GI lesions or external biliary drains.

THE VALUE OF UPPER GUT SECRETIONS

Role of Gastric Secretions

Normal endogenous secretions produced daily above the pylorus include both salivary and gastric secretions and amount to 2500–4000 mL per day (see Table 2). These secretions as well as the others listed in Table 2 are essentially recycled in the GI tract and contribute to the overall hydration status of the individual.

Saliva and gastric secretions are stimulated by the cephalic phase of eating—that is, just thinking about eating as well as taking the first bite of food. In addition to initiating protein digestion in the stomach, gastric secretions contain significant acid and are front line protection in terms of its bacteriocidal action. If significant gastric secretions are lost, then dehydration and hypochloremic metabolic alkalosis can result from excessive loss of acid, chloride and fluid (see

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**Table 3.**

<table>
<thead>
<tr>
<th>Body Fluid</th>
<th>Na</th>
<th>K</th>
<th>HCO₃⁻</th>
<th>H</th>
<th>Cl</th>
<th>pH</th>
</tr>
</thead>
<tbody>
<tr>
<td>Sweat</td>
<td>30–50</td>
<td>5</td>
<td>—</td>
<td>45–55</td>
<td>—</td>
<td>2</td>
</tr>
<tr>
<td>Saliva</td>
<td>45</td>
<td>20</td>
<td>60</td>
<td>44</td>
<td>7</td>
<td>2</td>
</tr>
<tr>
<td>Gastric</td>
<td>40–65</td>
<td>10</td>
<td>90</td>
<td>100–140</td>
<td>2</td>
<td></td>
</tr>
<tr>
<td>Pancreas</td>
<td>135–155</td>
<td>5</td>
<td>70–90</td>
<td>55–75</td>
<td>8</td>
<td></td>
</tr>
<tr>
<td>Bile</td>
<td>135–155</td>
<td>5</td>
<td>35–50</td>
<td>80–110</td>
<td>7</td>
<td></td>
</tr>
<tr>
<td>Jejunum/Ileostomy</td>
<td>100–120</td>
<td>10</td>
<td>50–70</td>
<td>50–60</td>
<td>7</td>
<td></td>
</tr>
<tr>
<td>Diarrhea</td>
<td>25–50</td>
<td>35–60</td>
<td>30–45</td>
<td>20–40</td>
<td>—</td>
<td></td>
</tr>
<tr>
<td>Normal stool</td>
<td>5</td>
<td>10</td>
<td>—</td>
<td>10</td>
<td>8</td>
<td></td>
</tr>
</tbody>
</table>

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**Table 4.**

<table>
<thead>
<tr>
<th>IV Fluid</th>
<th>Na</th>
<th>K</th>
<th>Chloride</th>
</tr>
</thead>
<tbody>
<tr>
<td>0.9 NS*</td>
<td>154</td>
<td>0</td>
<td>154</td>
</tr>
<tr>
<td>0.45 NS</td>
<td>77</td>
<td>0</td>
<td>77</td>
</tr>
<tr>
<td>Lactated Ringers (LR)</td>
<td>130</td>
<td>4</td>
<td>109</td>
</tr>
<tr>
<td>D₂W</td>
<td>0</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>D₂W 0.45 NS</td>
<td>77</td>
<td>0</td>
<td>77</td>
</tr>
</tbody>
</table>

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**Table 5.**

**Causes of Pancreato-biliary Insufficiency or Deficiency**

**Potential causes of Pancreatic enzyme deficiency or insufficiency**

- Pancreatic cancers
- Severe acute or chronic pancreatitis
- Cystic fibrosis
- External loss of pancreatic secretions due to distal duodenal tumor compression or enteroctaneous fistula
- Gastric acid dumped into the upper gut can inhibit lipases activity as well as decreased bicarbonate secretion from the pancreas, both result in a lowering of the luminal pH thereby affecting enzyme function.

