Parenteral versus Enteral Nutrition: Can We Get Rid of the Myths?

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Introduction

Modern clinical science asks for prospective randomized trials to assess the efficacy of treatment modalities. These trials can then be combined in a meta-analysis which, depending on the quality of the studies (level of evidence: $1 = $best, $2, 3$ or $4 = $worst), can lead to recommendations with varying grades. A grade-A recommendation relies on studies of the highest quality (prospective, randomized, double-blind, sufficient power, homogenous populations, single modality intervention, hypothesis testing, clinical endpoints), and is therefore stronger than grade-B, C and D recommendations, which rely on studies of decreasing quality [1]. A grade-D recommendation is in fact no recommendation, because it relies on a nonrandomized trial or on animal or in vitro studies. An important meta-analysis failed to demonstrate the efficacy of supplemental total parenteral nutrition (TPN) in a clinical setting [1], and this study and similar other studies have often led to a nihilistic approach, implying that no serious efforts are made to nourish patients. ‘It has never been demonstrated that nutritional support is of any benefit for intensive care unit (ICU) patients’ is what can often be heard among intensive care specialists.

Nevertheless every clinician in his right mind will acknowledge the fact that a distinct but small group of patients, who cannot eat normally or do not absorb adequately, owe their life to artificial nutritional support. Proof for this is difficult to achieve, because admittedly the group is relatively small and because a prospective randomized clinical trial (PRCT) with a nutritional support and a starved control group in most institutions would not receive the approval of the medical ethical committees. The patients that benefit
from nutritional support generally require artificial nutrition for a long period of time. In this group it becomes obvious that illness in combination with a long period of starvation (3–8 weeks) would have led to a 100% mortality. However, most clinical studies intending to show the efficacy of artificial nutritional support regard the effect of nutrition over a period of no longer than 7–10 days. The treatment effect over such short periods of time can only be small. The physiology is such that the human body can easily sustain trauma of limited size, if the organism is previously well fed and without major previous organ failure. Additional nutritional support may therefore under these circumstances have only modest or no benefit, and only add to the complications of the nutritional intervention. These considerations should also be taken into account when estimating the relative value of parenteral versus enteral nutrition. To adequately assess the merits of the administration route of artificial nutrition, it would be preferable to rely on evidence. As the quality of the evidence is not solid (see above), we have to rely on less than level-1 evidence and as a consequence formulate less than grade-A recommendations. An alternative approach is to rely on clinical facts and judgment and then to try to support the clinical recommendations with evidence from the literature.

**Why is it so Difficult to Convincingly Demonstrate Efficacy?**

**Variability of Patient Populations Studied**

The study referred to in the introduction [1] could not demonstrate the benefit of supplemental TPN in a widely differing population. Subgroup analysis suggested, however, that malnourished patients benefited more, that critically ill patients did not do well, whereas surgical patients did. Recently published papers (the better papers) did not report a benefit, whereas the older papers did. Patients that had received fat emulsions as part of their TPN did not benefit, whereas there was a clear tendency for patients not receiving fat as part of their TPN regimen to achieve benefit from their nutritional support regimen. This subgroup analysis was a retrospective analysis of the data, precluding a grade-A recommendation. Nevertheless the results of the study strongly suggest that the inclusion of a great variety of patients may have obscured a benefit in selected patients, due to the fact that artificial nutrition in other subgroups of patients is harmful. Unfortunately in clinical nutrition excellent studies are scarce for several reasons. Studies are of insufficient quality, because they do not observe generally accepted rules to enforce the validity of the results. Studies may not be randomized, not blinded, of insufficient power, or not focused on relevant clinical endpoints.

