Feasibility of jejunal enteral nutrition for patients with severe duodenal injuries

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Objective: The aim of this study was to evaluate the feasibility of enteral nutrition (EN) for critically ill trauma patients with severe traumatic duodenal injuries who received placement of concurrent decompressing and feeding jejunostomies.

Methods: Adult patients admitted to the trauma intensive care unit from January 2010 to December 2013, given concurrent afferent decompressing and efferent feeding jejunostomies for severe duodenal injury and provided EN or parenteral nutrition (PN), were retrospectively evaluated. Enteral feeding intolerance was defined as an increase in the decompressing jejunostomy drainage volume output, worsening abdominal distension, or cramping/pain unrelated to surgical incisions. Patients who failed initial EN were transitioned to PN.

Results: Twenty-six patients were enrolled. Of the 24 patients given EN within the first 2 wk posthospitalization, 18 (75%) failed EN within 2–24 d of initiating EN. EN was discontinued when increases were seen in decompressing jejunostomy drainage volume output (n = 11) and output with abdominal pain and/or distension (n = 6), or abdominal pain/distension was seen without an increase in output (n = 1). Jejunostomy drainage volume output increased from 474 ± 425 mL/d to 1168 ± 725 mL/d (P < 0.001) during EN intolerance. More patients with blunt intestinal injury than those with penetrating injuries (75% versus 15%, respectively; P = 0.035) tolerated EN. Patients initially given PN (n = 13) received more calories (P < 0.005) and protein (P < 0.001) than those given initial EN (n = 13).

Conclusion: The majority of patients with severe duodenal injuries and concurrent decompressing/feeding tube jejunostomies failed initial EN therapy.

Introduction

The importance of early enteral nutrition (EN) in reducing infectious complications and improving mortality for critically ill surgical and trauma patients is well established and recommended by current guidelines [1–5]. Early EN has been proven superior for reducing infectious complications compared with early parenteral nutrition (PN) when given to patients with major abdominal trauma [2,6,7]. As a result, early EN is given to critically ill patients with traumatic injuries whenever possible.

Duodenal injuries are associated with high rates of morbidity and mortality, especially when the injury is in combination with pancreatic injury [8,9]. However, traumatic duodenal injury occurs infrequently due to its protected retroperitoneal location. Patients enrolled in this study required placement of a nasogastric or orogastric tube, retrograde (afferent) decompressing jejunostomy, and antegrade (efferent) feeding jejunostomy in addition to provision of EN or PN. Unfortunately, little is known regarding EN management for these patients. Our anecdotal practice observations provided the opinion that the provision of EN for these patients was associated with a high rate of feeding intolerance. The purpose of this study was to evaluate the feasibility of EN for patients with severe duodenal injuries and concurrent decompressing and feeding jejunostomies.
Materials and methods

Patient selection

Adult critically ill patients, age ≥ 18 y, admitted to the intensive care units (ICUs) of the Presley Trauma Center of Regional One Health in Memphis, Tennessee, with a severe traumatic injury to the duodenum or proximal jejunum at or near the ligament of Treitz with placement of concurrent decompressing and feeding jejunostomies were eligible for the study. Patients were referred to the Nutrition Support Service for EN or PN. Study candidates were retrospectively identified from the Nutrition Support Service monitoring records from January 2010 to December 2013. The patients’ electronic medical and Nutrition Support Service records were reviewed for data retrieval. Injury Severity Score (ISS) [10], Abbreviated Injury Scale (AIS)-Abdomen [11], survival, ventilator days, hospital length of stay (LOS), and ICU LOS were retrieved from the trauma registry of Regional One Health. The assignment of the American Association for the Surgery of Trauma (AAST) duodenal injury score [12] was determined based on a consensus of participating attending surgeons from the institution for a different study.

The study was approved and conducted in accordance with guidelines established by the University of Tennessee Health Science Center Institutional Review Board and Regional One Health Office of Medical Research. Because all measurements were performed as part of routine clinical care of the patients and because confidentiality procedures for the patients were maintained, the requirement for written informed consent was waived.