**Potential causes of bile salt deficiency or insufficiency**

- Primary biliary cirrhosis
- Primary sclerosing cholangitis
- Cirrhosis
- Cholestatic processes
- External biliary drains
- Gastric hypersecretion
- Small bowel bacterial overgrowth
- Obstruction distal to the common bile duct requiring decompression above the site
- Disruption in enterohepatic circulation of bile salts due to resection of terminal ileum or small bowel enteroctaneous fistula

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Hypochloremic Metabolic Alkalosis

Metabolic alkalosis is a relatively common clinical problem that is often induced by the loss of chloride-rich gastric secretions due to vomiting or nasogastric suctioning. With vomiting or nasogastric suctioning, loss of acid initiates the alkalosis, but volume contraction along with hypochloremia and an increase in aldosterone secretion contributes to the reduction in bicarbonate excretion in the kidney by increasing proximal tubule reabsorption of bicarbonate and increasing hydrogen ion secretion in the distal tubule (aldosterone effect) (9). Hypokalemia is common in this setting also and further aggravates the alkalosis by enhancing bicarbonate absorption. Many patients are asymptomatic, or, if they have symptoms (weakness, muscle cramps, postural dizziness), it is typically a result of the hypovolemia that accompanies the excess loss of gastric fluid. Sodium loss can also be significant, and patients can be admitted with life-threatening hyponatremia. Intravenous saline corrects both volume contraction, hypochloremia and the alkalosis. In our case, homeostasis was achieved by collecting and reinfusing the vented gastric secretions via the jejunal tube.

Role of Pancreateo-biliary Secretions

Pancreatic enzymes and bile salts work in tandem to efficiently and effectively digest macronutrients, especially fat, in preparation for absorption across the brush border. Alterations in either one of these important digestive agents can significantly alter absorption. Pancreateo-biliary secretion deficiency, impairment,

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or exclusion can arise under several conditions (see Table 5).

**Pancreatic Secretions.** Pancreatic exocrine insufficiency occurs in 35–86% of patients with severe pancreatitis (5). It should be noted that diarrhea does not always accompany steatorrhea, particularly in the patient requiring narcotics for pain management. Bicarbonate secretion from the pancreas provides the ideal environment for pancreatic enzymes to function most effectively; a damaged pancreas may not secrete adequate bicarbonate, further aggravating pancreatic enzyme dysfunction and malabsorption.

Pancreatic insufficiency can be very effectively treated with pancreatic enzyme supplementation. In the enterally fed patient, low fat elemental or semi-elemental products can be used, however it is important to note that some of the semi-elemental products have considerable fat, despite a large percentage of medium chain triglycerides, and pancreatic enzymes may still be needed. Although this institution commonly used standard enteral formulas, new cost analysis and the loss of Viokase powder from the market, has made us reevaluate our practice (see Table 6 with new cost data of enteral products with and without pancreatic enzymes). Although enteric coated enzymes are not to be crushed, at least one institution has been doing this for several years (see Table 7 for their practice). Clinical progress with whatever plan will be most telling—if nutrient absorption is in question, the plan will need to be reevaluated.

**Bile Salts.** Bile salts are not only important for digestion and absorption of fat, but also of fat soluble vitamins. The bile salt pool is maintained via the enterohepatic circulation in the last 100 cm of ileum, such that 95% is recycled daily. Loss of bile salts for any reason decreases fat absorption up to 50% (10). There are no commercially available bile salt replacers, hence the only recourse the clinician has is to decrease the fat content in the diet or enteral formula, unless the patient’s bile salt loss is due to an external drain and this can be collected and reinfused in a feeding port below.

**Reinfusion Gastrointestinal Secretions**

Venting gastrostomies have been used successfully in both palliative situations as well as in aggressive treatment of disease processes as an alternative to salem sump type nasogastric tubes (5,11–15). Salem sump type tubes routinely used for gastric or intestinal decompression are very uncomfortable, and are associated with nasal necrosis, esophageal and gastric erosions. In most circumstances, patients use the venting port to relieve nausea, vomiting, abdominal distension and gas. Some patients are able to take some liquids by mouth and vent only as needed; while others require NPO status as part of the treatment of the disease process. Regardless of the circumstance, adequate hydration, nutrition and medication delivery of the patient will require attention from the clinician depending on the total volume lost in a 24 hour period.