**What Surrogate Endpoints Should be Aimed for?**

Another disturbing factor in finding support for the contention that artificial nutrition is useful is the difficulty in interpreting surrogate endpoints. One
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would welcome a study that delivers firm evidence that nutritional support improves function, for instance muscle function or immune function. Clear evidence that patients get stronger and have better muscle endurance, or improve their $\text{VO}_{2\text{max}}$ furnishes level-2 or even level-1 evidence and may lead to level-A recommendations, provided that, at the same time, the ability to resist infection for instance remains intact. It is, however, very difficult to interpret the different measures of immune function. When, for instance, as a result of nutritional support the cytokine response is upregulated, some authors claim that this is a positive effect, because apparently the acute phase response is required to overcome the ailment of the patient [2]. In other papers the conclusion is that downregulation of the cytokine response and other inflammatory parameters is useful, because apparently the nutritional intervention improves the ability of the patient to withstand disease, as shown by a faster abatement of the acute phase response [3]. The effects of n-3 fatty acids are also interpreted very differently. In inflammatory bowel disease [4, 5], rheumatoid arthritis [6], and end-stage malignancies [Fearon et al., personal commun.], n-3 fatty acids have been shown to decrease inflammatory activity with modest but clear clinical benefit, whereas in combination with RNA and arginine they have been claimed to be beneficial, because they upregulate the immune response [7–9].

These uncertainties are the result of the confusion that generally exists concerning the purpose of the stress response. Is it good or bad for the organism, and should it therefore be inhibited or stimulated? In clinical surgical practice we are trying to diminish the stress response by limiting the surgical trauma, by pain relief, by certain anesthesiological techniques, and have found that such techniques improve postoperative recovery and shorten hospital stay [10–14]. This approach does not, however, prevent the metabolic response to trauma that remains necessary to achieve an adequate acute phase response, including wound healing and a central immune response largely taking place in the liver. This is shown by early work from Clowes et al. [15] who found that, when the liver could not take up amino acids derived from peripheral tissues, septic liver patients would die. This shows that to a large extent the catabolic response to trauma/disease is crucially necessary to overcome the insult successfully. Severely depleted patients do not raise a normal stress response, do not exhibit normal wound healing, do not make normal pus and do not show normal clinical signs of inflammation, but at the same time they do not recover from the trauma or the disease from which they are suffering [16]. In this patient category it is therefore mandatory to stimulate the stress response. Most likely this is not the case in multiple organ failure (MOF), where there appears to be an overshoot of the stress response. This results in extreme leakiness of the membranes, extreme vasodilatation, very high cardiac output, and malfunction of virtually every cell in the organism. This sequence of events ultimately leads to a very high mortality. This ‘overshoot’ of the stress response is therefore most likely
harmful and may benefit from measures intended to inhibit the overshoot. The confusion regarding the significance of immune/stress parameters as endpoints makes it difficult to settle the question whether nutritional support and modulation of the nutritional regimen are beneficial especially with regard to immune function. The claim, that in certain clinical states, the stress response should be stimulated and in others inhibited may partly explain why immune-stimulating diets are not overall beneficial in different populations.

**The Clinical Benefit that can Reasonably be Expected in Short Periods of Time**

Although nobody would question the need for nutrition in the long run, the normal reserves of the body allow a period of starvation that may differ with nutritional state, extent of disease/trauma, duration of disease/trauma and co-morbidity. The effect of even short nutritional support may therefore be greater when patients are previously malnourished, are subjected to greater trauma or disease for longer periods. On the other hand, the ultimate clinical outcome also depends on other factors like the adequacy of the primary treatment and the amount of blood loss during operation [17]. Consequently, the effect of short-term nutritional support can only be modest. To detect this effect large studies are required, which are difficult for single institutions to perform.

It is even more difficult to detect differences in clinical endpoints, when different nutritional regimens are compared. This also applies to the potential differences in clinical outcome between the parenteral and the enteral route.