Surgical procedures

At our institution, usual surgical management for patients with AAST grade I duodenal injury is primary repair of the lesion. Patients with AAST grade II or III injury and selected grade IV duodenal injuries receive a primary repair of the intestinal lesion with placement of a decompressing naso or oro-gastric tube, retrograde (afferent) decompressing jejunostomy, and antegrade (efferent) feeding jejunostomy [13]. Other grade IV wounds are managed via repair and duodenal exclusion. A 14- to 16-French red rubber catheter is used for both jejunostomies. Each catheter is introduced into the jejunum through separate enterotomies and is secured using a Witzel technique. The proximal (afferent) decompressing tube is placed distal to the duodenal injury and left open to gravity for drainage. The distal (efferent) jejunostomy tube is used for continuous enteral feeding. A schematic representation for management of severe duodenal injuries with placement of decompressing gastric and jejunostomy tubes with a feeding jejunostomy is provided in Figure 1.

Nutrition therapy

In the present study, the route of initial nutrition therapy was determined by the attending trauma physician based on the extent of organ injuries; complications experienced during the operative procedure; amount of blood loss and fluid resuscitation; postoperative requirement for vasopressor therapy; or intestinal complications such as ileus, obstruction, anastomotic leak, or a fistula. The Nutrition Support Service managed the EN or PN. Patients were assigned energy and protein goals of 25 to 32 kcal/kg daily and 2 to 2.5 g/kg daily, respectively [14]. Prerescuscitation body weight was used to determine target nutritional goals. EN-fed patients were given an enteral formula (1.3 kcal/mL, 78 g of protein/L) containing glutamine, arginine, dietary nucleotides, and ω-3 fatty acids [15] at an initial rate of 15 to 25 mL/h via the feeding jejunostomy. The feeding was increased by 15 to 25 mL/h daily until the goal rate was achieved or feeding intolerance observed. Additional liquid protein supplements were provided as needed to achieve goal intakes. Parenteral nutrition therapy was initiated at 25 to 40 mL/h at approximately one-third of the goal amount of macronutrients, fluid, and electrolytes and advanced daily over 3 d until the goal regimen was achieved. Energy intake was decreased, whereas protein intake was maintained for those who received a propofol infusion containing 10% lipid emulsion. We achieved this by eliminating lipid calories and decreasing glucose calories for PN and by reducing enteral feeding rate and addition of liquid protein supplements for those receiving EN. Blood glucose concentrations were maintained between 70 and 150 mg/dL [16,17].

Enteral feeding intolerance was defined as approximately a > 300 mL/d increase in the decompressing jejunostomy volume output or worsening abdominal distension, patient complaint of nonspecific signs or symptoms of bloating, or both; abdominal cramping, severe nausea, emesis, or abdominal pain not related to the surgical incision. Because objective criteria derived from studies regarding jejunal EN feeding intolerance for this unique population were lacking, we defined intolerance based on our anecdotal experience and that of others regarding antecedent signs and symptoms associated with reports of complications of intestinal ischemia and bowel necrosis following jejunal enteral feeding in critically ill surgical and trauma patients [18–22]. When enteral feeding intolerance occurred, EN was held and PN was initiated for those who received initial PN therapy. Once the signs and symptoms of enteral feeding intolerance abated, another trial of EN was cautiously reinitiated. PN was discontinued once enteral feeding tolerance was established. Those who failed EN while receiving initial PN therapy continued to receive PN until enteral feeding tolerance was achieved.

