

WJG 20th Anniversary Special Issues (18): Pancreatitis**Enteral nutrition in acute pancreatitis: A review of the current evidence**

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Abstract

The use of enteral feeding as part of the management of acute pancreatitis dates back almost two decades. This review describes the indications for and limitations of enteral feeding for the treatment of acute pancreatitis using up-to-date evidence-based data. A systematic review was carried out to analyse current data on the use of enteral nutrition in the management of acute pancreatitis. Relevant literature was analysed from the viewpoints of enteral *vs* parenteral feeding, early *vs* delayed enteral nutrition, nasogastric *vs* nasojejunal feeding, and early oral diet and immunonutrition, particularly glutamine and probiotic supplementation. Finally, current applicable guidelines and the effects of these guidelines on clinical practice are discussed. The latest meta-analyses suggest that enteral nutrition significantly reduces the mortality rate of severe acute pancreatitis compared to parenteral feeding. **To maintain gut barrier function and prevent early bacterial translocation, enteral feeding should be commenced within the first 24 h of hospital admission.** Also, the safety of nasogastric feeding, which eases the administration of enteral nutrients in the clinical setting, is likely equal to nasojejunal feeding. Furthermore, an early

low-fat oral diet is potentially beneficial in patients with mild pancreatitis. Despite the initial encouraging results, the current evidence does not support the use of immunoenhanced nutrients or probiotics in patients with acute pancreatitis.

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Key words: Acute pancreatitis; Enteral nutrition; Immunonutrition; Probiotics

Core tip: The application of enteral feeding in acute pancreatitis is much debated. This systematic review provides global insight for clinicians on how to incorporate enteral feeding in the management of acute pancreatitis. The timing, route and composition of enteral nutrition are discussed with up-to-date evidence-based data, and the latest relevant guidelines are also detailed. Importantly, **enteral nutrition significantly reduces mortality in severe acute pancreatitis compared to parenteral nutrition.** Furthermore, **early commencement of enteral feeding (within the first 24 h) is beneficial, and the safety of the nasogastric route seems to be equal to that of the nasojejunal route.**

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INTRODUCTION

The treatment of acute pancreatitis is purely symptomatic because there is no effective therapy to prevent the activation of inflammatory and proteolytic cascades. This vicious cycle of cell signalling is believed to be triggered by bacterial infection, predominately Gram-negative strains.

The most likely hypothetical source of the bacterial infection is the gastrointestinal tract. Bacterial translocation is caused by increased permeability in the gut and a consequent migration of macromolecules such as bacteria, endotoxins and antigens from the gastrointestinal tract to the portal system, mesenteric lymph nodes, liver, spleen and pancreas. This process leads to the stimulation of macrophages, circulatory neutrophils and granulocytes, and then the release of pro-inflammatory cytokine causes an inflammatory response. If the inflammatory response, which is initially part of the defence mechanisms of the host, is over-activated, it may turn into a self-destructive process. The unbalanced production of inflammatory mediators might lead to the development of systemic inflammatory response syndrome (SIRS), infectious pancreatic necrosis and ultimately multi-organ failure (MOF)^[11].

Severe acute pancreatitis (SAP) represents a typical model of septic syndrome due to a failure of the gut barrier. Hence, one of the main therapeutic goals in SAP is to maintain gut integrity to prevent bacterial and endotoxin translocation and improve the immune system of the gut. Recently, various clinical methods have aimed to prevent or decrease bacterial translocation. These methods include enteral feeding, with or without immunonutrition, as well as the use of probiotics during treatment. This paper provides a review of the available data in the evidence-based literature on the application of enteral feeding to acute pancreatitis. Furthermore, the authors discuss the evidence supporting early as opposed to delayed commencement of enteral feeding. The latest results on immunonutrition and probiotic use are also presented. In addition, the debate on the adequate route for enteral feeding is outlined. Finally, up-to-date guidelines and clinical practices are discussed.