**The Clinician’s View not Taking into Account the Results of Meta-Analyses**

*Parenteral versus Enteral Nutrition?*

Parenteral nutrition is risky and expensive. It creates complications during insertion of the catheter. At a later stage, catheter sepsis can occur, which is generally self-limiting when the catheter is withdrawn, but in a small but substantial number of cases more severe complications can occur like thromboembolic processes, infected thromboses in rare cases leading to pulmonary abscesses. Another problem is that there is still some doubt about whether fat included in the parenteral nutrition regimen has immunosuppressive properties, which may be harmful in critically ill patients. The stability of the all-in-one solutions and the distribution of particle size are far from completely resolved issues. In some countries and hospital pharmacies little quality control exists and all-in-one solutions are mixed without good control and depending on which amino acid and carbohydrate solutions and which fat emulsions are available at any particular time. There are also suggestions from the literature that parenteral nutrition has less favorable effects on metabolism, especially in the splanchnic area, than enteral nutrition.
Enteral nutrition is cheaper and less complicated. Simpler and more polymeric food components are possible and the bowel is directly fed. This induces the secretion of trophic factors, secretion of bile and biliary immunoglobulin and promotes peristalsis. It has also been claimed that the administration and control of enteral nutrition is simpler and easier to do and causes fewer complications. This is not the case. Catheter placement problems and dislodgement are common, patients do not always tolerate a full enteral regimen well, and in very ill patients or patients who are not fully conscious there is always the risk of aspiration. To apply enteral nutrition safely and effectively more time investment is required from the attending staff than for parenteral nutrition. This is in some countries the main reason for the rather liberal use of parenteral nutrition. Nevertheless there appears to be general agreement that, when enteral nutrition can be safely administered, it is the preferred route. Most people would intuitively feel that, when evolution has supplied mankind with a gut, we should try to use it in patients in need of artificial nutrition. As advocate of the devil, one would raise the question, however, whether there are solid biological clinical reasons to use the gut rather than the intravenous access route.

**Can we Safely Starve the Gut?**

There is no substantial experimental evidence that gut starvation leads to a higher mortality in animals subjected to a toxic challenge (e.g. lipopolysaccharide) than animals receiving (moderate) amounts of enteral feeds [18, 19]. In dogs, it was found that parenteral nutrition and gut starvation diminish bile flow and enormously and significantly reduce the biliary secretion of sIgA [20–23]. It has also been shown in endotoxin-treated animal models that high-protein (and calorie?) diets lead to higher translocation of *Escherichia coli* administered enterally than low protein diets [24]. This in turn is in line with the clinical finding that some severely ill patients (generally MOF patients) in the ICU do not tolerate enteral nutrition. Even when enterally fed post-pylorically, they develop bowel distension, stomach retention, paradoxical diarrhea or no stools at all [25]. It appears very likely that force-fed enteral nutrition in such a stagnant gut may serve as an effective culture medium for bacterial proliferation and overgrowth. MOF affects all organs, in fact all cells in the body including the gut. Migrating motor complexes are abnormal [26, 27], cells become leaky, there is increased extracellular water (edema), atrophy and defective regeneration, ulceration, osmotic (due to malabsorption) and secretory diarrhea (due to defective active Na transport) or constipation. Therefore in such patients gut integrity and function have been demonstrated to deteriorate. The intestine may very well contribute to MOF, but clinically it is also apparent that these patients deteriorate by force feeding [28, 29]. There is also a category of less ill patients, in which enteral feeding is successful and these patients are less likely to develop MOF. The question is, however, what the chicken is and what the egg? Is the patient
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primarily less ill and therefore does not develop MOF, but is also able to tolerate enteral nutrition, or does enteral nutrition prevent the occurrence of MOF?