Measured and outcome variables

Serum laboratory tests were ordered either by the patient’s primary service or the Nutrition Support Service and performed by the hospital laboratory as part of the patients’ routine clinical care. Demographic, clinical outcomes, and nutrition data were collected. Patients who could communicate were interviewed daily for evidence of abdominal cramping, distension, bloating, nausea, or abdominal pain unrelated to the surgical incision. A physical exam of the abdomen was conducted daily. Nursing fluid intake and output records were reviewed for episodes of emesis, gastric and afferent drain volume output, bowel movements, and nutrition volume intake. Documentation of infectious and intestinal complications was obtained from the patient’s electronic medical records and daily progress notes. Pneumonia was evident by clinical signs and symptoms and confirmed by bronchoalveolar lavage with the presence of > 10^5 colony-forming units/mL. The number of days patients received antibiotic therapy was monitored; no effort was made to ascertain whether the therapy was empiric or therapeutic. A nitrogen balance determination was conducted for patients in the ICU while receiving EN or PN as previously described [14].

Statistical analysis

Data analysis was conducted using SigmaPlot for Windows, version 11.2 (Systat Software, Point Richmond, VA, USA). The data were evaluated for normality of the distribution by the Shapiro–Wilk test. Independent variables were compared by applying the t test for unpaired variables for normally distributed data or the Mann-Whitney U-test if not normally distributed. The t test for paired variables or Wilcoxon Signed Rank test was used for comparing pre- and post-variables. Two-way analysis of variance was used for assessing serial data with post hoc, pairwise comparison procedures by the Student-Newman-Keuls method. Nominal data were analyzed by χ² or Fisher Exact test. Continuous data were expressed as mean ± SD. The significance testing and reported probability values (P-value) were two-sided for all variables. A probability P ≤ 0.05 was established as statistically significant.

Results

Patient characteristics

Twenty-six critically ill patients admitted to the trauma or surgical ICU with severe duodenal or proximal jejunal injuries at or near the ligament of Treitz, who had placement of concurrent decompressing and feeding jejunostomies, and referred to the Nutrition Support Service for EN or PN, were enrolled into the
study. The majority of patients were men (92%) and African American (65%). The mean age was 32 ± 11 y and most patients were of adequate body weight with a mean body mass index of 29 ± 8 kg/m². Serum prealbumin concentrations were substantially decreased below normal, whereas serum C-reactive protein concentrations were markedly elevated. Two patients required vasopressor therapy. Intestinal injuries were due to penetrating trauma or blunt trauma in 78% and 24% of patients, respectively. Twenty patients experienced traumatic injury to the duodenum and five patients had proximal intestinal injuries at or near the ligament of Treitz. The exact location of the intestinal injury was not electronically recorded for one patient, but effenter and effenter jejunostomy tubes were present, which allowed inclusion of the patient into the study. Sixty-two percent of patients (n = 16) had concurrent pancreatic injury. Nutrition therapy (EN or PN) was initiated 3 ± 2 d after admission to the ICU. Infectious complications were common with a 77% incidence of occurrence for all patients. The patients’ nitrogen balance was −8.5 ± 7.8 g/d while receiving 1.6 ± 0.8 g/kg of protein daily after 10 ± 5 d of hospitalization.

Enteral feeding tolerance and clinical outcomes

Jejunal feeding was attempted within the first 14 d post-hospital admission while in the ICU in 24 of 26 patients. The remaining two patients received only PN during this time period; one died after 8 d in the ICU and the other developed an enterocutaneous fistula and received 7 wk of PN before initiation of an oral diet. Of the 24 patients, 18 (75%) who were given jejunal EN within the first 2 wk of hospitalization failed EN within 2 ± 2 d of initiating EN. Fourteen patients experienced intolerance within the first 2 d of initiation of EN. Intolerance occurred in two additional patients after 3 d, and one each by days 4 and 8 of EN. Ten of 13 patients (77%) initially given EN failed EN therapy. Eight of 14 (57%) who were given initial PN and then a trial of EN within the first 14 d of hospital admission also failed the first attempt at EN. Initiation of jejunal EN was delayed for those who received initial PN. Jejunal feeding was given 8 ± 4 d after hospitalization for those given initial PN compared with 3 ± 1 d for those given initial EN (P < 0.001). None of the patients were given prokinetic metoclopramide or erythromycin pharmacotherapy, nor received vasopressor therapy during EN administration.

