



ESPEN GUIDELINES

## ESPEN Guidelines on Enteral Nutrition: Pancreas<sup>☆</sup>

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Received 21 January 2006; accepted 21 January 2006

### KEYWORDS

Guideline;  
Clinical practice;  
Enteral nutrition;  
Oral nutritional  
supplements;  
Tube feeding;  
Pancreatitis;  
Undernutrition;  
Malnutrition

**Summary** The two major forms of inflammatory pancreatic diseases, acute and chronic pancreatitis, require different approaches in nutritional management, which are presented in the present guideline. This clinical practice guideline gives evidence-based recommendations for the use of ONS and TF in these patients. It was developed by an interdisciplinary expert group in accordance with officially accepted standards and is based on all relevant publications since 1985. The guideline was discussed and accepted in a consensus conference.

In mild acute pancreatitis enteral nutrition (EN) has no positive impact on the course of disease and is only recommended in patients who cannot consume normal food after 5–7 days. In severe necrotising pancreatitis EN is indicated and should be supplemented by parenteral nutrition if needed. In the majority of patients continuous TF with peptide-based formulae is possible. The jejunal route is recommended if gastric feeding is not tolerated.

In chronic pancreatitis more than 80% of patients can be treated adequately with normal food supplemented by pancreatic enzymes. 10–15% of all patients require nutritional supplements, and in approximately 5% tube feeding is indicated.

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**Abbreviations:** EN, enteral nutrition (both oral nutritional supplements and tube feeding); IU, international units; PEG, percutaneous endoscopic gastrostomy; MCT, medium chain triglycerides; ONS, oral nutritional supplements; TF, tube feeding

<sup>☆</sup>For further information on methodology see Schütz et al.<sup>77</sup> For further information on definition of terms see Lochs et al.<sup>78</sup>

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<sup>☆☆</sup>The authors of the DGEM (German Society for Nutritional Medicine) guidelines on enteral nutrition in pancreatitis are acknowledged for their contribution to this article.

**Summary of statements: Acute pancreatitis**

Subject	Recommendations	Grade <sup>77</sup>	Number
<b>Indications</b>			
Mild acute pancreatitis	Enteral nutrition is unnecessary, if the patient can consume normal food after 5–7 days.	B	1.3
	Enteral nutrition within 5–7 days has no positive impact on the course of disease and is therefore not recommended.	A	1.6
	Give tube feeding, if oral nutrition is not possible due to consistent pain for more than 5 days.	C	1.6
Severe necrotising pancreatitis	Enteral nutrition is indicated if possible.	A	1.3
	Enteral nutrition should be supplemented by parenteral nutrition if needed.	C	1.3
	In severe acute pancreatitis with complications (fistulas, ascites, pseudocysts) tube feeding can be performed successfully.		1.8
<b>Application</b>			
	Tube feeding is possible in the majority of patients but may need to be supplemented by the parenteral route.	A	1.4
	Oral feeding (normal food and/or oral nutritional supplements) can be progressively attempted once gastric outlet obstruction has resolved, provided it does not result in pain, and complications are under control. Tube feeding can be gradually withdrawn as intake improves.	C	1.10
Severe pancreatitis	Use continuous enteral nutrition in all patients who tolerate it.	C	1.7
<b>Route</b>			
	Try the jejunal route if gastric feeding is not tolerated.	C	1.4
	In case of surgery for pancreatitis an intraoperative jejunostomy for postoperative tube feeding is feasible.	C	1.7
	In gastric outlet obstruction the tube tip should be placed distal to the obstruction. If this is impossible, parenteral nutrition should be given.	C	1.8
<b>Type of formula</b>			
	Peptide-based formulae can be used safely.	A	1.5
	Standard formulae can be tried if they are tolerated.	C	1.5

Grade: Grade of recommendation; Number: refers to statement number within the text.

