Nutrition Support in Pancreatitis

Caitlin S. Curtis, PharmD\textsuperscript{a},
Kenneth A. Kudsk, MD\textsuperscript{b, c, *}

\textsuperscript{a}Department of Pharmacy, University of Wisconsin-Madison Hospital and Clinics,
600 Highland Avenue, CSC – 1530 F6/133, Madison, WI 53792, USA

\textsuperscript{b}Department of Surgery, Veterans Administration Surgical Services, William S. Middleton
Memorial Veterans Hospital, Madison, WI, USA

\textsuperscript{c}Department of Surgery, University of Wisconsin-Madison Hospital and Clinics,
600 Highland Avenue, CSC H4/736, Madison, WI 53792-7375, USA

Pancreatitis poses a significant nutritional risk for various reasons, requiring that caregivers pay special attention to provision of nutrition in their treatment. First, pancreatitis increases nutritional requirements, because the disease process causes massive local and systemic inflammatory responses resulting in hypermetabolism [1] and hypercatabolism [2]. Second, the disease usually obviates the ability of patients to meet their energy needs by oral intake, as the symptoms of pancreatitis (most notably abdominal pain) prevent them from doing so. Lastly, pancreatitis impairs intestinal function, making it difficult or impossible to feed orally or enterally.

In recent years, concepts in the nutrition support of the patient with pancreatitis changed, evolving from complete bowel rest with administration of parenteral nutrition (PN) to recent recommendations for nasojejunal (NJ) and nasogastric (NG) feedings. In the past, complete bowel rest with nutrition support with PN was standard of care for pancreatitis, even though this treatment was not evidence-based. Today, multiple trials and meta-analyses [3–9] support the recommendation that enteral feeding by means of the jejunum is safe and more cost-effective than PN. In several recent trials [9–11], evidence suggested that under some conditions, nasogastric feeding may be as safe and effective as jejunal feedings. Tolerance of enteral nutrition (EN), however, varies from patient to patient in acute pancreatitis, and during the course of patient care, clinicians must make decisions regarding nutrition support that will benefit the patient most. This article explores the
current evidence supporting nutritional supplementation in acute and chronic pancreatitis.

**Mild and moderate pancreatitis**

Mild and moderate pancreatitis is usually self-limiting, responds to short periods of bowel rest, and resolves before any significant malnutrition occurs. In a study by Louie and colleagues [3] comparing the route of EN to PN in acute pancreatitis, 184 patients had a Ranson’s severity score of 3 or greater; of these, 120 patients tolerated oral intake within 5 days of admission. Sax and colleagues [12] reported that 80% of patients who had Ranson’s criteria less than or equal to 2 tolerated an oral diet by hospital day 7. In addition, 54% of patients who had Ranson’s criteria greater than or equal to 2 tolerated a diet by hospital day 7. Abou-Assi and colleagues [4] treated patients with bowel rest, intravenous fluids, and analgesics for 48 hours after establishing a diagnosis of acute pancreatitis. Seventy-five percent of these patients improved within this time period without the need for specialized nutrition support. Furthermore, 87% of patients resumed an oral diet within the next 2 days. These studies all support withholding specialized nutrition support in mild and moderate pancreatitis, since an oral diet can be instituted within a short period of time after the initial diagnosis.