ENTERAL NUTRITION VS PARENTERAL NUTRITION

Prolonged parenteral feeding carries numerous unfavourable side-effects such as atrophy and increased permeability of the gut mucosa. Furthermore, the lack of peristaltic stimulation results in hypomotility of the gut, and the stagnant bowel contents also cause significant changes in the intestinal microflora. Conversely, enteral feeding prevents the aforementioned atrophic changes as the uptake of nutrients in intestinal epithelial cells comes directly from the intestinal lumen. In addition, enteral feeding facilitates gut motility due the hyperosmolarity of the nutrients. These pathophysiological mechanisms protect against the overgrowth of abnormal intestinal flora and increased gut permeability, hence, potentially alleviating subsequent bacterial translocation.

In the first two randomised prospective trials, McClave *et al*^[2] and Nakad *et al*^[3] demonstrated that nasojejunal feeding is feasible, safe and beneficial in mild to moderate pancreatitis, or even severe pancreatitis. Altogether, 16 randomised controlled trials involving 847 patients

with acute pancreatitis compared enteral to parenteral feeding^[2,4-18]. Eleven of these studies randomised patients with severe pancreatitis or predicted SAP. All meta-analyses demonstrated a statistically significant reduction of infectious complications with the use of enteral nutrition^[19-21], except two studies which failed to confirm the beneficial effect of nasojejunal feeding^[6,16]. Hence, enteral nutrition has been established as a key component in the management of SAP^[22].

The meta-analyses published by Marik and Zaloga^[21] as well as McClave *et al*^[23] demonstrated that the use of enteral nutrition resulted in a significant reduction of infectious complications and length of hospital stay, as well as a trend toward reduced organ failure. However, these meta-analyses failed to confirm that enteral feeding could reduce mortality. In a meta-analysis published by Petrov *et al*^[20], more homogenous subgroups were compared, and altogether 202 patients with predicted SAP were included. The mortality rate in the enteral nutrition group was 4% (4/95) *vs* 15.9% (17/107) in the parenteral nutrition group, a statistically significant difference (RR = 0.32; 95%CI: 0.11-0.98; *P* = 0.03). Furthermore, Cao *et al*^[19] analysed six randomised controlled trials involving 224 patients and demonstrated a statistically significant decrease in mortality (OR = 0.251; 95%CI: 0.095-0.666, *P* = 0.005) and MOF (OR = 0.306; 95%CI: 0.128-10.736) in patients receiving enteral feeding. Finally, in a recent meta-analysis involving 381 patients from eight randomised controlled trials, Yi *et al*^[24] found that total enteral nutrition is significantly superior to total parenteral nutrition in patients with SAP. A statistically significant difference was observed in mortality (*P* = 0.001; OR = 0.37; 95%CI: 0.21-0.68), infectious complications (*P* = 0.004; OR = 0.46; 95%CI: 0.27-0.78), organ failure (*P* = 0.02; OR = 0.44; 95%CI: 0.22-0.88) and surgical intervention (*P* = 0.003; OR = 0.41; 95%CI: 0.23-0.74). However, no difference was detected in terms of length of hospital stay and duration of nutrition administration.

In summary, the data accumulated so far provide strong evidence of the benefits of enteral over parenteral nutrition in patients with SAP, because the risk of mortality was statistically less in patients given enteral compared to parenteral nutrition. Importantly, in the clinical setting, there are no specific contraindications for enteral nutrition. It can be performed safely even when SAP is complicated by fistulas, ascites or pseudocysts. The role of parenteral nutrition is limited in conditions such as severe ileus, when enteral nutrition can be restricted by paralysis. Nevertheless, enteral feeding in reduced amounts is still suggested in these cases, which provides the physiological benefits discussed above^[25] (Table 1).