EN was discontinued due to an increased decompressing jejunostomy (afferent) tube output for 11 patients, increased afferent drainage output with abdominal pain and/or distention in 6 patients, and abdominal pain/distention without an increase in afferent tube volume output in 1 patient. Afferent jejunostomy drainage output increased by an average of 700 mL for those who experienced EN intolerance (P < 0.001; Fig. 2). Gastric drainage output also increased from 412 ± 304 to 904 ± 594 mL/d (P = 0.007) for those with EN intolerance. It is unknown to what proportions the increased drainage output was comprised of gastrointestinal secretions, bile, or enteral feeding. The amount of enteral nutrition volume received within the past 24 h before EN intolerance was 480 ± 359 mL/d while receiving EN at a rate ranging from 15 to 50 mL/h. None of the patients experienced severe intestinal ischemia and bowel necrosis.

**Table 1** compares various patient characteristics of those who were intolerant to EN and those who tolerated EN. There were no differences between EN tolerance groups with respect to demographic and laboratory data. Severity of injuries, as evidenced by ISS, AAST duodenal injury score, AIS-Abdomen, presence of pancreatic injury, selection of route for nutrition therapy, or baseline gastric or afferent drainage output were not significantly different between EN tolerance groups. Patients with EN feeding intolerance tended to have more other small bowel and colon injuries; however, these differences were not statistically

### Table 1

<table>
<thead>
<tr>
<th>Variable</th>
<th>EN intolerant</th>
<th>EN tolerant</th>
<th>P-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>N</td>
<td>18</td>
<td>6</td>
<td>-</td>
</tr>
<tr>
<td>Age (y)</td>
<td>33 ± 10</td>
<td>28 ± 12</td>
<td>NS</td>
</tr>
<tr>
<td>Sex, male/female</td>
<td>16/2</td>
<td>6/0</td>
<td>NS</td>
</tr>
<tr>
<td>Weight (kg)</td>
<td>91 ± 25</td>
<td>92 ± 25</td>
<td>NS</td>
</tr>
<tr>
<td>BMI (kg/m²)</td>
<td>29.3 ± 8.2</td>
<td>29.3 ± 7.5</td>
<td>NS</td>
</tr>
<tr>
<td>Race, n (%)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>White</td>
<td>3 (16)</td>
<td>2 (33)</td>
<td>NS</td>
</tr>
<tr>
<td>African American</td>
<td>12 (67)</td>
<td>4 (67)</td>
<td>NS</td>
</tr>
<tr>
<td>Other</td>
<td>3 (16)</td>
<td>0</td>
<td></td>
</tr>
<tr>
<td>Admission diagnosis</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>GSW/KSW</td>
<td>15</td>
<td>4</td>
<td>NS</td>
</tr>
<tr>
<td>MVC/fall</td>
<td>3</td>
<td>2</td>
<td></td>
</tr>
<tr>
<td>Initial T-max (°F)</td>
<td>101 ± 0.8</td>
<td>101.5 ± 1.2</td>
<td>NS</td>
</tr>
<tr>
<td>WBC, cells/mm³</td>
<td>8.7 ± 3.6</td>
<td>8.6 ± 4.3</td>
<td>NS</td>
</tr>
<tr>
<td>Injury severity score</td>
<td>22.5 ± 7.9</td>
<td>22.7 ± 4.0</td>
<td>NS</td>
</tr>
<tr>
<td>Mechanism of injury, n (%)</td>
<td></td>
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<td></td>
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<tr>
<td>Penetrating</td>
<td>17 (83)</td>
<td>3 (50)</td>
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<tr>
<td>Blunt</td>
<td>1 (17)</td>
<td>3 (50)</td>
<td></td>
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<tr>
<td>Location of injury</td>
<td></td>
<td></td>
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<tr>
<td>Duodenum, n</td>
<td>15 (83)*</td>
<td>3 (50)</td>
<td>NS</td>
</tr>
<tr>
<td>At or near ligament of Treitz, n</td>
<td>2 (11)</td>
<td>3 (50)</td>
<td>NS</td>
</tr>
<tr>
<td>AAST duodenal injury score</td>
<td>2.5 ± 0.7</td>
<td>2.3 ± 1.0</td>
<td>NS</td>
</tr>
<tr>
<td>Other small bowel injury, n (%)</td>
<td>9 (50)</td>
<td>1 (17)</td>
<td>NS</td>
</tr>
<tr>
<td>Colon injury, n (%)</td>
<td>14 (78)</td>
<td>3 (50)</td>
<td>NS</td>
</tr>
<tr>
<td>Gastric injury, n (%)</td>
<td>6 (33)</td>
<td>1 (17)</td>
<td>NS</td>
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<tr>
<td>Pancreatic injury, n (%)</td>
<td>11 (61)</td>
<td>4 (67)</td>
<td>NS</td>
</tr>
<tr>
<td>AIS-Abdomen</td>
<td>3.8 ± 0.9</td>
<td>4.2 ± 0.8</td>
<td>NS</td>
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<tr>
<td>Initial EN/PN, n/n</td>
<td>10/8</td>
<td>3/3</td>
<td>NS</td>
</tr>
<tr>
<td>Hospital day of initial EN, n (%)</td>
<td>5.1 ± 3.5</td>
<td>5.0 ± 3.3</td>
<td>NS</td>
</tr>
<tr>
<td>Initial pH</td>
<td>7.35 ± 0.06</td>
<td>7.38 ± 0.03</td>
<td>NS</td>
</tr>
<tr>
<td>Prealbumin (mg/dL)</td>
<td>7.5 ± 2.9</td>
<td>9.2 ± 4.1</td>
<td>NS</td>
</tr>
<tr>
<td>Creatinine (mg/dL)</td>
<td>1.4 ± 0.6</td>
<td>1.7 ± 1.6</td>
<td>NS</td>
</tr>
<tr>
<td>SUN (mg/dL)</td>
<td>19 ± 8</td>
<td>21 ± 13</td>
<td>NS</td>
</tr>
<tr>
<td>Baseline gastric output (mL/d)</td>
<td>374 ± 355</td>
<td>538 ± 536</td>
<td>NS</td>
</tr>
<tr>
<td>Baseline afferent output (mL/d)</td>
<td>492 ± 452</td>
<td>492 ± 523</td>
<td>NS</td>
</tr>
</tbody>
</table>