**Severe pancreatitis**

Severe acute pancreatitis, including necrotizing pancreatitis, carries a high mortality rate and requires surgical intervention in many cases. Standard therapy for these patients includes fluid resuscitation followed by nutrition support until the patient is capable of taking an oral diet. For many years, the standard of care dictated complete bowel rest and initiation of PN, but recent trials challenge this practice and now support feeding by means of the gastrointestinal (GI) tract [3–6]. These trials show that EN in the form of NJ feeding is as safe as PN and is more cost-effective. Results of EN trials show that this treatment approach often shortens the time to initiation of oral diet, reduces complications, and shortens hospital stay. The theory is that the beneficial effects of enteral feeding are because of the maintenance of intestinal barriers and decreases in bacterial translocation. Another potential mechanism proposes that enteral feeding modulates the immune response, decreasing the systemic inflammatory response, which contributes to morbidity in acute pancreatitis. Many trials [5,6,13] have shown the benefits of jejunal feeding in pancreatitis while documenting cost savings and lower complication rates. One of the first trials by McClave and colleagues compared 32 patients admitted for acute pancreatitis; 16 patients were randomized to receive enteral nutrition by means of NJ feeding tubes, and 16 patients were randomized to receive PN. The primary goal in
This study was to determine safety of EN in pancreatitis, with additional goals of determining efficacy, clinical outcomes, and cost-effectiveness of EN. The study found no difference between caloric advancement or serial pain scores between the groups but noted that Acute Physiology and Chronic Health Evaluation (APACHE) II scores, Ranson’s criteria, and multiple organ failure scores decreased in the NJ group.

Every 2 to 3 days, the investigators compiled Ranson’s scores. NJ-fed patients had significantly decreased Ranson’s scores on the third serial measurement when compared with PN-fed patients. NJ feeding significantly reduced mean costs per patient ($3294 in the PN group and $761 in the NJ group, $P < .02)$. The authors concluded that NJ feeding was as safe and effective as PN and more cost-effective [5]. Kalfarentzos and colleagues [6] studied 38 patients with acute pancreatitis, of which 18 patients received EN and 20 patients received PN. The primary aim of the study was to compare whether nutritional status could be maintained equally in each group. Secondary endpoints included the rate of complications and cost of treatment. Both groups tolerated feedings well with no significant differences in nitrogen balance between the two groups. Nitrogen balance improved through the course of the study in both groups. EN feeding significantly reduced complication rates compared with PN feeding and, specifically, lowered the mean number of infections per patient. The authors concluded that EN maintains lean tissue mass as effectively as PN and produces fewer complications. Windsor and colleagues [13] reported the feasibility of EN in patients who had severe pancreatitis and measured C-reactive protein (CRP) levels before and after treatment. They enrolled 34 patients; 16 patients were randomized to NJ feedings and 18 patients to PN. Of these patients, 13 had severe pancreatitis, and 21 had mild/moderate pancreatitis. The primary outcome of the study was the incidence of systemic inflammatory response syndrome (SIRS), with the secondary endpoints being sepsis, organ failure, hospital stay, and mortality. The authors measured CRP levels within 48 hours from time of enrollment and 7 days later. EN feeding significantly lowered CRP levels at 7 days compared with baseline, whereas PN feeding resulted in no significant change in CRP levels at day 7 compared with baseline. At baseline, 23 patients had SIRS. SIRS was present in 11 of the EN-fed patients before starting nutrition therapy, but present in only two patients after 7 days of EN, a significant decrease ($P < .05$). SIRS was present, however, in 12 of the PN-fed patients before enrollment and in 10 patients after 7 days of PN (nonsignificant). Hospital stays were not significantly different between the groups. Additionally, EN feeding was associated with significantly lower APACHE II scores from baseline, while PN feeding produced no significant effect on these scores. The authors concluded that EN is “both feasible and desirable” in patients who have acute pancreatitis, and suggested that EN stimulation attenuated the acute-phase response that improved clinical outcomes.
Several other trials testing EN versus PN support these conclusions [3]. Abou-Assi and colleagues [4] showed that pancreatitis patients randomized to NJ feeds had a significantly shorter duration of nutrition support, lower costs, and significantly fewer septic and metabolic complications when compared with patients fed with PN. Louie and colleagues showed that EN was safe and more cost-effective in pancreatitis than PN. Meta-analysis and systematic reviews [7,8] comparing methods of feeding in pancreatitis patients showed that EN is superior in cost, decreases the inflammatory response, and leads to earlier transition to oral intake when compared with PN.