EARLY VS DELAYED ENTERAL NUTRITION

Although the exact pathophysiological mechanisms of bacterial infection have not been determined, it seems unequivocal that it is a significant risk factor for pancre-

Table 1 Studies investigating the potential benefits of enteral *vs* parenteral feeding

Ref.	Year	Country/institution	No. of patients	Control arm	Benefits of enteral <i>vs</i> parenteral feeding
McClave <i>et al</i> ^[2]	1997	United States/University of Louisville, KY	30	Parenteral feeding	Cheaper, better glucose control
Kalfarentzos <i>et al</i> ^[4]	1997	Greece/University of Patras	38	Parenteral feeding	Lower complication rate, cheaper
Windsor <i>et al</i> ^[5]	1998	United Kingdom/St James's Univ Hospital London	34	Parenteral feeding	Decreased organ failure and complication rates
Paraskeva <i>et al</i> ^[7]	2001	Greece/Pireus General Hospital	23	Parenteral feeding	Lower surgical intervention rate
Oláh <i>et al</i> ^[9]	2002	Hungary/Petz A. Teaching Hospital, Gyor	89	Parenteral feeding	Less septic complications
Abou-Assi <i>et al</i> ^[9]	2002	United States/Virginia Univ. Hosp., RA	53	Parenteral feeding	Less septic complications, cheaper
Gupta <i>et al</i> ^[10]	2003	United Kingdom/Southampton General Hospital	17	Parenteral feeding	Shorter hospital stay, cheaper
Louie <i>et al</i> ^[12]	2005	Canada/University of Alberta	28	Parenteral feeding	Lower complication rate, better glucose control
Eckerwall <i>et al</i> ^[37]	2006	Sweden/Lund University Hospital	69	Parenteral feeding	Lower complication, MOF and mortality rates
Petrov <i>et al</i> ^[13]	2006	Russia/Nizhny Novgorod Hosp	22	Parenteral feeding	No significant difference

MOF: Multi-organ failure.

atic necrosis and the development of MOF during SAP. Importantly, bacterial translocation and pathogen overgrowth can be detected in the very early phase of acute pancreatitis. In a multicentre study, Besselink *et al*^[20] demonstrated that bacteraemia can be detected as early as day 7 and that infected necrosis can be detected on average 26 d after hospital admission. Furthermore, early bacterial invasion may aggravate SIRS, which in turn makes the patient even more susceptible to organ failure. This can result in a vicious cycle, because the development of organ failure frequently precedes bacterial infection. Hence, if bacterial translocation can be reduced or prevented through the maintenance of the intestinal barrier with enteral feeding, then it is reasonable to begin enteral feeding as early as possible.

A systematic meta-analysis published by Petrov *et al*^[27] involving 11 randomised controlled trials demonstrated that the risk of MOF, pancreatic infectious complications and mortality were significantly reduced in patients with acute pancreatitis who were enterally fed within the first 48 h of admission as opposed to parenteral feeding. Importantly, the differences were not statistically significant, if enteral nutrition was commenced 48 h after admission. In fact, a large amount of evidence-based data support the administration of enteral nutrition within 24 h of hospital admission^[28].

This has been further confirmed by Sun *et al*^[29] in a recently published randomised controlled trial. The authors investigated the effects of early administration of enteral nutrition on the immune function and clinical outcomes of 60 patients with SAP. The incidences of multiple organ dysfunction syndrome, SIRS and pancreatic infection, as well as the duration of stay in the intensive care unit, were significantly lower in the early administration group (commenced within 48 h of hospital admission) than in patients whose enteral feeding began on the eighth day of hospital stay. However, the authors did not report a difference in mortality between the two groups, which could have been due to the rela-