Recently a claim has been made that the deleterious effects of increased gut wall permeability is mediated by the lymphatic system via endotoxin and cytokines and that, as a consequence, the first organ exposed is the lung, leading to acute respiratory insufficiency. There is, however, much clinical evidence supporting the presence of a gut-liver axis. In a high proportion of cases, starvation of the gut, stagnant loops or bypassed small bowel segments all lead after weeks to intrahepatic cholestasis without accompanying other organ failure. This cholestasis ranges from very mild to extremely severe, leading to liver cirrhosis, increased morbidity and mortality [30–32] (references in children and bypass patients). It is often claimed in clinical practice that the cholestasis occurring in patients receiving TPN is the result of the decrease in bile flow induced by TPN. This may be true, but it is only part of the explanation. In a clinical practice experiment, the *succus entericus* collected from a group of patients with high-output proximal small bowel fistulas was reinfused into the distal defunctionalized bowel segment, which until then was starved as it had not received normal proximal bile, gastric and pancreatic secretions [33] (Fig. 1, 2). Cholestasis was significantly alleviated despite ongoing TPN. This proves that a starving small bowel segment in some way inhibits the active secretion of bile and bilirubin by the liver. This finding is not limited to small bowel fistula patients receiving TPN, but can also be observed in patients with an intact but starving gut [34]. Other examples are neonates with short bowel, patients with blind loop syndrome and patients subjected to jejuno-ileal bypass [35, 36]. A starving gut can therefore be detrimental to the liver and TPN may worsen the situation. The question is how relevant this is clinically. In severe chronic cases as in neonates and obese patients undergoing bypass surgery, it may induce liver cirrhosis, liver insufficiency and mortality, but it is not certain to what degree the often mild cholestasis during TPN deteriorates the metabolic and immunologic functions of the liver and influences clinical outcome. It is certainly a matter of concern and a negative aspect of TPN and gut starvation.

*The Clinically Obvious*

Some clinical practices are so beneficial that we should not make the mistake of not applying them, because no meta-analyses exist that prove the obvious clinical truth. Withholding parenteral nutrition from a patient with a proximal small bowel high output stoma, and giving enteral nutrition exclusively as part of a randomized clinical trial to prove the efficacy of parenteral nutrition in these cases would not be accepted by any medical ethical committee. The fact that this ‘truth’ is not evidence-based should therefore not distract us from the clinically obvious.
Fig. 1. Bilirubin levels of each patient before and after reinfusion of gastrointestinal secretions. Adapted from Rinsema et al. [33].

Fig. 2. Alkaline phosphatase levels of each patient before and after reinfusion of gastrointestinal secretions. Adapted from Rinsema et al. [33].
Another example may be the famous patient with necrotizing pancreatitis and multiple operations to remove the infected necrosis and secondary complications. A not negligible proportion of these patients has a large inflammatory mass in the pancreatic region, in which the duodenum and the proximal small bowel are embedded, and consequently suffer from paralytic ileus, precluding the successful use of enteral nutrition. Half to 60% of this patient category survives after a protracted illness of months in duration. Several factors determine survival, such as surgical expertise, comorbidity, adequate ventilatory, nephrologic, cardiologic and nursing support. All patients, however, would have died, if they had not received adequate parenteral nutrition. This does not imply that all pancreatitis patients require TPN. Necrotizing pancreatitis occurs in only 15–20% of patients with acute pancreatitis, and of this group approximately half suffer from the severe pancreatitis described earlier. In most pancreatitis populations, this subgroup is diluted by a large group with noncomplicated necrotizing pancreatitis or even just self-limiting edematous pancreatitis. It is very difficult to demonstrate the benefit of parenteral nutrition in the subgroup described in a prospective randomized trial, when patients in whom no benefit of TPN will be achieved are also included. Thousands of patients would have to be included. The patients with pancreatitis of moderate severity do not require artificial nutrition, because the disease is self-limiting within a few days or enteral nutrition, can be safely applied. However, one would have difficulty in withholding parenteral nutrition from the ‘complicated necrotizing pancreatitis’ group, in which it is obvious that enteral nutrition cannot be safely and effectively applied.

A third category of patients, in which it is clinically obvious that the clinical condition dictates the nutritional regimen, is those with full blown MOF. As described in an earlier section, this patient group has an increased extracellular water space amounting to a surplus of 15–20 liters, and has severe disturbances of peristalsis, digestion and absorption. These abnormalities deteriorate when the patients are enterally force-fed. On the other hand, it is also questionable whether they could benefit from full-speed TPN. Full-blown MOF should either improve within days or the patients die. In some patients MOF improves but changes to a state of low-grade slowly improving toxicity. There is no evidence available about what the nutritional regimen should include under these circumstances. Clinical judgment generally dictates the administration of TPN and gradual introduction of enteral nutrition, depending on tolerance. It is believed, and supported by some data in the literature, that underfeeding is less detrimental than overfeeding.