AAST, American Association for the Surgery of Trauma; AIS, Abbreviated Injury Scale; BMI, body mass index; EN, enteral nutrition; GSW, gunshot wound; ICU, intensive care unit; KSW, knife stab wound; LOS, length of stay; MVC, motor vehicle collision; PN, parenteral nutrition; SUN, serum urea nitrogen concentration; Tmax, maximum body temperature; WBC, white blood cell count.

- Excludes 1 patient in whom the exact location of the duodenal injury was unknown.

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significant (Table 1). However, a greater proportion of patients with blunt intestinal injuries tolerated jejunal delivery of EN compared with those with penetrating abdominal injuries (75% versus 15%, respectively, \( P = 0.035 \)). Clinical outcomes for those with EN intolerance compared with those who tolerated EN including survival (100% versus 83%), ICU LOS (12 ± 11 d versus 10 ± 7 d), ventilator days (5 ± 8 d versus 6 ± 7 d), infectious complications (72% versus 83%), days of antibiotic therapy (12 ± 9 d versus 14 ± 9 d), and surgical complications (17% versus 17%) were not significantly different (\( P = NS \) for all comparisons). However, a longer hospital LOS was noted for those with EN intolerance compared with those who tolerated EN (39 ± 31 versus 22 ± 9 d, respectively; \( P = 0.039 \)).