tively low number of patients in the study. In another recent randomised controlled trial involving 197 patients with SAP, Wereszczynska-Siemiatkowska *et al*^[30] found that enteral feeding within 48 h of admission significantly decreased the incidence of infective necrosis/fluid collection, respiratory failure, intensive care treatment and mortality compared to enteral feeding started after 48 h of hospital admission. While a clear trend towards a reduction in the rate of multi-organ dysfunction and surgical interventions was observed in patients with early enteral feeding, these differences were not statistically significant. An interesting aspect of the pathophysiology of acute pancreatitis was investigated in a pilot study by Sun *et al*^[31], which compared the incidence of intra-abdominal hypertension in 60 patients with early (within 48 h) or delayed (after day 8) administration of enteral nutrition. Intra-abdominal hypertension was more prevalent in patients with delayed administration of enteral nutrition. They also argued that higher intra-abdominal pressure (over 15 mmHg) may correlate with intolerance to feeding (Table 2).

The above findings were confirmed by a recent meta-analysis on the benefits of early administration of enteral nutrition commenced within 48 h of hospital admission^[32]. Based on 11 studies involving 775 patients, Li *et al*^[32] concluded that early enteral feeding was associated with significant reductions in all infections (OR = 0.38; 95%CI: 0.21-0.68; $P < 0.05$), catheter-related septic complications (OR = 0.26; 95%CI: 0.11-0.58; $P < 0.05$), pancreatic infection (OR = 0.49; 95%CI: 0.31-0.78; $P < 0.05$), hyperglycaemia (OR = 0.24; 95%CI: 0.11-0.52; $P < 0.05$), length of hospitalisation [mean difference -2.18; 95%CI: -3.48(-0.87); $P < 0.05$] and mortality (OR = 0.31; 95%CI: 0.14-0.71; $P < 0.05$). Importantly, a multi-centre randomised controlled trial that investigated 208 patients with predicted SAP has yet to report its results. The PYTHON trial, which was organised by the Dutch Pancreatitis Study Group, compared very early nasojejunal feeding (within 24 h of hospital admission) to standard

Table 2 Studies investigating the potential benefits of early *vs* late enteral feeding

Ref.	Year	Country/Institution	No. of patients	Control arm	Benefits of early <i>vs</i> late enteral feeding
Sun <i>et al</i> ^[29]	2013	China/Nanjing Medical University	60	Late enteral feeding	Lower infective complication, MOF and SIRS rates
Wereszczynska-Siemiatkowska <i>et al</i> ^[30]	2013	Poland/Medical University Bialystok	197	Late enteral feeding	Lower complication and mortality rates
Sun <i>et al</i> ^[31]	2013	China/Nanjing Medical University	60	Late enteral feeding	Lower intra-abdominal hypertension rate

MOF: Multi-organ failure; SIRS: Systemic inflammatory response syndrome.

practice (oral nutrition on demand, or if needed, enteral feeding after 72 h)^[33].

NASOGASTRIC VS NASOJEJUNAL FEEDING

While the placement of a nasogastric tube is a simple routine procedure that can facilitate the commencement of early enteral feeding, nasojejunal feeding requires an endoscopist or radiologist for tube placement, which may cause a delay in the start of early enteral feeding. Hence, nasogastric feeding seems to be the most feasible option in clinical practice. However, arguments against nasogastric feeding are based on the effects of stimulating pancreatic secretion and gastric emptying problems due to paralysis.

Eatock *et al*^[34] were the first to investigate these concerns in a prospective pilot study and found that nasogastric feeding is safe and well-tolerated. Then, two randomised controlled trials that compared nasogastric and nasojejunal feeding^[35,36] concluded that nasogastric nutrition at a slow rate of infusion was well tolerated, and there were no differences in the outcome measures (discharge, surgery and mortality rate) between the two groups. Another randomised controlled trial, which compared early nasogastric feeding to total parenteral nutrition in patients with predicted SAP^[37], demonstrated that enterally fed patients had significantly more total complications and pulmonary complications within the first 3 d. Two meta-analyses based on the above studies involving a total of 131 patients^[38,39] revealed no significant differences in mortality rate, length of hospital stay, infectious complications or MOF in SAP between nasogastric enteric feeding and conventional feeding. A recent meta-analysis based on three randomised trials involving 157 patients drew the same conclusion, that nasogastric feeding is not inferior to nasojejunal feeding^[40]. Although nasogastric feeding seems safe and well tolerated compared to nasojejunal feeding, more high-quality randomised controlled trials are needed to provide strong evidence, because the sample sizes in the studies conducted to date have been relatively low.