While no prospective studies exist supporting the practice of reinfusing intestinal secretions, many case reports are available in the literature. Procedures or devices to preserve biliary secretions, in particular, have been accomplished using PEGs (16–18), percutaneous endoscopic duodenostomy (PED) (19), external transhepatic biliary drainage tubes connected to PEG/J or PEG/D tubes (20–23), gastric secretions or external

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**Table 7. Another Institution’s Practice of Using Creon with Enteral Feeding**

For Creon capsules:
- Gastrically-fed pts: minimicrospheres are whole in a slurry of mildly thickened fruit juice down the feeding tube.
- Postpylorically-fed, we crush and activate the minimicrospheres using bicarbonate of soda. Use one “Creon 10” capsule or two “Creon 5000” capsules to one Sodibic capsule (840 mg sodium bicarbonate) (made by Aventis or Aspencare) + 10 mL warm water or 1 teaspoon of sodium bicarbonate (baking soda).
- **Procedure:** use a mortar and pestle to mix 10 mL warm tap water with the contents of 1 Sodibic capsule.
- Empty the Creon capsules into this mixture and crush well.
- Add a little more lukewarm water as needed.
- This mixture is then instilled into the feeding tube with a 10 mL syringe, and given every 2 hours or so.

Submitted by: Suzie Ferrie, AdvAPD, CNSC
Critical Care Dietitian, Royal Prince Alfred Hospital, Australia
## Table 8.
Cases of Reinfusion of Gastric or Biliary Secretions

<table>
<thead>
<tr>
<th>Study</th>
<th>Population</th>
<th>N</th>
<th>Access for Reinfusion</th>
<th>Comments</th>
</tr>
</thead>
<tbody>
<tr>
<td>Cheong, 1993</td>
<td>Cholangio CA; one w/lung mets</td>
<td>2</td>
<td>PTC drainage tube to j-port of PEG/J^</td>
<td>Pt 1 survived 3.8 months; 2 bouts of sepsis; Pt 2, length of survival not reported</td>
</tr>
<tr>
<td>Decher, 2009</td>
<td>Pancreatic CA</td>
<td>1</td>
<td>PEG/J^ &amp; external biliary drain</td>
<td>Bile collected and bolused into j-port.</td>
</tr>
<tr>
<td>Foutch, 1989</td>
<td>Unresectable pancreatic CA</td>
<td>2</td>
<td>PTBD* to PEG</td>
<td>Pt 1 survived 4 weeks; Pt 2 survived &gt;4 months</td>
</tr>
<tr>
<td>Friman, 1989</td>
<td>CBD stones</td>
<td>1</td>
<td>T-tube collection and replaced via nasogastric tube</td>
<td>Temporary replacement of bile via NGT; volume &amp; tolerance not reported; pt discharged after 4 weeks</td>
</tr>
<tr>
<td>Lenthall, 1970</td>
<td>Cirrhosis</td>
<td>7</td>
<td>Gastrostomy</td>
<td>Only 1 pt reinfused bile (up to 2 liters/day)</td>
</tr>
<tr>
<td>Levy, 1983</td>
<td>All w/ peritonitis</td>
<td>30</td>
<td>16 w/enterocutaneous fistulas w/distal mucous fistula; 14 had 1 or more temp double enterotomies</td>
<td>Intestinal losses can be decreased during the early phase of treatment; as sepsis is controlled, optimal utilization of remaining absorptive area for EN is permitted</td>
</tr>
<tr>
<td>Makola, 2006</td>
<td>Pancreatitis w/ large pancreatic pseudocysts</td>
<td>18/126</td>
<td>Venting of gastric secretions and reinfusion via j-port of PEG/J^</td>
<td>Avg of 4.4 months to resolution of pancreatitis; &gt;500 mL gastric secretions reinfused/ 24 hours</td>
</tr>
<tr>
<td>Mohandas, 1991</td>
<td>GB CA</td>
<td>1</td>
<td>PTBD* &amp; 18 Fr PEG w/ 8 Fr feeding tube placed duodenally***</td>
<td>Bile and tube feedings were both infused into 8 Fr duodenal tube</td>
</tr>
<tr>
<td>Morita, 1988</td>
<td>Pancreatic CA</td>
<td>1</td>
<td>PTBD* to PED***</td>
<td>Pt survived &gt;4 months; external drain converted to internal</td>
</tr>
<tr>
<td>Ponsky, 1982</td>
<td>Inoperable gall bladder &amp; pancreatic CA</td>
<td>2</td>
<td>PTBD* to PEG</td>
<td>Pt 1 drained 800-1000mL of bile/day; survived 4 months; Pt 2 drained 600-800 mL; absorption improved w/replacement</td>
</tr>
<tr>
<td>Rumley, 1985</td>
<td>All w/unresectable malignancies</td>
<td>12</td>
<td>Stamm gastrostomies connected to jejunostomies by T-connector</td>
<td>All pts tolerated bolus jejunal feeding of 125–300 mL q 2 hours; 8 pts needed reinfusion gastric juices—7 went home doing this; 1 pt had jejunal irritation as pH of reinfused secretions was 2.</td>
</tr>
<tr>
<td>Shike, 1989</td>
<td>Unresectable pancreatic CA</td>
<td>1</td>
<td>PTBD to PED***</td>
<td>Pt survived &gt; 5 months</td>
</tr>
<tr>
<td>Tokumo, 1997</td>
<td>Pancreatic CA</td>
<td>1</td>
<td>PEG for feeding PTBD* connected to PEG/J^</td>
<td>Nutritional status &amp; daily living activities improved</td>
</tr>
<tr>
<td>Wolfer, 1935</td>
<td>Review of jejunal feeding since 1885</td>
<td>Various case reports</td>
<td>Jejunostomy</td>
<td>Report experience w/ reintroducing 200–1600 mL of gastric aspirates daily; along w/pabulum, it improved results of feeding. All reported on cases of bile reinfusion into the jejunum.</td>
</tr>
</tbody>
</table>