One limitation to jejunal feeding remains the difficulty in obtaining access [5,14–16]. Only 5% to 15% of feeding tubes placed into the stomach migrate into the jejunum spontaneously. Thus, feeding tube advancement requires another method, usually by radiographic or endoscopic procedures [14]. These procedures are expensive and inconvenient, as they involve transporting a potentially unstable patient from the ICU setting. Additionally, they delay EN until successful placement of the tube. Bedside placement is a more popular method of placing NJ tubes; however, the procedure requires significant training with varying success rates reported [14,17].

**Nasogastric feeding**

Historically, the leading theory espoused that EN stimulates the release of pancreatic enzymes and worsening the pancreatitis. Therefore the belief persisted that gastric feeding was contraindicated in this condition. Animal models of acute pancreatitis, however, showed that exocrine function in the pancreas remains unresponsive to enteral feeding [18]. Several trials have shown that patients with pancreatitis tolerate nasogastric (NG) feeding well [9–11]. Preliminary work by O’Keefe and colleagues [4] showed that stimulation of exocrine function remains suppressed at basal rates in patients who have varying severity of pancreatitis. These observations provide the basis for recent trials investigating the safety and efficacy of intragastric feeding. In one trial by Eatock and colleagues [9], 50 patients who had acute pancreatitis received either NG feeding or NJ feeding after randomization. The authors compared APACHE II scores, CRP measurements, and pain patterns by visual analog scale (VAS). Twenty-two patients received NG feedings, and 27 patients received NJ feedings with no significant difference in rate of administration or target calories. Neither APACHE II scores nor CRP measurements differed significantly on any given day. VAS and analgesic requirements were not significantly different between the two groups. The authors concluded that NG feeding shows no evidence of exacerbation of disease when compared with NJ feeds, rendering NG feeding a viable option when supporting patients who have acute pancreatitis [9]. Kumar and colleagues [10] randomized 15 patients to NG feeding and 16 patients to NJ feeding, comparing tolerance and recurrence of pain between the groups. Pain recurred in only one patient in each group, with no significant rise in
serum amylase or worsening of pancreatitis. The authors, however, noted requirements for partial PN in four NJ-fed patients and six NG-fed patients, because intolerance of EN limited delivery of calories. These numbers were not significant, however. Eckerwall and colleagues [11] compared patients receiving NG feeding with patients receiving PN. Outcomes measurements included serum levels of inflammatory markers (interleukin [IL]-6, IL-8, and CRP) and clinical morbidity and feasibility of the nutritional route. Baseline inflammatory markers IL-6 and CRP levels remained similar in the groups at every time point tested. Individual complications were similar between the groups, although total complications significantly increased in the NG group compared with the PN group (P = .04). The complications, however, included pleural effusion and atelectasis, but the authors noted that these complications were non-nutrition related. Nevertheless, the results of these three trials comparing NG feeding with another route of feeding demonstrate that NG feeding may be a safe option in patients who have acute pancreatitis. Ultimately, tolerance determines the route of feeding, but patients benefit from NG feeding because of easy accessibility, lower costs, and earlier initiation of EN. Most practitioners (including the authors of this article) remain wary of instituting NG feedings in severe pancreatitis, preferring the NJ route if no evidence of ileus or obstruction exists on abdominal imaging. Patients generally tolerate the feedings well and advance to a general diet within 10 to 14 days. For patients who have increased nausea, bloating, or abdominal pain, after initiation of NJ feeds, the authors discontinue enteral feeds and begin PN, but monitor for resolution of symptoms to reinitiate tube feedings as soon as possible. The authors do not feed by means of an NJ tube if patients require pressor support, as it is difficult to determine tolerance to jejunal feedings in this population, and they are at increased risk of bowel ischemia. Under these conditions, the authors might attempt NG feedings while following residuals closely. High gastric residual volumes (greater than 300 mL) demonstrate that tube feedings are not tolerated. The authors initiate PN if patients develop high residuals or increased pain; as these symptoms resolve, the authors attempt NJ feeding again.