**Summary of statements: Chronic pancreatitis**

Subject	Recommendations	Grade <sup>77</sup>	Number
<b>General</b>	Adequate nutritional therapy as well as pain treatment may have a positive impact on nutritional status. Caloric intake is increased after an attenuation of postprandial pain.	C	2.4

<b>Indications</b>	More than 80% of patients can be treated adequately with normal food supplemented by pancreatic enzymes.	B	2.4
	10–15% of all patients require oral nutritional supplements.	C	2.4
	Tube feeding is indicated in approximately 5% of patients with chronic pancreatitis.	C	2.4
<b>Specific contraindications</b>	Stenosis of duodenum	C	2.5

Grade: Grade of recommendation; Number: refers to statement number within the text.

## 1. Acute pancreatitis (AP)

**Preliminary remarks:** The management of acute pancreatitis (AP) differs according to its severity. Classified by the Atlanta criteria<sup>1</sup> approximately 75% of the patients have mild disease with a mortality rate below 1%.<sup>2</sup> Mortality increases up to 20% if the disease progresses to its severe necrotizing form<sup>3–8</sup> and in the most severe cases mortality can rise to 30–40%.<sup>7,8</sup> Severe AP with its related systemic inflammatory response (SIR) causes increased metabolic demands and may progress to multiorgan disease (MOD). Using imaging methods and laboratory parameters, progression can be predicted. Until recently, EN, either orally or by tube, was believed to have a negative impact on the progression of the disease due to stimulation of exocrine pancreatic secretion and the consequent worsening of the autodigestive processes of the pancreas. Even though nutritional deficits are frequent in severe pancreatitis, nutrition as a part of therapy was neglected for a long time. Even now, few nutritional studies in this condition have been published.

### 1.1. What influence does acute pancreatitis exert on nutritional status and on energy and substrate metabolism?

**Mild pancreatitis has little impact on nutritional status or metabolism. In severe necrotising pancreatitis energy expenditure and protein catabolism are increased (IIa).**

**Comment:** In mild acute pancreatitis the clinical course is usually uncomplicated and patients can consume normal food, low in fat (<30% of total energy intake [vegetable fat are preferred]), within three to seven days. The disease has little impact on nutritional status or on energy and substrate metabolism. It is not clear whether this is also true in the presence of pre-existing undernutrition, although it is probably important to meet nutri-

tional requirements in such cases by whatever means are most appropriate.

Both specific and non-specific metabolic alterations occur in AP<sup>9</sup> (Ib). Basal metabolic rate increases due to inflammatory stress and pain, leading to enhanced total energy expenditure.<sup>9</sup> In severe necrotising pancreatitis, 80% of all patients are catabolic<sup>9</sup> (Ib), with high energy expenditure and enhanced protein catabolism<sup>10</sup> (IIa). The negative nitrogen balance can be as much as 40 g/day<sup>11,12</sup> and can have a deleterious effect on both nutritional status and disease progression. In one trial, patients with a negative nitrogen balance had a ten-fold higher mortality than those with a normal balance.<sup>13</sup> This conclusion has to be treated with caution since no study has been stratified according to disease severity, and the relation between nitrogen balance and progression might, therefore, merely reflect the severity of disease.

Starvation for more than seven days should always be avoided, since protein and energy catabolism induces undernutrition—and probably worsens the prognosis. It has been shown, that as little as five days of conservative therapy without nutritional support in previously healthy men suffering from severe pancreatitis results in severe undernutrition, water retention and decreased muscle function proportional to decreased protein stores.<sup>14</sup>

Hyperlipidaemia occurs frequently in acute pancreatitis.<sup>15,16</sup> It is not clear whether this is a consequence of disease or due to pathogenic factors or a combination of both<sup>17</sup> (Ib). The latter seems more likely, since serum lipids normalize during recovery from AP. Severe hyperlipidaemia itself may be the sole cause of AP. It is a particular problem in the most severe cases, reflecting severe disturbances of fat metabolism secondary to sepsis and treatment.

The enhanced metabolic rate and protein catabolism necessitate an increased energy intake from both fat (30%) and carbohydrates (50%). 1.0–1.5 g

proteins are usually sufficient. Carbohydrates are the favoured source of calories, since administration is easy, although hyperglycaemia, secondary to insulin resistance and in some cases islet cell damage, has to be avoided, placing a limit on the rate of administration of glucose and, in some cases, necessitating the use of insulin<sup>10</sup> (IIa).