EARLY ORAL DIET

Regarding early oral feeding, a pilot study was the first

to demonstrate the feasibility of administration of an oral diet an average of 3 d after hospital admission^[41]. To determine if oral feeding is feasible for treating mild pancreatitis, Eckerwall *et al*^[42] randomised 60 patients with mild acute pancreatitis to compare the efficacy and feasibility of immediate oral feeding and traditional fasting. No differences were found in amylase values or the systemic inflammatory response between the two groups. This trial proved that immediate oral feeding is feasible and safe for treating mild acute pancreatitis. Furthermore, two randomised controlled trials demonstrated that it is not necessary to keep the patient on a liquid diet after acute mild pancreatitis^[43,44], as no detrimental effects were observed from a solid diet. In fact, a solid diet was associated with a shorter length of hospital stay^[44]. Hence, patients with mild acute pancreatitis can be started on a low-fat oral diet, although an initial period of fasting is still reasonable^[28].

IMMUNONUTRITION: GLUTAMINE SUPPLEMENTATION

Immunoenhanced nutrients involve substrates that modulate the activity of the host immune system and inflammatory response (Table 3). Immunonutrition formulas include glutamine, arginine, nucleotides and omega-3 fatty acids, as well as enteral nutrients supplemented by probiotics. Experimental studies have suggested that supplementation of enteral feed with glutamine or omega-3 fatty acids may reduce the severity of experimental acute pancreatitis^[45]. However, results have been rather moderate in the clinical setting. The four randomised controlled trials on this subject^[46-49] demonstrated that immunonutrition has some beneficial effects, such as a shortened length of hospital stay, reduced gut permeability and decreased plasma endotoxin levels, but no significant differences were found in terms of clinical outcomes. Furthermore, a meta-analysis published by Petrov *et al*^[50] based on three randomised controlled trials^[48-50] clearly demonstrated that immunonutrition, compared to standard enteral nutrition, was not associated with a significantly reduced risk of total infectious complications (RR = 0.82; 95%CI: 0.44-1.53; *P* = 0.53) or mortality (RR = 0.64; 95%CI: 0.20-2.07; *P* = 0.46).

As for glutamine supplementation, emerging evidence suggests that glutamine supplementation should be con-

Table 3 Anti-infective and immunomodulatory properties of immunonutrients

Anti-infective and immunomodulatory properties of immunonutrients
Reduced bacterial overgrowth
Maintenance of natural balance of intestinal flora
Reduced intestinal permeability
Reduced serum endotoxin levels
Antagonist effect against pathogenic bacteria
Prevent pathogenic bacterial adherence to intestinal mucosa
Bacterocidal and bacterostatic effect (lactic acid production)
Increased proportions of NK cells, T-lymphocytes, Ig-A producing plasma cells
Increased phagocytosis

sidered in patients with a critical illness associated with a catabolic response. A meta-analysis published by Asrani *et al.*^[51], which included 505 patients from 12 studies, demonstrated that glutamine supplementation resulted in a significantly reduced risk of mortality (RR = 0.30; 95%CI: 0.15-0.60; $P < 0.001$) and total infectious complications (RR = 0.58; 95%CI: 0.39-0.87; $P = 0.009$), but not the length of hospital stay (MD = -1.35; 95%CI: -3.25-0.56; $P = 0.17$). However, a clear advantage of glutamine supplementation was seen in patients who received total parenteral nutrition as opposed to enteral nutrition. We drew a similar conclusion when we investigated the effects of intravenous glutamine and early administration of enteral nutrition on SAP outcomes in a prospective randomised controlled trial with 45 patients^[52]. This study demonstrated that enteral nutrition supplemented by intravenous glutamine reduced the rate of complications (infected acute and post-necrotic peripancreatic fluid collections, infected pseudocysts and walled-off pancreatic necrosis), but the extent of reduction was not statistically significant. However, the mean hospital stay of the group that received intravenous glutamine and enteral feeding was 10.6 d, significantly shorter than that of the control group, who received enteral feeding alone (15.9 d; $P = 0.00104$).

