Reinfusion of Gastrointestinal Secretions: The Bedside Experience

Amy J. Berry

Both pancreatic enzymes and bile salts are necessary for complete absorption of dietary fat. A deficiency of either results in suboptimal absorption of not only fat, but fat soluble vitamins. While pancreatic enzymes are available to replace insufficient pancreatic secretion, there is no replacement for bile salts. Unabated external loss of upper intestinal secretions can result in dehydration, acid/base imbalance, electrolyte abnormalities as well as malabsorption. This article describes the reinfusion of GI secretions, various methods of reinfusion, as well as potential and significant pitfalls when embarking on this form of treatment.

INTRODUCTION

Successful digestion and absorption of long chain fat and fat soluble vitamins require coordinated release of adequate pancreato-biliary secretions, along with ingested nutrients. Bile, produced in the liver, flows into the right and left hepatic ducts before joining the common bile duct, where it finally enters the duodenum via the sphincter of Oddi (Figure 1). Between meals, bile is shunted into the gallbladder for storage. After fat ingestion, secretin stimulates bile release from the liver, while cholecystokinin (CCK) stimulates gallbladder contraction as well as relaxation of the sphincter of Oddi. Pancreatic enzyme release is dependent on both secretin and CCK.

Approximately 1 liter each of bile and pancreatic fluid, as well as 2-3 liters of gastric secretions are produced daily. However, some conditions may prevent these digestive secretions from ever reaching the small intestine, leading to malabsorption, dehydration, and electrolyte disarray (Table 1). Reinfusion of these secretions may be a viable option in select populations, e.g., those with prolonged hospitalization, high volume upper GI losses with persistent fluid and electrolyte imbalance, and imperfect absorption. To date, only case reports have been published on this practice. Bile reinfusion can be complex; for example:

1. When might a clinician consider this option?
2. How is it actually executed on in-patient units?
3. How does this work when transitioning the patient to home or a facility?

Case A

Pancreatitis with gastric outlet obstruction

PG, a 59-year-old male was transferred from an outside hospital to our service for continued management of severe gallstone pancreatitis after cholecystectomy. He was 5’11” with a pre-illness weight of 88 kg (admit weight of 102 kg). He reported normal oral intake up until the time of admission for pancreatitis, however he had been NPO for 7 days prior to transfer. He was

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briefly placed on parenteral nutrition (PN) due to gastric outlet obstruction with prolonged NPO status, catabolic state, and lack of appropriate enteral access.

Two days after admission, a 24 Fr percutaneous endoscopic gastrostomy tube was placed with a 12 Fr jejunal extension tube (PEG/J) (Wilson-Cook; Cook Medical). Standard enteral nutrition (EN) was initiated through his j-tube, followed by PN discontinuation. The PEG was left to gravity drainage to decompress his gastric contents while continuing to feed his jejunum. PG’s gastric output was ~1.5L over 24 hours with reported nausea and emesis. He was currently receiving 15mg of a proton pump inhibitor (PPI) suspension twice daily, via his j-tube, which was increased to 30mg twice daily. The surgical team allowed sips of clear liquids for comfort; however, his gastric output increased to 3-4 L/day. He was having difficulty tolerating the 150mL water flush through his j-tube, causing nausea and hiccups leading to emesis over his vented secretions. His gastric pH was checked to ensure PPI efficacy and confirmed to be >6. Due to the high gastric volume loss, the surgical team started replacement fluids through his j-tube using lactated ringers as ½ mL for each 1 mL of output from his gastric tube, run continuously. He complained of frequent “orange colored” stools, which coincided with the increase of his EN rate.

Two days after the standard EN reached goal rate, it was changed to a semi-elemental formula (Perative; Abbott Nutrition). The rationale for this change was the presumption that the external loss of pancreato-biliary secretions from gastric venting, known pancreatic damage, and his orange colored stools suggesting malabsorption. Due to his current complexity of care, with simpler methods available to stabilize fluid and electrolytes, it was deemed not the time in his course to consider GI reinfusion. After the change to the semi-elemental feeding, the patient reported firmer and color normalization of his stools.

PG required a negative pressure wound therapy vac due to dehiscence at his proximal surgical midline incision necessitating discharge to a facility for wound management, as well as ongoing dependence on intravenous volume replacement. He was changed to a nocturnal EN regimen in anticipation of discharge. His gastric output had decreased to 1-1.5L /day with small sips of clear liquids for pleasure. For volume replacement, he discharged with intravenous Lactated Ringer’s (1:1 replacement of gastric output), as well as water flushes via the j-tube. Weight on discharge was 97 kg.