### 1.2. Does nutritional status influence outcome?

***Although not investigated in this context, severe undernutrition is likely to affect outcome negatively.***

**Comment:** Since there are no studies addressing this issue, the question cannot be properly answered for AP. It has to be considered that undernutrition is a well-known risk factor for more complications and higher morbidity in other diseases. It also has to be considered that undernutrition is known to occur in 50–80% of chronic alcoholics and that alcohol is a major aetiological factor in acute pancreatitis (30–40% of patients).<sup>18</sup> Overweight, with a high body mass index is also associated with a poorer prognosis.

### 1.3. Is EN indicated in acute pancreatitis?

***In mild acute pancreatitis EN is unnecessary, if the patient can consume normal food after five to seven days (B).***

***In severe necrotising pancreatitis, EN is indicated if possible (A). This should be supplemented by parenteral nutrition if needed (C).***

**Comment:** Parenteral nutrition (PN) has been the standard way of meeting nutritional requirements since it avoids pancreatic stimulation and improves nutritional status. A positive benefit has, however, not yet been confirmed in trials. There are two investigations in mild to moderate pancreatitis comparing parenteral to no nutritional support<sup>19</sup> (Ib) or to TF<sup>20</sup> (Ib). In the trial by Sax et al. no difference in mortality or complication rate between the two regimens could be demonstrated.<sup>19</sup> Catheter induced septicaemias as well as hyperglycaemia occurred significantly more often in the PN group. McClave et al., in a prospective randomised controlled study, compared early EN via a jejunal tube to PN in patients with mild to moderate pancreatitis.<sup>20</sup> Early EN was initiated within 48 h after admission to hospital. No difference in the investigated parameters was found, although PN was found to be four times more expensive. All patients in both groups survived.

Windsor et al.<sup>21</sup> (Ib) compared PN with EN in patients with mild to moderate (total peripheral PN vs. ONS) and severe pancreatitis (total central PN vs. TF). The systemic inflammatory response

syndrome (SIRS) was significantly attenuated in all enterally fed patients. Sepsis and multiorgan failure as well as incidence of surgery were reduced. Whereas two patients died in the PN group, no death occurred in the EN group. Major weaknesses of this study are the small number of patients with severe pancreatitis and the marked differences in nutrient intake between the enteral and the parenteral groups.

A further trial by Powell et al.<sup>22</sup> (Ib) could not confirm these findings. They compared early TF in patients with severe AP to patients without nutritional support. One possible explanation could be the different patient populations studied. In the Windsor group the mean APACHE II was 8 in the EN group and 9.5 in the PN group.<sup>21</sup> In the Powell series APACHE II scores were 13 or more.<sup>22</sup>

In a randomised prospective controlled trial, comparing EN (TF) vs. PN in patients with severe pancreatitis Kalfarentzos et al.<sup>23</sup> (Ib) scored less than half of those studied, but, in the remainder, mean APACHE II scores were 12.7 in the EN group and 11.8 in the PN group. EN was well tolerated and was associated with fewer septic and other complications than PN as well as cost were more than three times less.

In recent years it has become clear, that PN related complications have often been the consequence of overfeeding or even just catheter sepsis.<sup>24</sup> Van den Berghe et al. showed, irrespective of the route of nutritional support, that the control of hyperglycemia with insulin reduced mortality in critically ill patients.<sup>25</sup> Hyperglycaemia may occur with EN as well as PN.

Several studies in patients with trauma, thermal injury and major gastrointestinal surgery have shown a reduction in septic complication with EN<sup>26,27</sup> (Ib) which also helps to maintain mucosal function and limit absorption of endotoxins and cytokines from the gut.<sup>28,29</sup> In animals with induced pancreatitis, EN prevented bacterial translocation,<sup>30</sup> but whether this occurs in patients with AP is still unclear.<sup>31</sup>

Recent evidence has encouraged a much greater use of EN than PN in severe acute pancreatitis, whenever possible. EN, by down-regulating splanchnic cytokine production and modulating the acute phase response, reduces catabolism and preserves protein.<sup>21</sup>