Surgical jejunal access

Jejunal feedings are efficacious in pancreatitis, and some patients who have severe disease may require long-term jejunal access until they recover fully [15,19]. Surgical jejunostomy tubes or gastrostomy tubes with jejunal extension are both ways in which long-term jejunal access is achieved. In the event of pancreatitis with infection requiring surgical debridement, it is reasonable to gain enteral access by surgically placing a feeding jejunostomy tube. Kudsk and colleagues [19] described success in placing feeding jejunostomies during exploratory laparotomy for complicated pancreatitis. In this prospective study, 11 patients underwent placement of a large bore
Red Robinson or small bore needle–catheter jejunal tube at the time of celiotomy. Two of the patients died of pancreatitis-related complications, with a jejunal leak in one of the nonsurvivors. Otherwise, there were no complications. The nine survivors were supported entirely with tube EN after surgery; none required PN. Weimann and colleagues [15] reported the use of needle catheter jejunostomies in 13 patients with acute pancreatitis who underwent surgery for necrosis or an acute abdomen. They initiated EN by the tube while tapering PN off. No severe tube-related complications occurred in any of the patients. One patient required replacement of the needle catheter jejunostomy after dislodgement during a subsequent laparotomy and washout [15]. These studies verify the safety of placing a jejunostomy in acute pancreatitis patients during surgery, and the ability to feed by this route after surgery [15,19]. Yoder and colleagues [16] investigated the safety and efficacy of jejunal feedings in pancreatitis patients discharged home. Thirty-three patients went home with jejunal feedings, either by means of NJ tubes or percutaneous endoscopic gastrostomy (PEG)-jejunostomy tubes. Of these patients, 77% achieved goal tube feeding rates, and 61% maintained weight or gained weight on this regimen. The authors concluded that home jejunal nutrition is safe and efficacious.

Parenteral nutrition

Even though EN provides the optimal route when feeding patients who have acute pancreatitis, intolerance can occur because of severity of disease, including pain, prolonged ileus, or gastric outlet obstruction caused by pancreatic pseudocyst. These complications often limit the use of enteral nutrition; therefore starting PN is reasonable in these cases. Patients classified as having mild or moderate pancreatitis (eg, Ranson’s scores no more than 2) should be treated with fluid resuscitation alone without nutrition support, because early PN provides no benefit (Fig. 1) [20]. Sax and colleagues [12] showed this in a randomized study comparing early PN with conventional therapy. Patients who received PN within 24 hours of diagnosis of pancreatitis experienced similar outcomes to those patients who received no early PN, with no significant differences in lengths of hospital stay, numbers of complications, or days to advancement to oral diet. Early PN was not beneficial, and in fact, the authors reported that early PN may have been harmful, as the patients randomized to this arm had a significantly higher rate of catheter-related sepsis than those patients who received conventional therapy. Therefore, a prudent approach is to wait several days before initiating PN in those who need it [17]. Patients who have severe pancreatitis requiring PN often need several days of fluid resuscitation and pain management before they tolerate any form of nutrition support [17,20]. Patients should get a NJ tube for enteral feeding after receiving adequate analgesia and correcting fluid and electrolyte abnormalities. If patients fail to tolerate enteral feeding because of pain or abdominal distention, or if other
contraindications to EN such as ileus or small bowel obstruction exist, it is appropriate to place a central line and begin PN therapy. The clinician must take special care in initiating nutrients by this route, as hyperglycemia and electrolyte abnormalities are common. The clinician should assess the patient’s nutritional status at the onset of onset of pancreatitis, because approximately one-third of pancreatitis patients present with malnutrition at baseline [21]. PN requires careful monitoring when initiated in patients with pre-existing malnutrition, as they are at high risk of refeeding syndrome and the resulting fluid and electrolyte abnormalities. The initial PN should contain half or less of the calculated goal kilocalories for the patient, with increasing kilocalories in subsequent bags of PN until the patient is at goal [17]. The recommended goal kilocalories for PN are 25 to 35 kcal/kg/d, with 1.2 to 1.5 g of protein/kg. Advancing PN kilocalories requires frequent monitoring of blood glucose levels, because hyperglycemia occurs because of impaired exocrine function [22]. To prevent hyperglycemia, add insulin to the first bag of PN in a dose of 0.1 units/g of dextrose, and increase the insulin as dextrose kilocalories increase to keep blood glucose levels no more than 150 mg/dL. Guidelines for glycemic control recommend adding 0.15 units of insulin per gram of dextrose to patients who remain hyperglycemic before starting PN [23,24]. Often, adding a continuous insulin infusion helps, especially if blood glucose levels are highly variable, or very high levels of insulin are required [24]. In addition to protein and