Five days later, PG was readmitted with failure to...

Table 1. Causes for External Loss of GI Secretions

<table>
<thead>
<tr>
<th>♦ Decompressive gastric tube</th>
</tr>
</thead>
<tbody>
<tr>
<td>o Compression at the pylorus or duodenum causing inability of secretions to flow downward through the GI tract (e.g., pancreatic disease process or duodenal cancer)</td>
</tr>
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<table>
<thead>
<tr>
<th>♦ External biliary drain</th>
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<tbody>
<tr>
<td>o E.g., Percutaneous transhepatic cholangiography (PTC) tube</td>
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| ♦ Enterocutaneous fistulization of the upper GI tract |

<table>
<thead>
<tr>
<th>♦ Upper GI anastomotic leak</th>
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<tbody>
<tr>
<td>o Drain placed at an anastomotic leak involving the pancreas, biliary tree, liver or duodenum</td>
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</table>
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thrive (FTT) and acute kidney injury (AKI) (Table 2); lipase was elevated to 6700. PG’s PPI had been stopped upon arrival at the outside facility and a standard, fiber containing formula had been substituted for the semi-elemental formula he was previously receiving. His fluid replacement was unknown, and the patient reported persistent and severe nausea/vomiting. PG required 10L of resuscitation, the PPI and semi-elemental EN were resumed. Due to his metabolic disarray and dehydration, the decision was made to start GI reinfusion through his j-tube. PG’s gastric pH was rechecked (7.9), therefore appropriate for reinfusion into the small bowel (goal gastric pH = ≥ 6 to mimic normal pH). The patient was stabilized and ready for discharge. The day prior to discharge, tubing was fashioned to shunt gastric output directly from his G-tube into his j-tube. This direct connection of gastric output to jejunal input was to occur during the day when EN was off; at night when j-tube was used for feeding, the G-tube would be left to gravity and those contents bolused into the jejunum in the morning; which varied between 400-600mL. He initially tolerated this change, and PG was discharged to the transitional care facility weighing 95 kg.

While at the transitional care facility, PG did not tolerate his G-tube being hooked directly up to his j-tube, reporting increased nausea/emesis. He also reported not seeing any fluid moving from his G-tube to his j-tube during the day. Intolerance of the large jejunal bolus of GI secretions collected overnight was now occurring. The facility was having difficulty reinfusing all of his upper GI losses. A follow up computed tomography (CT) scan showed a new peri-pancreatic fluid collection, in which a drain was placed. Over the next week, his stools became more watery and profuse and he was found to be C. Diff positive. Additionally, his glycemic control became erratic.

After 33 days, and exhibiting signs of sepsis, with increased nausea and vomiting, PG was readmitted to the hospital. Despite documentation reporting PG was receiving the majority of his goal EN on a consistent basis, he was down to 82 kg. This was presumed due to dehydration as well as his recent poor glycemic control altering his inability to fully utilize his EN. A CT scan demonstrated a recurrent retroperitoneal abscess. At this time, surgery was deemed necessary due to his persistent pancreatic fluid collection, associated fevers, and leukocytosis. Therefore, 2 months after PG’s first admission to our facility, he underwent an open necrosectomy for pancreatic abscess debridement, and 2 additional drains placed in the abscess cavity.

PG’s semi-elemental feeding was started post-operative day (POD) 1 at 20mL/hr, but he suffered from abdominal distention and discomfort when the rate was advanced. PG remained NPO at this time. Therefore, trophic EN continued for 3 days, after which slow advancement by 20mL/day commenced until goal rate was reached. PN was discussed with the surgical team during this time, but they preferred to avoid PN and continue to work on slow enteral advancement given his infectious risk and hyperglycemia. His PEG remained open to gravity, putting out 2-3L/day, with continued nausea. GI reinfusion was resumed as a bolus infusion. Unfortunately, due to his high volume losses and imprecise instruction to the nursing staff, jejunal