Abou-Assi et al.<sup>32</sup> studied 156 patients with AP over 12 months. During the first 48 h all patients were treated with i.v. fluid and analgesics. 87% of patients had mild, 10% moderate, and 3% severe disease. Those who improved went on to normal food as soon as possible. The non-responders were randomized to receive nutrients either by a

naso-jejunal tube or by PN. 75% of the initially enrolled patients improved with the oral regimen and were discharged within four days. 54% of the TF group ( $n = 26$ ) and 88% of the PN group ( $n = 27$ ) received adequate energy intake. The patients in the TF group were fed for a significantly shorter period (mean 6.7 days vs. 10.8 days [PN]), and had significantly fewer metabolic and septic complications. Hyperglycemia, requiring insulin therapy, occurred more frequently in the parenterally fed patients. Despite fewer complications with TF, mortality was similar in the two groups. The authors concluded that hypocaloric TF is safer and less expensive than parenteral feeding and bowel rest in patients with AP. Jejunal TF may also reduce the frequency of pain relapses in patients with mild to moderate AP.<sup>33</sup> A recent meta-analysis of TPN versus TF in patients with acute pancreatitis by Marik and Zaloga concluded that TF should be the preferred route of nutritional support in patients with AP, because EN was associated with a significantly lower incidence of infections, reduced rate of surgical interventions and a reduced length of hospital stay. There were no significant differences in mortality and non-infectious complications.<sup>34</sup>

There are no studies comparing TF to oral nutrition.

#### 1.4. Is TF possible in practice and what is the preferred route of feeding?

**TF is possible in the majority of patients with AP (Ia) but may need to be supplemented by the parenteral route (A).**

**If gastric feeding is not tolerated the jejunal route should be tried (C).**

**Comment:** Four prospective studies have shown that jejunal delivery is possible in most patients with AP<sup>20,35–37</sup> (Ib). Rarely, proximal migration of the feeding tube and a subsequent pancreatic stimulation can aggravate AP.<sup>38</sup> If the jejunal tube cannot be placed blindly or with the aid of fluoroscopy, adequate endoscopic placement is usually feasible. In a recent study,<sup>39</sup> naso-gastric feeding proved safe, since little difference in pain, analgesic requirements, serum CRP concentrations, or clinical outcome was seen between the two methods. It seems, AP could not have been very severe, if gastric emptying was maintained.

Although TF appears to have been possible in most prospective studies of TF in acute pancreatitis, in more general studies, dealing with larger patient groups including all treated patients, this was not the case. Oleynikow et al. reported, that

TF was not possible in most (25 out of 26) patients with severe AP (mean APACHE II 17.2 and mean Ranson score 4.3 on admission) most probably due to severe retroperitoneal inflammatory changes.<sup>40</sup>

TF is also possible in the presence of ascites and pancreatic fistulas. Neither intrajejunally delivered glucose, protein nor fat stimulate the exocrine pancreas if they are infused alone<sup>41</sup> (III). If fat is administered, serum triglycerides should be monitored regularly. Values below 10–12 mmol/l are tolerated but serum lipid levels should ideally be kept within normal ranges.

#### 1.5. Which formulae should be used in AP?

**Peptide-based formulae can be used safely in AP (A).**

**Standard formulae can be tried if they are tolerated (C).**

**Comment:** Most trials (human and animal) have been carried out using peptide-based formula, which can therefore be recommended for feeding.<sup>41–47</sup> Whether standard formulae can be used safely or whether immune-modulating formulae have an additional impact on the course of the disease remains unclear (IV). Today it is common to start with a standard formula and if this is not tolerated a peptide-based formula is tried.

#### 1.6. How should nutritional support be given to patients with mild pancreatitis?

**In mild pancreatitis EN within five to seven days has no positive impact on the course of disease and is therefore not recommended (A). Oral food intake should be tried as soon as possible.**

**If oral nutrition is not possible due to consistent pain for more than five days, TF should be given (C).**

**Comment:** In mild pancreatitis fluid and electrolytes are initially given parenterally. When pain ceases, oral food intake is initiated.