---

**Fig. 1.** Pancreatitis evaluation algorithm. (*From* McClave SA, Snider HL. Nutrition support in pancreatitis. In Kudsk KA, Pichard C, volume editors. From nutrition support to pharmacologic nutrition in the ICU. Vincent J, series editor. Updated in intensive care medicine. New York: Springer; 2002. p. 323; with kind permission of Springer Science and Business Media.)
dextrose, PN should contain fat to provide 20% to 30% of nonprotein kilocalories. Initiating fat mandates monitoring triglyceride levels. If triglycerides rise to greater than or equal to 400 mg/dL, it is appropriate to withhold fat and reinitiate it when levels decrease below this level. PN should be continued until the enteral route can be accessed, or oral intake is initiated [17].

**Enteral formula**

An issue in feeding enterally during pancreatitis is the choice of enteral formula. Prior approaches suggested that semielemental or elemental formulas were more appropriate for use in pancreatitis, as these minimally stimulated pancreatic secretions and were absorbed without pancreatic enzymes [17,25]. Some authorities [6], however, argue that because the GI tract absorbs these formulas in the proximal gut, they may not maintain healthy intestinal flora in the distal intestine. One trial addressed this issue by comparing a semielemental versus a polymeric (ie, intact protein and long-chain triglycerides) formula in 30 patients requiring EN with a diagnosis of acute pancreatitis. This trial randomized patients to receive either a semielemental formula or a polymeric formula by means of an NJ tube and found no significant difference between the groups in terms of tolerance (measured by pain visual analog scales, presence of bloating, and days of analgesics) or absorption (measured by 24-hour stool weights, the presence of protein in the stool, presence and quantity of steatorrhea, and number of stools per day). Patients receiving the semielemental formulas (versus the polymeric diet) experienced a shorter length of hospital stay (23 days plus 2 days versus 27 days plus or minus 1 day, respectively, \(P = .006\)) and showed significantly less weight loss (-1.3 plus or minus 1.1 kg versus -2.4 plus or minus .0 kg, respectively, \(P = .01\)). Neither tolerance nor numbers of infectious complications differed significantly between the groups. The authors hypothesized that a semielemental diet led to more favorable outcomes in these patients by better maintaining gut integrity than a polymeric formula, and thus preventing bacterial translocation. The authors concluded that only a larger trial testing the rate of GI-derived infections could confirm this theory [25].

**Benefits of enteral nutrition**

One of the leading theories to explain the benefit of EN is that EN maintains gut integrity and decreases intestinal permeability [5,13,17]. Results from a recent study [11] refute this theory, however. Eckerwall and colleagues [11] assessed the safety and efficacy of early NG feeding compared with PN in patients who had predicted severe acute pancreatitis. They also tested intestinal permeability by measuring excretion of polyethylene glycol. Blood levels of polyethylene glycol were not significantly different between the two groups except for day 3, when patients randomized to NG feeding had higher blood levels than the patients randomized to PN. They
concluded that EN does not decrease intestinal permeability and that the benefits of EN may be due to other reasons.