IMMUNONUTRITION: PROBIOTIC SUPPLEMENTATION

Probiotics are live microorganisms that confer a health benefit to the host, are responsible for the maintenance of the natural balance among gut flora and possess an *in vivo* antagonist effect against pathogenic bacteria. The most widely used probiotic bacteria are *Lactobacillus* and *Bifidobacterium*, which can be isolated from human faeces or intestinal mucosa. Prebiotics are non-digestible food ingredients that are necessary for the propagation of probiotics. Prebiotics selectively stimulate the growth and activity of certain bacteria in the normal gut flora. Synbiotics are nutritional supplements containing both probiotics and prebiotics^[53].

Physiologically, some probiotics have been shown to have significant anti-infective and immunomodulatory properties. In addition, they can also prevent pathogenic

bacteria from adhering to the gut mucosa *via* their strong affinity for enterocytes. The complex bacteriostatic and bactericidal effects of probiotics are mainly due to the production of lactic acid and antimicrobial peptides.

Basic data from experimental pancreatitis models initially confirmed the beneficial effects of probiotics. Application of *Lactobacillus plantarum* (*L. plantarum*) reduced the rate of infective necrosis^[54], while *Saccharomyces boulardii* with concomitant ciprofloxacin lowered the histopathologic scores of acute necrotizing pancreatitis^[55]. Furthermore, probiotics reduced the severity of acute experimental pancreatitis, as well as bacterial translocation to extra-intestinal sites due to a reduction in duodenal bacterial overgrowth, the latter reducing late-phase mortality^[56,57].

Similarly, prospective randomised controlled trials have demonstrated beneficial effects of probiotics in acute pancreatitis. Karakan *et al.*^[58] showed that probiotics reduced the length of hospital stay in enterally fed patients when prebiotics were also applied. Supplementation of enteral nutrients with *L. plantarum* improved clinical outcomes, although control group patients in this study were fed parenterally^[17]. In clinical studies, the effects of lactic acid-producing bacteria in acute pancreatitis was investigated for the first time in our department^[59]. We found that the rate of pancreatic infectious complications was significantly lower in patients who received live *L. plantarum*. However, mortality was not significantly different between the two groups. We also investigated the use of a combination formula called "Synbiotic 2000" in patients with predicted severe pancreatitis^[60]. A decreasing trend in the rate of MOF and septic complications was detected, but these differences did not reach statistical significance. In a multicentre, double-blind, placebo-controlled trial called PROPATRIA organised by the Dutch Acute Pancreatitis Study Group^[61], 298 patients with predicted SAP were randomised to receive fibre-enriched enteral nutrition for 28 d with a multispecies probiotic preparation (Ecologic 641) or a placebo. The rate of infectious complications was comparable in both groups, and the mortality rate was higher in the synbiotic group (16% *vs* 6%), which was mainly due to bowel ischemia. Furthermore, organ failure and MOF were more common in the probiotic group (13.2% *vs* 4.9% and 3.0% *vs* 0.7%, respectively), although these differences did not reach statistical significance. Certainly, the synbiotic composition used in the PROPATRIA trial should not be used in critically ill patients^[62]. Interestingly, a recent retrospective analysis^[63] revealed that probiotic treatment had no apparent negative effect on patients with predicted SAP without initial organ failure, although the authors could not demonstrate the beneficial effects of probiotics in this subgroup of patients. However, the latest randomised controlled trial by Cui *et al.*^[64] supported the use of probiotics in combination with enteral feeding. The authors compared 70 patients with SAP who received parenteral feeding, enteral feeding or enteral feeding supplemented