Table 2. Weight and Lab Values for PG and SL

<table>
<thead>
<tr>
<th>Lab Results</th>
<th>PG at Discharge</th>
<th>PG Readmit</th>
<th>SL at Discharge</th>
<th>SL Readmit</th>
</tr>
</thead>
<tbody>
<tr>
<td>Na mmol/L</td>
<td>137</td>
<td>139</td>
<td>133</td>
<td>119</td>
</tr>
<tr>
<td>K mmol/L</td>
<td>3.5</td>
<td>3.8</td>
<td>3.5</td>
<td>5.3</td>
</tr>
<tr>
<td>Cl mmol/L</td>
<td>113</td>
<td>113</td>
<td>109</td>
<td>93</td>
</tr>
<tr>
<td>CO2 mmol/L</td>
<td>14</td>
<td>11</td>
<td>16</td>
<td>9</td>
</tr>
<tr>
<td>BUN mg/dL (mmol/L)</td>
<td>19 (6.8)</td>
<td>80 (28.6)</td>
<td>13 (4.6)</td>
<td>101 (36.1)</td>
</tr>
<tr>
<td>Cr mg/dL (umol/L)</td>
<td>1.3 (115)</td>
<td>4.2 (371.4)</td>
<td>0.6 (53.1)</td>
<td>3.0 (265.3)</td>
</tr>
<tr>
<td>Ca mg/dL (mmol/L)</td>
<td>9.3 (2.3)</td>
<td>8.9 (2.2)</td>
<td>8.2 (2.1)</td>
<td>9.5 (2.37)</td>
</tr>
<tr>
<td>Mg mg/dL (mmol/L)</td>
<td>1.8 (0.74)</td>
<td>2.2 (0.91)</td>
<td>1.5 (0.62)</td>
<td>1.8 (0.74)</td>
</tr>
<tr>
<td>Phos mg/dL (mmol/L)</td>
<td>3.4 (1.1)</td>
<td>7.1 (2.3)</td>
<td>2.4 (0.77)</td>
<td>9.4 (3.04)</td>
</tr>
<tr>
<td>Weight</td>
<td>97 kg</td>
<td>95 kg</td>
<td>93 kg</td>
<td>78.2 kg</td>
</tr>
</tbody>
</table>
boluses of >500mL were administered, causing the patient increased nausea and vomiting. EN advancement was further delayed as was adequate EN delivery.

At this time, one of the nurses devised a method for gravity feeding the patient’s gastric output through the j-tube using an enema bag. Biliary secretions were collected (over 4-6 hours), poured into the enema bag, hung alongside the EN, and then simply Y’d in utilizing the gravity method (Figure 2 A-D). The nurse and patient reported good tolerance to this method and patient was able to tolerate 100% reinfusion. Nocturnal EN was reattempted, but was met with intolerance. Therefore, he was discharged back to the transitional care hospital on continuous EN with gravity infusion of GI secretions. Three weeks later, the patient was able to tolerate G-tube clamping for 24 hours; one week later a clear liquid diet was restarted. Two weeks after this, his PPI was stopped. He was discharged home at 87kg. Two weeks later he followed up in surgical clinic, reporting he had stopped his nocturnal EN and was taking solid food. He had no signs or symptoms of malabsorption and weight had been stable since discharge. His feeding tube was removed in clinic.

**Case B Surgical Leak**

SL, a 41-year-old male, transferred to our facility for management of post-op complications. His history prior to transfer included: long term NSAID use, perforated duodenal ulcer requiring Billroth II/gastrojejunostomy and duodenal stump patch (Figure 3). On POD4 he required an exploratory laparotomy for drain dislodgement, and an additional drain placement. POD7, blood was noted in his drains. CT angiography was negative for bleeding; however, esophagogastroduodenoscopy (EGD) revealed a friable anastomosis with ulceration. POD13, he underwent an exploratory laparotomy: his anastomosis remained intact, but bile staining was found at the duodenal stump. It was at this time SL was transferred to our facility with AKI requiring continuous renal replacement therapy (CRRT), an open abdomen and ABThera wound vac. PN was started at the outside facility and was continued on admission. SL was 5’8” and weighed 97.7kg.

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SL was taken to the OR after transfer and underwent:
- Drainage of multiple fluid collections
- Ligation of the gastroduodenal artery
- Closure of a dehisced duodenal stump
- Cholecystectomy
- 2 intraluminal drains placed to divert bile drainage
- J-tube placement
- Abdomen remained open

An elemental formula (Vivonex, Nestle Health Science) was started POD 1 due to loss of pancreato-biliary secretions via external drains (Figure 4 A-B) as follows:
- Drain #1 (most proximal) in the hepatobiliary limb put out ~1L/day of pancreato-biliary secretions (Figure 4 A).
- Drain #2 (distal) put out ~50mL/day (Figure 4 A-B).
- The tip of drain #3 in the right upper quadrant terminated near the duodenal perf repair with minimal serosanguinous fluid (Figure 4 A)

The patient’s GI secretions (drain #1 and #2) continued to increase up to 3L/day; concurrently SL’s stool output also increased requiring a rectal management system. Prior to initiation of reinfusion, the duodenal drainage pH was confirmed to be 7.6. GI reinfusion was then initiated via j-tube on the following day by continuous pump method (continuous method of bile reinfusion required 2 feeding pumps) (Figure 5). In the case of SL, GI secretions were collected every 3 hours and transferred into an empty EN bag to be infused via pump. By utilizing a “Y” adapter, this volume was infused concurrent with EN formula into the jejunum. The combined rate of bile and EN infusion averaged between 300-400mL an hour. Although SL tolerated this volume without complaint, he was changed to a more calorie dense, semi-elemental formula (Vital 1.5, Abbott Nutrition) given his high duodenal and stool output to maximize absorption potential. Of note, it is likely he would have done well on a concentrated standard formula also.