**Recommendations Based on Clinical Judgment**

TPN is life saving in a small but distinct group of patients. These include abdominal catastrophes like high-output proximal small bowel fistulas,
extensive inflammatory masses leading to adynamic ileus of long duration, and short bowel syndrome.

TPN may also be of benefit in patients recovering from MOF or patients with lingering toxicity, in whom bowel function has not yet recovered and in whom full enteral nutrition is impossible for longer periods of time.

Starvation of the gut has several damaging effects. The bowel becomes more permeable for endotoxin and cytokines, which may aggravate or precipitate MOF via the lymphatic route. It is difficult to substantiate this clinically. Convincing clinical evidence exists indicating that a gut-liver axis exists, including toxic effects of starving small bowel on the excretion of bile and the clearance of fat [37–39]. In specific patients groups, this may lead to liver insufficiency. TPN also has an inhibiting effect on bile flow and on the excretion of sIGA in the bile and therefore contributes to the toxic effects of bowel starvation on the liver. The composition of TPN and especially the fat component have been claimed to contribute to cholestasis [32, 37]. During clinical TPN the relevance of these findings is difficult to assess. It is not known whether the liver abnormalities have detrimental effects on liver function. These abnormalities should certainly be viewed as potentially detrimental for the body, and should encourage the use of enteral nutrition whenever feasible.

Enteral nutrition is less expensive than parenteral nutrition, but is not easier to apply. In fact, the investment of more time and energy of the attending staff is necessary to successfully implement enteral nutrition.

A small group of severely ill patients (MOF: see p. 185) does not, or only very partially, tolerate enteral nutrition. In this patient group, force-fed enteral feeding aggravates the adynamic ileus, often present in these patients, leading to bowel distension [25], abdominal compartment syndrome, deterioration of MOF, and, even in rare cases, bowel necrosis [28, 29]. It is not certain to which extent this patient group benefits from TPN.

**What can we Learn from Meta-Analyses?**

Supplemental parenteral nutrition did not show benefit in a meta-analysis reported by Heyland [1]. Neither the complication rate nor mortality was influenced by TPN. Subgroup analysis revealed that malnourished patients and surgical patients benefited in early studies and that in critically ill patients the effects may have been harmful. There was also a suggestion that patients receiving fat had more complications than patients who did not. In most of these studies, patients were included who were well nourished and suffered from short-term critical illness. Many of these patients, therefore, did not need nutritional support. In addition, the period during which nutritional support was provided was short, precluding a distinct benefit even in the patients who might have benefited.
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The results of the available studies comparing enteral and parenteral nutrition yield the following recommendations.

Enterally fed trauma patients have fewer septic complications than patients fed parenterally [40, 41]. The same is true in the total group of pancreatitis patients [1]. Such meta-analyses are not able to furnish recommendations for subgroups of patients within these patient groups. The clinical impression is that in the most severely ill patients enteral nutrition is not feasible and may be harmful. This is, however, not visible in the total group, as the majority of patients tolerate enteral nutrition well and, when nourished parenterally, suffer from the complications of gut starvation and the insertion and presence of central catheters. The available meta-analyses do not establish whether enteral nutrition prevents the development of MOF. Several clinical observations, although not of PRCT structure, show that bowel starvation has detrimental effects on liver morphology and function. The presence of a gut-liver axis, implying the toxic influence of a starving gut on the liver, is a clinically obvious fact.

The varied populations, including very different types of patients, may have obscured positive effects in distinct subgroups.

In the meta-analyses the type of parenteral regimen was also not taken into account. It appears that emulsion stability and fat composition may have deleterious effects on immune function, cholestasis and liver function. These problems require further study to arrive at final answers and solutions.

References


Discussion

Dr. Becker: I’d like to make a comment on cholestasis resulting from distal small bowel exclusion. Bile salts are regarded as the primary choleretic influence on the liver; 97% of bile salts are reabsorbed and this causes choleresis. The effect is not a direct toxic one on the liver cell, but a choleretic effect, so as soon as you get loss of bile salts through bile salt diarrhea, short bowel syndrome, loss of the distal ileum, and so on, you get stasis because of the choleresis effect.