with *Bifidobacterium*. They found that the incidence of upper gastrointestinal bleeding, infection and abscess were significantly lower in the probiotic group, and that the length of hospital stay was also significantly shortened in this group. Nevertheless, the results of this study, which was a single-centre study with a relatively low patient number, do not deny the conclusion of the PROPATRIA trial warranting the cautious application of probiotics in SAP. In fact, probiotics cannot be recommended for the management of acute pancreatitis based on the presently available evidence-based data^[65,66].

LATEST GUIDELINES

Recently, an International Consensus Guideline was published based on 11 previous guidelines of various societies, which was endorsed by the American Society for Parenteral and Enteral Nutrition^[67]. The committee established three categories for the level of evidence in their statements. Importantly, enteral nutrition is generally preferred over parenteral nutrition, and if feasible should be initiated first. Furthermore, continuous enteral nutrition infusion over bolus or cyclic administration is preferred. Similarly, the use of a nasogastric tube for administering enteral nutrition (and the lack of a need to position a postpyloric feeding tube) is also suggested by the committee. Finally, the committee recommends using enteral nutrition in the presence of pancreatic complications such as fistulas, ascites and pseudocysts.

Two other guidelines on the management of acute pancreatitis were published last year, which involved statements on nutritional support during acute pancreatitis. The American College of Gastroenterology guideline recommends enteral nutrition in SAP to prevent infectious complications, whereas parenteral nutrition should be avoided^[68]. The most detailed guidelines are based on the collaboration of the International Association of Pancreatology and the American Pancreatic Association involving 121 expert authors, which suggest that enteral nutrition is the preferable method of nutritional support in acute pancreatitis^[69]. As far as the composition of nutrients, either elemental or polymeric nutrition formulations can be used. In addition, nasogastric feeding is equally as effective as nasojejunal feeding, and the relevant evidence supports nasogastric feeding. However, a prospective randomised controlled trial called the Study on Nutrition in Acute Pancreatitis is currently underway, which will provide further evidence on nasogastric *vs* nasojejunal feeding (<http://clinicaltrials.gov/ct2/show/NCT00580749>).

Despite the relatively clear guidelines, everyday clinical practice does not necessarily follow these recommendations. In a recent study by Sun *et al*^[70], 43.3% of United States physicians utilised total parenteral nutrition for the treatment of pancreatitis and 36.5% used nasojejunal feeding. Moreover, private physicians use nasojejunal tube feeding in only 19.9% of cases. In a transatlantic survey of nutrition practices in the United Kingdom, the

Republic of Ireland and Canada, 54.2% favoured early feeding in SAP. There was a higher tendency towards enteral nutrition in university hospitals with the nasojejunal route being preferred^[71]. In Sweden, enteral feeding is a routine practice in 60% of the hospitals^[72]. The positive effects of a national consensus conference were demonstrated by Rebours *et al*^[73] in a study involving 176 hospitals in France. While enteral feeding was applied in 25% of the hospitals in 2001, it increased to 58% after the consensus conference.

CONCLUSION

Current evidence confirms that the administration of enteral nutrition is beneficial for the treatment of SAP. Enteral feeding reduces mortality, infectious complications and MOF. As far as the route of enteral feeding is concerned, nasogastric tube feeding is likely to be equally as effective as nasojejunal feeding in SAP. In terms of the timing of enteral nutritional support, relatively early administration within 48 or 72 h of hospital admission is suggested. However, current evidence does not support the application of immunoenhanced nutrients or probiotic supplements, and therefore they cannot be recommended for the management of acute pancreatitis at this time.

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