*Percutaneous transhepatic biliary drain (PTBD)

**Percutaneous transhepatic cholangio-drain (PTCD)

***Percutaneous endoscopic duodenostomy (PED)

^Percutaneous endoscopic gastrostomy w/ jejunal extension (PEG/J)
biliary drainage bolused periodically into a jejunal port of PEG/J (5,24), gastrostomy with separate jejunotomy (25,26) and via nasogastric tube (27). One study even described a cohort of patients with double enterotomies where the effluent from the upper enterotomy was infused into the lower enterotomy resulting in an overall net decrease in fluid lost from the above site (28). See Table 8 for a summary of published reports.

Despite the number of case reports published, the clinician is still left with surmising how one might orchestrate the collection, followed by the reinfusion of these electrolyte, enzyme, and bile salt rich secretions.

**REINFUSING INTESTINAL SECRETIONS— ONE INSTITUTION’S EXPERIENCE AND PRACTICE**

Although reinfusing gastric secretions may seem benign to a healthcare provider, the mere concept of reinfusing intestinal secretions can be shocking to a patient. Taking the time to thoroughly explain the rationale for the intervention, answer questions and allay anxieties facilitates the reinfusion process. Similarly, asking the patient to notify you of any difficulties with the reinfusion establishes a concrete role for the patient and builds support for the procedure. Creating a partnership with the patient on this venture reduces the need for extended hospitalizations, central access or the need for periodic intravenous fluids after discharge. This procedure can be achieved with either a PEG/J, separate g and j tubes, or a nasogastric-jejunal tube (NG-J). Below describes our experience using PEG/J tubes; see Table 9 for the type of equipment used at our institution.

**Procedure**

1. All patients are started on a liquid (lansoprazole [Prevacid]) proton pump inhibitor (PPI). The PPI is administered via the jejunal port the evening the PEG-J is placed. Occasionally, a patient’s insurance will not cover the liquid PPI; in such cases, oral dosing will be tried if the patient does not require constant gastric gravity drainage or wall suction. The rationale for using liquid PPI as adjunctive therapy includes the following:

   - To reduce the total volume of gastric secretions in patients who require gastric venting (and in some, it may decrease the volume enough to obviate the need to reinfuse).
   - To increase the pH of gastric secretions (>5) reinfused into the jejunum to more closely mimic the physiologic pH that the jejunum is accustomed to and avoid jejunal injury from acid (25).
   - To reduce the likelihood of gastritis or ulceration while under physiological stress and without oral intake.