**Initial EN versus initial PN therapy**

Initial EN was provided to thirteen patients while the remaining thirteen patients received initial PN. Nutrition therapy was started 3 ± 1 d versus 3 ± 2 d following hospital admission for initial EN and PN groups, respectively (\( P = NS \)). ISS and AIS-Abdomen indicated comparable traumatic and abdominal injuries for both PN and EN feeding groups. All patients who received initial PN had experienced a duodenal injury compared with 62% of patients who received initial EN (Table 1), but these differences were not significantly different (\( P = NS \)). AAST duodenal injury scores were similar between initial EN and PN feeding groups (2.5 ± 0.8 versus 2.5 ± 0.7, \( P = NS \), respectively) for those with duodenal injuries. Additional details regarding patient characteristics for initial route of feeding groups are presented in Table 2.

Patients who initially were given PN received more calories (\( P < 0.005 \)) and protein (\( P < 0.001 \)) than those given initial EN over the course of 2 wk of therapy (Fig. 3). The post hoc pairwise comparisons denoted a significant difference (\( P < 0.05 \)) in caloric and protein intakes between feeding groups for days 4 through 7. Caloric and protein intakes for days 8 through 14 were not different between EN and PN feeding groups due to the initiation of PN for those who failed EN therapy. Enteral intake for the initial EN-fed group accounted for <40% of total nutrient intake for the postoperative week 2 (Fig. 4). All clinical outcomes for those initially given EN were not significantly different (\( P = NS \)) from those initially given PN including survival (100% versus 85%), ICU LOS (9 ± 5 d versus 16 ± 15 d), ventilator days (3 ± 3 d versus 10 ± 11 d), hospital LOS (36 ± 35 d versus 34 ± 18 d), infectious complications (69% versus 85%), and days of antibiotic therapy (10 ± 8 d versus 13 ± 8 d). Those who initially received PN had an increased incidence of intestinal complications (0 versus 38% for the initial EN versus initial PN group; \( P = 0.039 \)). These intestinal complications included duodenal repair (n = 5), obstructed ileus (n = 1), and anastomotic leak from the surgical repair (n = 1). However, these intestinal complications are what led to the selection of initial PN therapy for these patients and not necessarily associated with morbidity from PN.

**Discussion**

The benefit of early EN therapy for critically ill trauma patients is well established [1–4,23,24]. However, early jejunal feeding for patients who require major intestinal surgery or with severe intestinal injury is not without risk. Patients with severe intestinal injuries are more likely to experience feeding jejunostomy-associated complications such as intestinal leaks, perforations, volvuli, secondary infections, and bowel necrosis [18]. Although rare, published cases of nonocclusive intestinal ischemia leading to catastrophic bowel necrosis have been reported for surgical patients fed via jejunostomy [18–22] as well as anecdotally observed in our clinical practice. Thus, diligent and meticulous monitoring of the critically ill patient with severe intestinal trauma and jejunostomy feeding is mandatory.

The findings of the present study indicate that clinicians should have a heightened awareness for the likelihood of enteral feeding intolerance for jejunostomy-fed patients with severe duodenal injuries. The data demonstrated that the 75% incidence of enteral feeding intolerance within the first postoperative week was markedly higher than anticipated compared with other critically ill trauma patients at our institution [2,25]. Our past experience with jejunal feeding intolerance (defined as significant abdominal distension) indicated only 4% of critically ill patients with blunt or penetrating abdominal trauma who required emergent laparotomy experienced EN intolerance [2]. However, only 3 of the total of 51 patients who were given jejunal feeding in that study experienced a duodenal injury and it is not known if the severity of the injury warranted placement of concurrent afferent and efferent jejunostomies [2].

It was possible that our conservative criteria for evidence of jejunal feeding intolerance may have contributed to this high EN intolerance rate. Our definition for enteral feeding intolerance was developed independently by clinicians at our institution due to limited guidance from the literature as published objective data for defining appropriate markers for the safe administration of small bowel feeding were lacking. It was our goal to successfully gain the benefits of early nutrition therapy [1–4,23,24,26,27], yet avoid intestinal-related morbidity from jejunostomy feeding [18–22]. Patient safety was of utmost importance. Because the intent of decompression via the afferent and gastric drainage tubes was to reduce intraluminal duodenal pressure,
increase in gastric tube drainage (Fig. 2) was included as strict criteria for discontinuation of EN. Worsening distension, determined by physical examination of the abdomen, was included in the criteria for discontinuing EN. Finally, patient complaint of nonspecific signs or symptoms of bloating, abdominal cramping or pain not related to the surgical incision, severe nausea, or emesis were considered early signs or symptoms of feeding intolerance because these insidious findings have been suggested to be prodromal to catastrophic bowel necrosis for patients receiving early jejunal tube feeding [18–22]. However, despite implementation of these empiric safeguards, it has been suggested that nonocclusive bowel necrosis may not reliably manifest clinical signs for early detection in trauma patients who receive small bowel feeding [20].