Patients with mild pancreatitis can be fed orally after a short period of starvation if pain has ceased and amylase and lipase values are decreasing<sup>48</sup> (Ib). Oral refeeding with a diet rich in carbohydrates and protein and low in fat (<30% of total energy intake) is recommended, but no clinical trials on this are available. If the diet is well tolerated, oral nutrition can be increased continuously. Specific products do not have to be used.

#### 1.7. How should nutritional support be given to patients with severe pancreatitis?

**Early EN improves the course of severe pancreatitis (III). Continuous EN is therefore recommended in all patients who tolerate it (C).**

***In case of surgery for pancreatitis an intraoperative jejunostomy for postoperative TF is feasible (C).***

**Comment:** In severe pancreatitis EN should be initiated as early as possible, particularly when alcoholism, with its associated undernutrition, is the cause<sup>18</sup> (Ib). Water, electrolyte and micronutrient requirements must be met by the intravenous route and decreased gradually as the enteral supply increases. According to expert opinion EN should be provided over 24 h via a pump assisted jejunal tube, but the evidence base for this statement is weak. It has also been recommended that TF should be supplemented by PN if requirements cannot be met enterally or there are contraindications to TF (e.g. prolonged ileus).

Two recent studies using special formulae have been reported. In a small study<sup>29</sup> comparing a glutamine rich, multifibre formula with a standard fibre-containing formula, there was a beneficial effect of the glutamine rich formula on the recovery of IgG, IgM proteins and a shortening of the disease. A second study examined the efficacy of the tube administration of the probiotic lactobacillus plantarum 299v in patients with severe AP.<sup>49</sup> 22 patients received live bacteria with oat-fibre, and 23 patients the same formula with heat-killed bacteria. In the group with live bacteria, only one patient developed a septic pancreatic complication requiring surgery, compared to the control group in which seven patients developed such complications ( $P < 0.023$ ). These observations are interesting but at present it is not possible to recommend this approach on the basis of this small study. Larger trials are required to confirm these results.

Thiamine deficiency and therefore increased requirements are common, especially in alcoholic patients. Extra supply by the intravenous route is therefore recommended.

It has been shown in one study<sup>50</sup> that patients with severe AP are selenium deficient and therefore benefit from additional selenium supply. These results should be confirmed by other studies; on the other hand, when choosing an EN formula it is advisable to check whether it contains selenium.

The switch from TF to oral nutrition should be early and gradual according to the clinical situation and course of the disease (IV). A general recommendation is not possible.

Septic complications are important causes of increased resting energy expenditure.<sup>10,11</sup> In AP, since the Harris Benedict formula has not proved adequate to estimate energy expenditure with accuracy indirect calorimetry should be performed,

if possible (IIa). The results must be interpreted with caution, however, as indirect calorimetry shows only part of the picture. At best, it evaluates the patient's basic requirements and therefore helps in formulating an appropriate prescription. Overfeeding during the acute phase should be avoided. Afterwards the calories can gradually be increased until full requirements are met.

**1.8. How is nutritional management affected by complications?**

***In severe AP with complications (fistulas, ascites, pseudocysts) TF can be performed successfully. In gastric outlet obstruction the tube tip should be placed distal to the obstruction. If this is impossible, parenteral nutrition should be given (C).***

**Comment:** Postoperative TF was successful, in one small study.<sup>36</sup> There are no controlled studies of feeding AP patients with gastric outlet obstruction so that no general recommendation can be given. Nutrition support has to be planned according to the clinical situation and course of the disease.

**1.9. Are there contraindications to EN?**

***There are no specific contraindications known for EN.***

**Comment:** Since there are no prospective studies, nutritional therapy is given according to the clinical situation. Expert opinion advises that EN should always be tried if an adequate intake of normal food is not possible. In the presence of gastric distension, double lumen tubes, allowing simultaneous feeding into the jejunum and aspiration of gastric contents, have proved to be of value (IV). Maintenance of mucosal integrity is regarded as important. Although, in humans, changes in mucosal integrity (villous architecture or intestinal permeability) have rarely been assessed. Neither of these parameters has been shown to be associated with bacterial translocation although there is some evidence to implicate the gut origin of sepsis in acute pancreatitis.<sup>51</sup> A low volume jejunal delivery of enteral formula, supplemented by PN should be considered.<sup>27,52</sup> In 1990 Kudsk et al. reported eleven patients in whom needle catheter jejunostomy was placed during laparotomy for complications of severe pancreatitis, with only one leak around the tube.<sup>36</sup> Hernandez-Aranda et al. found no differences between groups of patients who received postoperative PN or EN via jejunostomy.<sup>53</sup> However, jejunostomies should be placed only, when the risk of leak or tube dislodgement is minimal.