Another hypothesis of why EN feeding improves outcome proposes that EN feeding maintains gut mucosal immunity to decrease infectious complications [17,26]. Most of the body’s immune capability resides just below the mucosa of the intestine, where T and B cells produce large amounts of IgA. IgA is the body’s primary specific immune defense against bacterial antigens. Work in people and animals demonstrates that starving the gut alters T and B cell mass and function, therefore leading to a decrease in luminal levels of IgA. Also, the lack of enteral stimulation up-regulates gut endothelial adhesion marker expression and levels of inflammatory cytokines, both of which can alter immune system response to injury, resulting in more inflammatory complications [26]. When EN is used, all of these pathways for immunity are normalized and capable of normal responses to infectious challenges. When PN is used, however, lack of enteral stimulation impairs mucosal immunity. This perhaps explains why pancreatitis patients provided enteral feeding have lower complication rates than those patients fed with PN.

Potential therapies

Numerous potential nutritional therapies for acute pancreatitis exist, including supplementation of enteral feeding with probiotics and/or supplementation of formulas with omega-3 fatty acids, glutamine, and arginine. As inflammation and bacterial invasion appear to complicate acute pancreatitis, a proposed preventative treatment is through administration of prebiotics and probiotics to suppress pathogenic bacterial overgrowth and dampen the inflammatory response [27]. The few trials investigating enteral probiotic administration pancreatitis show promising results. Olah and colleagues [27,28] randomized patients who had acute pancreatitis to receive live cultures of *Lactobacillus plantarum* and substrate of oat fiber or to similar nutrients with heat-inactivated *Lactobacillus*. Forty-five patients completed the study, with 22 patients receiving active cultures and 23 patients receiving inactive cultures. The clinicians were blinded to randomization. All patients received enteral feeding via by means of NJ tube. Main outcome variables included organ failure, septic complications requiring surgery, length of hospital stay, and death. Results showed a significant reduction in the number of patients with septic complications requiring surgery with active treatment, as seven patients in the control group required surgery versus one patient in the treatment group ($P = .046$). Treatment reduced hospital length of stay compared with the control group (21.4 days versus 13.7 days), but this failed to reach statistical significance [27]. Another study by Olah and colleagues [28] randomized 62 enterally fed patients with severe acute pancreatitis to receive or not receive a mixture of lactobacilli preparation. Active treatment of patients was associated with a significantly reduced
incidence of complications. The incidence of SIRS and multi-organ failure combined was lower in the treatment group (8 versus 14, \( P < .05 \)).

No data exist to support the use of enteral supplementation with a combination of arginine, glutamine, and omega-3 fatty acids. Pearce and colleagues [29] randomized 31 patients who had pancreatitis to standard enteral feedings or an enteral diet containing glutamine, arginine, tributyrin, and omega-3 fatty acids. The primary outcome of the study was reduction of CRP by 40 mg/dL after 3 days of enteral feeding. Other clinical outcomes measured included length of stay, APACHE II scores, incidence of multi-organ failure, incidence of SIRS, and mortality. The investigators also measured carboxypeptidase activation peptide (CAPAP) levels throughout the study as an indicator of pancreatic damage. Fifteen patients received the study formula, and 16 patients received the standard formula. Results showed no significant reduction of CRP levels in the supplemented group or the control group after 72 hours of enteral feeding. In fact, when CRP levels were measured during an extension period up to a maximum of 15 days of enteral feeding, CRP levels significantly increased in the supplemented group compared with the control (\( P = .028 \)). Mortality dropped in the treatment group versus the control (no patients versus three patients, respectively), but this difference failed to reach statistical significance. CAPAP levels dropped significantly compared with baseline in both groups, including every day in the initial 3-day period of the study. The authors concluded that although the treatment group had higher levels of CRP and lower levels of CAPAP with no worsening in mortality, that a rise in CRP may reflect a better immune response. They suggested that immunonutrition required more trials with greater numbers of patients enrolled [29].