Our observation regarding the incidence of feasibility for jejunal EN tolerance is alarming particularly if clinicians exclusively rely on EN therapy for highly catabolic, critically ill trauma patients with duodenal injuries as inadequate nutrition therapy over a prolonged period has been shown to have detrimental effects. Before the routine use of prokinetic therapy for patients with gastric feeding intolerance, patients with traumatic brain injury (known to have a high incidence of gastric feeding intolerance [25]) were randomized to receive EN versus PN [23]. Data from that study indicated that improved nutrient delivery with PN as opposed to inadequate EN intake during the first 2 wk postinjury resulted in improved nitrogen balance and survival [23]. In a recent randomized control trial (RCT) for critically ill patients with an anticipated prolonged ICU stay, patients given supplemental PN for EN feeding intolerance, as opposed to allowing them to continue with suboptimal EN therapy, resulted in a reduced incidence of nosocomial infections [28]. Other recent data further support this concept of optimizing caloric and protein intake in general for critically ill patients to improve clinical outcomes as opposed to allowing hypocaloric, low-protein feeding [29–33]. Results from a recent large RCT demonstrated no difference in 30-d mortality when nutrition therapy was given via the parenteral or enteral route for early nutrition support in critically ill patients [34].

However, these data should not be extrapolated to imply that all critically ill patients should receive supplemental or early PN. It has been demonstrated that provision of PN within 1 d after ICU admission versus no nutrition for 3 d resulted in an increased rate of infectious complications with early initiation of PN [35]. Additionally, no improvement in 60-d mortality for those given early PN was evident [35]. Therefore, duration of lack of nutrition intake as well as the severity and duration of the critical illness are likely important factors regarding the potential anticipated clinical outcome response with respect to timing, route, and amount of nutrition intake.

One retrospective study, more closely reflective of our critically ill trauma patient population, suggested that supplemental PN, when given to EN-tolerant patients, was associated with increased infectious complications [36]. However, those patients who received supplemental PN also had more severe injuries, which may have confounded their findings. In contrast to their patient population [36], all patients who were transitioned to PN in our study were demonstrated to be intolerant to EN and would have received minimal or no nutrition therapy for as long as 1 to 2 wk for the majority of patients if PN was not initiated. Further study to elucidate when PN is most beneficial for EN-intolerant critically ill trauma patients is clearly warranted.

One strength of this study was that we were able to detect an exaggerated rate of enteral feeding intolerance in patients with severe duodenal injuries and concurrent decompressing and feeding jejunostomies. However, this study had limitations. There are no established criteria or consensus recommendations for the definition of enteral feeding intolerance for this unique patient population. The selection of initial route of delivery of nutrition therapy was not randomized. Patients who failed EN were not allowed to remain without nutrition therapy and the initiation of PN may have skewed clinical outcomes. Finally, the
number of patients was limited despite a 4-y observation period at a busy urban level 1 trauma center.

Conclusion

Data from the present study indicate that about 75% of critically ill patients with severe duodenal injuries and with concurrent decompressing and feeding jejunitostomy do not tolerate early enteral feeding. Because of their high level of catabolism, anticipated prolonged ICU and hospital LOS, and high morbidity risk, we have chosen to initiate PN when EN tolerance cannot be established. A large, multicenter RCT would be useful in evaluating the clinical benefit of early transition to PN when EN intolerance occurs for this unique population.

References