### 1.10. How and when can patients be weaned from TF?

**Oral feeding (normal food and/or ONS) can be progressively attempted once gastric outlet obstruction has resolved, provided it does not result in pain, and provided that complications are under control. TF can be gradually withdrawn as intake improves (C).**

**Comment:** There are currently only two studies investigating initiation of an oral diet.<sup>33,48</sup> In the study of Levy et al., 21% of patients experienced pain relapse on the first and second day of refeeding. Serum lipase concentration  $>3 \times$  the upper limit of the normal range and higher Balthazar's CT scores at the onset of refeeding were identified as risk factors for pain relapse<sup>48</sup> (IIa).

## 2. Chronic pancreatitis

**Preliminary remarks:** Alcohol is the etiological factor in 60–70% of patients with chronic pancreatitis. Other causes of chronic pancreatitis are much less common (pancreatic duct obstruction, pancreas divisum, hereditary or tropical pancreatitis). Between 15% and 35% of patients have no apparent underlying diseases (idiopathic chronic pancreatitis).<sup>54</sup>

The morphological changes include oedema, acute inflammation and necrosis, superimposed on the background of chronic changes that include fibrosis, calcification, inflammation, and loss of exocrine tissue.<sup>55,56</sup> During the course of chronic pancreatitis, enzyme secretion is gradually decreased, resulting in maldigestion with steatorrhea, and azotorrhea when more than 90% of pancreatic tissue is destroyed. At this stage of the disease, diabetes will also develop due to the loss of insulin producing beta cells in the pancreas.

### 2.1. How does chronic pancreatitis influence nutritional status and metabolism?

**Protein energy undernutrition occurs frequently in the terminal phase of chronic pancreatitis, partly due to pain induced anorexia and continuing alcohol abuse.**

**30–50% of patients with chronic pancreatitis have increased resting energy expenditure.**

**Comment:** Abdominal pain, malabsorption and diabetes mellitus are complications, which have a negative impact on nutritional status in chronic pancreatitis. If exocrine (lipase and trypsin) and endocrine pancreatic function are reduced by more than 90%, maldigestion and diabetes mellitus

result. In the early stages of the disease digestion of fat is more affected than that of carbohydrate and protein<sup>57,58</sup> (IIa) and results in steatorrhea, although, as function deteriorates, and lipase and trypsin secretion decline further, azotorrhea may also develop.

In 30–50% of patients with a chronic pancreatitis, enhanced resting energy expenditure occurs.<sup>59</sup> Deficiencies in vitamins A, D, E and K<sup>60,61</sup> result from steatorrhea. Specific deficiencies in Ca, Mg, Zn, thiamine and folic acid have also been reported (IIa).

### 2.2. Does nutritional status affect outcome?

**The degree of undernutrition probably correlates with complications and has a negative impact on outcome (IV).**

**Comment:** There are no specific studies investigating this issue.

### 2.3. What are the goals of nutritional therapy?

**The main goal is to influence malabsorption and prevent undernutrition.**

**Comment:** Late in the course of chronic pancreatitis, weight loss is often seen, due to a reduced calorie intake (pain, persistent alcohol intake) and malabsorption of macronutrients. Undernutrition is, therefore, common in patients with chronic pancreatitis and its severity is one of the major factors predicting complications and outcome.