Two recent trials have shown promise using parenteral glutamine in acute pancreatitis [30,31]. Sahin and colleagues [30] compared 40 patients with pancreatitis with a Ranson’s score between 2 and 4 randomized to glutamine-supplemented PN or standard PN. Outcomes included nutritional parameters, occurrence of complications, and length of hospital stay. Of the nutritional parameters measured, most failed to reach a statistical difference between the two groups, except for serum transferrin levels, which increased by 11.7% in the treatment group and decreased by 12.1% in the control group. Complication rates dropped significantly in the treatment group versus the control group (10% and 40%, respectively, \( P < .05 \)). The authors concluded that parenteral glutamine supplementation provided a benefit to patients who have acute pancreatitis [30].

Ockenga and colleagues [31] showed that PN enriched with glutamine benefited patients with acute pancreatitis. This trial randomized 28 patients who had acute pancreatitis to PN-enriched glutamine or standard PN. Results showed a decrease in length of PN therapy in patients receiving the glutamine (\( P < .05 \)) and significant increases in levels of albumin and lymphocyte count compared with those not receiving glutamine. The authors concluded that PN enriched with glutamine provided some benefit
[31], but it remains unclear why. It is unknown whether enteral glutamine alone produces the same benefits, but no trials have compared patient groups with and without enteral glutamine.

Chronic pancreatitis

Chronic pancreatitis involves progressive loss of endocrine and exocrine pancreatic function, both of which must be addressed in the treatment. Pharmacologic therapy can manage losses of both pancreatic functions [32]. Because 70% to 90% of chronic pancreatitis is alcohol-related, however, compliance with medication regimens remains a challenge, especially for blood glucose control [33]. The American Diabetes Association classifies the loss of endocrine function as diabetes mellitus type IIIc [34]. Destruction of acinar cells results in reduced pancreatic secretion of insulin and glucagon, and impaired ability to tightly regulate blood glucose. Treatment with long-acting insulin for basal control of blood glucose is best, as patients use few injections and are more compliant.

Loss of exocrine function manifests as steatorrhea (fecal fat greater than 7 g/d) and weight loss. It is appropriate to initiate pancreatic enzymes, beginning with 1000 U (USP units) of lipase per meal and with snacks, titrating upwards as needed for this problem [32]. The best form of pancreatic enzymes are enteric-coated tablets or microspheres (Pancrease, Cotazym, and Promylin-HL16) [35]. Patients should consume smaller, more frequent meals, and not be fat-restricted when taking the pancreatic enzymes. If severe steatorrhea occurs despite adequate pancreatic enzyme supplementation, then consider restricting fat and administering medium-chain triglycerides. Patients who have severe steatorrhea risk deficiencies of fat-soluble vitamins and require these as supplements. Patients with alcoholic pancreatitis who continue to drink remain at risk of water-soluble vitamin depletion also. Clinicians should encourage abstinence, because studies show abstinence from alcohol improves outcomes [32].

Summary

Nutrition support in acute and chronic pancreatitis presents challenges related to choice of route, formula, and use of supplements. Evidence supports refraining from nutritional support during mild or moderate pancreatitis, because patients usually recover ability to take oral nutrition within 7 days. Evidence currently supports NJ or NG feeding in patients who have severe pancreatitis, with the caveat that some patients may be intolerant to feeding and develop bloating or increased pain. PN may be instituted in these patients, but only after failing a trial of EN. Enteral feeding by means of a surgically placed jejunal feeding tube is safe and effective. The optimal type of enteral feeding used remains controversial, but most literature supports the use of semielemental tube feedings. Management of chronic pancreatitis includes the use of insulin to control blood glucose.
levels and pancreatic enzymes to control steatorrhea. Management also includes supplementation with fat- and/or water-soluble vitamins based on the clinical situation. Research has changed the nutritional approach to this disease process dramatically.

References