2. The maximum amount of gastric secretions discarded in a 24 hour period without disruption of fluid and electrolyte homeostasis is empirically set at 500 mL. If >500 mL of gastric secretions are
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Table 10. Pump Instructions

1. Empty the contents of the urinal into the feeding bag. If there are thick secretions at the bottom of the urinal do not pour the thicker secretions into the feeding bag. They will clog the tubing. Instead, pour the thin liquid contents and hold this small volume of thick secretions to be delivered with the 60 mL catheter tip syringe (see “syringe” below).

2. Flush the jejunal port with 30 mL of water using the 60 mL catheter tip syringe to verify tube patency.

3. Prime the feeding pump using the pump feeding bag containing the gastric secretions. Set the volume to be infused and the rate. The rate of the reinfusion depends on patient tolerance. Patients may tolerate rates significantly higher than tube feed infusion rates—as high as 400 mL/hour. However, if bloating, cramping or other symptoms develop, reduce the rate.

4. Connect the feeding bag to the jejunal port and start the feeding pump.

5. If the sight of the gastric secretions is bothersome to the patient or visitors, a pillow case, t-shirt, etc. can be hung over the bag to obscure the process.

6. If the scent of the gastric secretions is unpleasant to the patient or visitors, verify that the cap on the top of the feeding bag is tightly sealed and consider a room deodorizer to mask the odor.

7. Once the entire volume of gastric secretions is infused, flush the jejunal port vigorously with 30–60 mL of water using the 60mL catheter tip syringe to clear the tubing of residual contents to prevent tube clogging.

8. Document the volume collected from the gastric port on the output sheet and the volume infused via the jejunal port on the input sheet (see Table 13).

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collected in the drainage bag over a 24 hour period, we reinfuse all the secretions. However, none of the gastric secretions are reinfused until we know that the pH is above 5 (see #5 below). All secretions that are collected prior to obtaining a satisfactory pH reading are emptied from the drainage bag, measured, recorded and discarded. Along with water flushes, IV fluids are used to maintain adequate hydration during this transition period.

3. After PEG/J placement, begin collecting gastric secretions by placing the gastric port of the tube to gravity drainage. Use the Adult Nasogastric Lopez® Valve to connect the drainage bag to the gastric port of the feeding tube. The tapered ends of the Lopez® Valve and the rubberized blue connector create a tight seal between the gastric port and the drainage bag which eliminates leakage of the effluent. Creating a tight seal is important as gastric secretions can be very caustic if they leak onto the patient or their clothing; if this happens, they often become frustrated and will be reluctant to proceed.

4. If the patient is venting more than 500 mL/24 hours (and is unable to tolerate clamping of the g-port for short periods—i.e. patient becomes nauseated or distended), we collect the patients secretions and prepare for reinfusion every 8 hours or so as needed.

5. Prior to reinfusing, a small aliquot of gastric secretions are taken directly from the gastric port and sent to the lab for pH testing to assess the efficacy of the prescribed PPI dose. This is done the morning after the second dose of PPI has been given. If the gastric pH is <5, and we have verified that the patient actually received their PPI doses per guideline, the PPI is increased to BID dosing and the pH is rechecked again 2 days later. If the gastric pH is >5, we proceed with reinfusion. Note: It is unusual for patients to vent more than 1500 mL; further investigation is warranted in this setting. However, some patients take in more orally for quality of life purposes than can be accommodated with reinfusion. There is a volume that exceeds the number of hours in a day for patients to achieve reinfusion, deliver all of their EN, and have a life away from their pump, (quality of life is always central to our patient care).

6. The actual reinfusion may be performed via pump, gravity or syringe. The decision is based upon patient/clinician preference and availability of equipment (see Table 9). All three methods are reviewed below (see Table 10–12). The first step for all methods is to empty all of the gastric contents from the drainage bag into a dedicated 1000 mL male urinal. Using the urinal facilitates precise measurement of the volume of gastric secretions and allows for easy transfer of the gastric contents to the appropriate device for reinfusion.
Reinfusion of Intestinal Secretions

“If mock" Gastric Secretions for Reinfusion
If despite adequate preparation, the patient is not agreeable to gastric reinfusion, the patient is instructed to make their own reinfusion solution (see Table 14).