A week after admission, SL returned to the OR for debridement of his abdominal wall and fascia, as well as abdominal closure. His AKI was resolving; he was now off CRRT. Stooling had decreased to once daily. Over the next few days, the volume of his most proximal biliary drain (#1), significantly dropped, and
### Table 4. Various Methods for GI Reinfusion

<table>
<thead>
<tr>
<th>Various Methods</th>
<th>Patient Selection</th>
<th>Administration Technique</th>
<th>Positives</th>
<th>Negatives</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Bolus</strong></td>
<td>Home or hospitalized patient</td>
<td>Collection of bile q 4-6 hours. Pour into a container, draw contents into a syringe (may have to discard some solid components of output). Hold EN Syringe into j-tube.</td>
<td>Minimal supplies needed – only syringe for bolusing</td>
<td>High volume at one time may not be tolerated.</td>
</tr>
<tr>
<td></td>
<td></td>
<td><strong>Supplies Required:</strong> Bolus syringe (such as Bard Toomey Syringe)</td>
<td></td>
<td>There is a limit to how much volume can be infused. Start with 100-200mL bolus and increase only per patient tolerance.</td>
</tr>
<tr>
<td><strong>Gravity</strong></td>
<td>Home (if supplies available) or hospitalized patients</td>
<td>Collect bile every 4-6 hours. Pour into bag with tubing (enema bag). Place Y port connector to j-tube. Run jejunal feeding and bile feeding simultaneously until bile empties from bag.</td>
<td>No need for enteral pump</td>
<td>Requires gravity bags/tubing to administer</td>
</tr>
<tr>
<td></td>
<td></td>
<td><strong>Supplies Required:</strong> Enema Bag (Medichoice Cleansing Enema bag) “Y” connector (Blake Cardio connector 2:1 or Lopez valve/ICU Medical, plus additional universal adapter)</td>
<td>Volume infused slower, potentially better tolerated</td>
<td>May require increased time patient is connected to bile reinfusion</td>
</tr>
<tr>
<td><strong>Pump infusion along with EN</strong></td>
<td>Hospitalized or transitional care facility; Home (if supplies available and patient/caregiver competent to administer)</td>
<td>Collect bile every 4-6 hours. Obtain 2 enteral feeding pumps. Obtain 2 sets of enteral bags and tubing. Place Y port connector to j-tube. Connect EN and bile reinfusion to jejunal Y port. Measure the volume of bile output over the hours you will infuse, and run at that rate (300mL total volume to infuse over the next 4 hours run at 75 mL/hr)</td>
<td>Controlled rate Infuses along with EN; more closely mimics secretions with feeding</td>
<td>Possible insurance issue not covering 2 enteral feeding pumps/bag supplies for home.</td>
</tr>
<tr>
<td></td>
<td></td>
<td><strong>Supplies Required:</strong> Two enteral feeding pumps. Enteral feeding bag and tubing “Y” connector (Blake Cardio connector 2:1 or Lopez Valve (ICU Medical, plus additional universal adapter)</td>
<td>Less volume infused at one time, may increase tolerance</td>
<td></td>
</tr>
</tbody>
</table>
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Table 5. Alternatives to Bile/GI Reinfusion

- Use very low fat elemental enteral formula
- ½ normal saline for replacement*
  - ½ Normal Saline = ¾ teaspoon salt added to 1 liter water
  - Mimics gastric secretions most closely.
  - Consider providing the patient with a liter container of sterile water to ensure they have a container at home to make this (they do not need to use sterile water).
- Frequent lab monitoring until stable—initially weekly after discharge.
  - Monitor electrolytes to ensure the reinfusion plan maintains serum levels.
- If a patient requires prolonged use (> 3-4 weeks) of very low fat formula, monitor for essential fatty acid deficiency

*Be advised, this may be too complicated for some patients to execute at home and should only be utilized in very astute patients and/or those with excellent family support.