Dr. Soeters: I don’t think that is entirely true. For instance, in rats with an interrupted enterohepatic circulation, in which the bile is taken from the common bile duct and reinfused, you find that, when you take out some of the bile, there is an increase in the secretion of bile in those rats, but the composition of the bile changes. That is another crucial element in the cholestasis – some of the bile acids that are produced have a further cholestatic effect on the liver, in reverse.

Dr. Labadarios: We have heard a lot about early vs. late enteral nutrition support. Could you give us some guidelines as to what is meant by ‘early’? I know this is very difficult to define, but maybe you have some thoughts about it.

Dr. Soeters: I did not give you figures, and I don’t think any figures are in fact available, but I think it is important first to look at the nutritional state of the patient at the time when surgery is under consideration. If there is severe depletion – that is, more than 15% body weight loss – the complications of any type of intervention will rise dramatically, so there would be an indication for providing preoperative nutrition. The indication for artificial nutrition, preferably enteral, depends on the patient’s nutritional state before surgery, on co-morbidity, and also on what you expect these patients to go through. Some patients are admitted acutely with an abdominal catastrophe, for example, and you can foresee that it will take weeks or months before recovery, if indeed they survive at all. So in those patients there is an immediate indication to start enteral (if possible) and parenteral nutrition in a reciprocal manner. If there is distended bowel or paradoxical diarrhea, don’t push the enteral nutrition, but if there is normal bowel function, then you can try enteral feeding in conjunction with parenteral. So in summary, there are two main issues: the degree of pre-existing morbidity and the nutritional state at surgery, and what you expect the patient will have to go through.
Dr. Segal: There are two particular clinical situations in which there is controversy over when you should start enteral vs. parenteral nutrition. One is the patient with acute pancreatitis: at what stage do you decide to feed? The other is the patient with severe ulcerative colitis: again, when does one intervene, and should the decision be influenced by whether there is small bowel pathology or large bowel pathology?

Dr. Soeters: Patients with chronic pancreatitis are in general malnourished to start with, so if there is an acute exacerbation of the pancreatitis and if the patient is malnourished, then I think there is an indication to give enteral nutrition. It has never been shown that enteral nutrition is harmful in those patients, and you can give it by jejunal or duodenal tube. So, in that situation, a decision should be made rapidly to give some form of enteral artificial nutritional support.

What I demonstrated about the harmful effects of gut starvation largely refers to the small bowel. The small bowel is much more vulnerable and sensitive to stasis and to non-function than the large bowel. A degree of ileus can be tolerated much longer, if only the large bowel is affected. In ulcerative colitis, my gastroenterologic colleagues push parenteral nutrition, not because of the nutritional state – which strangely enough is often quite good in those patients – but because it limits the number of stools and the harmful effects of these persistent watery stools on the perianal region and on lifestyle (these patients have trouble sleeping and often have to get out of bed every hour to defecate, and they may be incontinent). So parenteral nutrition is used more for nursing reasons than for nutritional reasons. In itself it does not decrease the inflammatory activity of the colitis, but it does diminish the number of stools.

Dr. Haschke: A well-designed study by Bozzetti and co-workers from Milan was recently published in *Lancet*. One of the advantages of this study is that the experimental design was clear: the subjects were malnourished patients undergoing elective surgery for gastrointestinal cancer, the procedure involved a comparison between parenteral and enteral nutrition in the first 5 days, and the outcome variable was clearly defined. The study also had the advantage that the sample size was adequate – 158 patients finished the study in each group and the outcome was very clear. In the parenteral group the complication rate was 49%, in the enteral group 34%; 15% of the enteral group had infections vs. 35% in the parenteral group. This emphasizes the probable importance of the gastrointestinal tract in the functioning of the immune system – the local immune system is not stimulated when you give parenteral nutrition alone and allow the gut to starve, whereas when you give early enteral nutrition you encourage this function of the gut.