**REINFUSION OF BILIARY SECRETIONS**

Procedure
1. Begin the process of reinfusing biliary secretions promptly upon return to the unit after the insertion of the biliary drain and the PEG/J tube. Start collecting secretions right away and plan for reinfusion every 4–6 hours to coincide with their EN delivery for maximal micelle formation required for fat absorption.
2. Reinfusion may be performed via pump, gravity or syringe. The decision is based upon clinician preference and availability of equipment.
3. Instill 100–200 mL every 4 hours while the patient is receiving EN. The syringe method works well for this small amount, however, pump or gravity can also be used.
4. Regardless of the method used, the first steps for all methods include emptying all of the bile contents into the 1000 mL male urinal or similar collection device, and placing the EN on hold. Using the urinal

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### Table 11.
**Gravity Instructions**

1. See “Pump” steps 1 and 2.
2. Hang the gravity feeding bag on an IV pole and prime the gravity feeding bag using the roller clamp.
3. Connect the gravity feeding bag to the jejunal port and open the roller clamp on the feeding bag. The rate of the reinfusion depends on patient tolerance. Many patients tolerate the roller clamp fully open. However, if bloating, cramping or other symptoms develop the rate can be reduced.
4. See “Pump” steps 5–8.

*Used with permission from (1)*

### Table 12.
**Syringe Instructions**

1. See “Pump” steps 1 and 2.
2. Pull the secretions into the empty 60 mL syringe.
3. Tightly secure the syringe tip to the jejunal port and slowly instill the secretions.
4. Repeat this process until all secretions are reinfused
5. If the instillation becomes sluggish, flush the tube vigorously with 30–60 mL of water until the flow increases and continue the reinfusion process.
6. If the sight of the gastric secretions is bothersome to the patient or visitors, a pillow case can placed around the urinal to shield the contents from sight.
7. See “Pump” steps 6–8.

*Used with permission from (1)*

### Table 13.
**Sample Intake/Output Log**

<table>
<thead>
<tr>
<th>DATE/TIME</th>
<th>WEIGHT</th>
<th>URINE</th>
<th>STOOL</th>
<th>GASTRIC OUTPUT</th>
<th>JEJUNAL INPUT</th>
<th>FEEDINGS/WATER</th>
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facilitates precise measurement of the volume of biliary secretions and facilitates transfer of the contents to the appropriate device for reinfusion. Other clinicians have reported various practices—see Table 15 (personal communications with the author [CRP]).

**CONCLUSION**

Fifteen to 20% of patients with severe pancreatitis will need nutrition support; a percentage of these patients will also require ongoing gastric decompression from functional obstruction due to an inflamed pancreas. In order to prevent dehydration and electrolyte imbalances, we present an option of reinfusing gastric secretions to allow patients to return home and avoid central intravenous access or the need to return to clinic for periodic intravenous infusion. Reinfusion maintains electrolyte and fluid status without the inherent risks and expense of intravenous infusion and access; in those with loss of pancreatico-biliary secretions, it also corrects malabsorption.

### Table 15. Anecdotal Reinfusion Practices

1. Initiate EN and stop every 4 hours or so and bolus in what has been collected up to that point, then restart EN.
2. Place collected bile in a gravity bag and infuse through a y-port of the j-tube while the EN is running; if only a g and j-port, can attach a universal adaptor (#08750300,Compat®, www.nestle-nutrition.com/Products, 800-422-2752 to the j-port so both EN and reinfusion can be done concurrently.
3. Stop EN q 4–6 hours and gravity drip or pump in @ 150 mL/hr or > the biliary secretions that have been collected.
4. In some patients who still seem to malabsorb (or lose a substantial portion of their 24 hour secretions for a variety of reasons), consider using a lower fat formula.

References


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Reinfusion of Intestinal Secretions

(continued from page 38)


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Send in a brief case report. No more than one double-spaced page. One or two illustrations, up to four questions and answers and a three-quarter to one-page discussion of the case. Case to include no more than two authors. A $100.00 honorarium will be paid per publication.

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