### 2.4. What are the treatment options?

**More than 80% of patients can be treated adequately with normal food supplemented by pancreatic enzymes (B).**

**10–15% of all patients require ONS (C).**

**TF is indicated in approximately 5% of patients with chronic pancreatitis (C).**

**Adequate nutritional therapy as well as pain treatment may have a positive impact on nutritional status. Caloric intake is increased after an attenuation of postprandial pain (C).**

**Comment:** The standard therapeutic measures in chronic pancreatitis include abstinence from alcohol and pain control (IIa). With these measures, improvement in nutritional status can generally be achieved. If analgesics are required, they should be consumed before the meal, since a reduction in postprandial pain results in an increased food intake. Whether alcohol abstinence improves outcome is difficult to say, since data are controversial.<sup>54</sup> Exocrine pancreatic insufficiency is manifest by steatorrhea (faecal fat excretion  $>7$  g/day).

With a reduced fat diet (0.5 g/kgBW/day), partial symptom control is possible (IIa). Pancreatic enzymes taken with meals with a normal fat content (30% of total energy intake) are the mainstay of treatment.

Glucose intolerance occurs in 40–90% of all cases with severe pancreatic insufficiency (IIa). In 20–30% of all patients manifest diabetes occurs, associated with impaired glucagon release.<sup>62–64</sup> Glucagon secretion is also reduced in type 1 diabetes after a few years, impairing counter-regulation and making patients more susceptible to hypoglycaemia during insulin treatment.

Normal food is sufficient in most cases, but, if calorie intake is low, whole protein ONS and pancreatic enzymes can be provided. If they are not well tolerated, one should try peptide-based ONS, which are probably more efficient than whole-protein ONS (III). The palatability of peptide supplements is low and compliance is poor.

Reduction in steatorrhoea and an adequate intake of energy are the most important principles of nutrition therapy in chronic pancreatitis.

Treatment of exocrine pancreatic insufficiency starts with nutritional counselling as well as substitution of pancreatic enzymes.<sup>65</sup> Frequent small meals are important in order to achieve an adequate intake. The diet should be rich in carbohydrates and protein, although carbohydrate intake can cause problems with intercurrent diabetes. A protein intake of 1.0–1.5 g/kg is sufficient and is well tolerated. 30% of calories can initially be given as fat, which is well tolerated, especially in the case of vegetable fat.

If adequate weight gain cannot be achieved and steatorrhoea is persistent, then medium chain triglycerides (MCT) can be administered<sup>38,66</sup> (III). Due to lipase independent absorption MCT can be recommended. MCTs however, have a lower energy density (8.3 kcal/g), are not very palatable, and may induce side effects such as abdominal pain, nausea and diarrhoea. The diet should be low in fibre, since fibres absorb enzymes and lead to a reduced intake of nutrients. Fat-soluble vitamins (vitamin A, D, E, K) as well as other micronutrients should be supplemented if clinical deficit is apparent.<sup>62</sup>

A lot of enzyme supplements are available that differ in enzyme content and pharmacological preparation.<sup>67–69</sup> An adequate intake of enzyme products is crucial<sup>67,70–72</sup> (Ib). In cases of therapeutic resistance despite an adequate diet, good compliance, correct pharmacological preparation and dosage of enzyme supplements, then H<sub>2</sub>-antagonists or proton-pump-inhibitors can be added.<sup>70,73,74</sup>

The role of enzyme products to manage pain is controversial.<sup>75,76</sup>

EN is indicated if the patients cannot ingest sufficient calories (in pain or pyloro-duodenostenosis due to an enlarged pancreatic head or pseudocyst formation, if weight loss continues despite apparently adequate normal food, in the presence of acute complications (acute pancreatitis or fistulas), or prior to surgery. It is recommended that EN be delivered via a jejunal tube (IV). For long-term therapy a percutaneous endoscopic gastrostomy (PEG) with a jejunal tube is probably best. A peptide or amino acid based formula is recommended, given overnight (IV). There are no long term studies available showing the efficacy of this approach which is based on clinical experience.

PN is only indicated when EN is not possible e.g. in severe stenosis of the duodenum prior to surgery. There are no published data on patients fed intravenously for a longer period.

## 2.5. Are there specific contraindications to normal food or EN (ONS & TF) in chronic pancreatitis?

**Except for stenosis of the duodenum, there are no contraindications to normal food or EN (C).**

**Comment:** There are no data available concerning this topic.

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