Dr. Soeters: I think in the meta-analysis of Moore [2,3] and Kudsk [4,5] there is a grade B recommendation that enteral nutrition creates fewer septic complications than parenteral nutrition. There are some criticisms of the quality of those studies, which is why it is a level B recommendation. For example, some of the end points were analyzed retrospectively (e.g., septic morbidity), and there was a subgroup post hoc analysis in some of the studies, but on the whole I think the message is clear that enteral nutrition creates fewer septic complications than parenteral nutrition. It would have been nice, if there had been a third group who were conventionally treated, but I agree that the data published in *Lancet* were clear.

Dr. Waitzberg: Are there any tests that can be done to indicate when it is safe to start giving enteral nutrition, so that one can avoid the complications related to starting enteral nutrition when the patient cannot tolerate it?

Dr. Soeters: We are desperately looking for an easy test to decide whether the gut is at risk, and whether if you push enteral nutrition you will do more harm than good. Tonometry should be useful, but there is enormous variability in the results, even
within the same patient. Measures like blood pressure are not very useful—no one would give enteral nutrition to a patient in shock. A bladder pressure above 15–20 cm H2O has been advocated recently as a measure of abdominal compartment syndrome. However, that has not been validated and there is enormous variability. So you are still left with the usual clinical criteria and clinical judgment.

**Dr. Waitzberg:** Would you advocate the use of enteral nutrition in patients who require cardiogenic drugs to maintain a stable hemodynamic status?

**Dr. Soeters:** The cardiac index is increased by around 15–20% by giving enteral nutrition, because of increased mesenteric blood flow, but if there is a reason to think that mesenteric blood flow is compromised you should not give enteral nutrition. The need for inotropic agents may be warning you of this. If the patient needs inotropic agents to maintain blood pressure, I would be very careful.

**Ms. Marino:** In our cardiac intensive care unit, we commonly feed patients who are on inotropes. Could you suggest the level of inotropic support that would indicate the use of TPN rather than enteral feeding?

**Dr. Soeters:** I'm afraid I have no personal experience with cardiac intensive care unit patients, but in general you should not give enteral feeding to unstable patients. Also, if you have to give high doses of inotropic drugs to maintain blood pressure, you should avoid enteral feeding, as you have no idea what these agents are doing to mesenteric flow.

**Dr. Chioléro:** When the gut is sick, when you have acidosis and the abdominal compartment syndrome, it is very difficult to give enteral nutrition. You are only going to benefit the patient who has a good gut—burns patients, patients with severe trauma, for example—but not those with gut failure and gut ischemia. Such patients should not be given enteral nutrition; it may do them more harm than good. So whatever you measure in the gut may give you the contraindications to enteral feeding, but it will not give indications for it. That is a very important point.

**Dr. Soeters:** I agree.

**Dr. Labadarios:** I have a comment relating to expense, a subject that is constantly reiterated. Expense in relation to what? You ventilate a patient day after day, you haemodialyse a patient as necessary, you do anything that is necessary for the patient, and expense does not come into it. When nutrition is initiated, then everything is expensive! Then again, there has been a tendency to promote enteral or parenteral feeding on the grounds of expense, but I don’t think there is really much difference between the two. Can you comment on this concept of ‘expensive’? I feel the word should not be used, as it is inhibitory to the growth of nutrition in our clinical practice.

**Dr. Soeters:** People who are against parenteral nutrition always say that it is expensive. In terms of efficacy, when used in the right patient, I don’t think it is expensive. But although the materials used for parenteral nutrition may themselves be more expensive, the energy that you have to invest in making enteral nutrition successful is also expensive, and probably more so than parenteral nutrition. I know there are areas in the world, for instance China, where a lot of parenteral nutrition is given just because it is so easy to give. The doctor prescribes it and it flows in. Enteral nutrition requires much more attention related to gastric retention, the need to reinset tubes, and so on, so more nursing care is necessary. In some countries the nursing care is available and cheap, in other countries nursing care is not available and is expensive. So on the whole I think the costs even out, provided you use the right indications and do not give parenteral nutrition to patients who can easily be nourished enterally.
References