The patient tolerated the changes well, and the new regimen appeared to keep SL’s electrolytes and volume status in balance. His weight was 93 kg. SL and family were educated and provided extensive written instructions, including home EN regimen, GI reinfusion, and home reconstitution of 1/2NS. However, it was noted that during the education, the patient and family asked very few questions and did not seem engaged. SL was discharged home and followed up in surgery clinic 4 days later. He reported not tolerating home EN, having increased nausea and vomiting; he was only reinfusing his bile on average of 2x/day (down from 6x/day in the hospital). He reported no BM since discharge. His weight in clinic was 79.5 kg. It was recommended he be readmitted for IV hydration; the importance of GI reinfusion was stressed. The patient declined readmission promising improved compliance at home.

One week later, SL was readmitted with FTT, nausea/vomiting, dehydration, and metabolic disarray (Table 2). SL reported not using EN, and that bile output had vastly tapered off. He was completely unable to eat due to gastric discomfort/fullness; feeling like food was “just sitting there” when he ate. He had not stooped in days. Of note, patient was on narcotics at home and only on stool softeners to relieve his constipation. SL was rehydrated and restarted on appropriate medications:

- PPI via j-tube, maximum dose BID
- Stool softeners and osmotic laxatives prn; goal = 1 stool at least every other day
- Oxycodone prn, for abdominal discomfort
- Reglan, before meals and HS to help with nausea
- 50mL, ½ NS flush after anything administered via j-tube

His EN was changed back to continuous and bolusing of GI secretions into the j-tube every 4 hours was resumed. SL remained in the hospital 4 days tolerating regimen well. He reported less nausea; however, oral intake remained poor. His EN continued to meet his nutritional needs. EN was readjusted to a 16-hour nocturnal regimen with bolus of bile reinfused 6x/day as follows (ideally prior to any oral intake):

- 250mL before and after 16 hour EN run (1800 & 1000)
- 250mL ~ 4 hours into EN infusion (before bed at 2000) and upon waking (0600)—(stop EN, infuse, resume EN)
- 250mL while EN off during day (1200 & 1500)
For total of: 250mL 6x/day (1500mL); discard remaining. In addition:

- Patient to use ½ normal saline (NS) flushes (made with ¼ teaspoon table salt mixed with 1 liter of water—NOTE: would only have those patients prepare this mixture if you are sure they can measure correctly)
  - Give 1 syringe (60mL) after medications, EN infusion, or GI secretion bolus.
  - Patient to increase volume of 1/2NS bolus if oral intake of liquids decreased.

The patient and his family were much more interactive with education for home with a much higher level of interest in EN/reinfusion instructions—they wanted to go home and stay home.

Three days after discharge, patient’s family called reporting increase in stools, increase in nausea and decrease in bile output. He reported infusing all GI secretions, but reported the output had decreased to <1.5L/day. We reviewed exact EN and flush regimen over the phone; the patient had not quite received goal EN or flushes due to his abdominal discomfort. SL’s labs had been checked the day prior and were normal, showing no signs of dehydration. Due to his nausea and diarrhea, EN was changed back to a continuous regimen again. His medications were reviewed; upon discharge his PPI had been changed to an oral capsule vs. the suspension BID via jejunal tube and his antiemetic and prokinetic coverage were inadvertently left off his discharge medications; all of which were resumed.

SL was seen twice in surgery clinic over the next month; his weight maintained at 79.5 kg and he remained well hydrated with decreasing GI secretions from his drains. Three months after his first admission, his final surgical drain was removed. He was off EN, maintaining his weight, and his feeding tube was removed.

APPLICATION AND CONCLUSION

These two cases highlight clinical applications of successful GI reinfusion along with EN to provide nutrition, hydration, electrolyte balance, and improved absorption in patients with excessive loss of GI secretions, avoiding both PN/IV fluids and a central line. They also highlight the complexity of this process, as well as the potential number of mistakes and pitfalls that can easily occur along the way. A multidisciplinary team is clearly needed to achieve success. Once a patient is deemed a good candidate for GI reinfusion (Table 3 lists poor candidates), the best method needs to be determined (Table 4). In deciding on how to reinfuse, it may not only depend on whether the patient tolerates it, but whether the patient will be in a facility or at home. Ideally, GI secretions should be administered concurrent with EN for optimal pancreatic-biliary secretion and nutrient mixing once a pH >6 has been verified. If it is not in the best interest for the patient to receive this therapy, there are other options for EN without reinfusion of endogenous secretions in order to maintain hydration and electrolyte balance (Table 5).

Thank you to the surgical nurses, residents and attendings who help conceptualize unique solutions for complex patients, and who are always willing to take the time to teach me. A special thanks to Joe Freeze, NP.

References