Nutrition support for patients in the intensive care unit

R D Griffiths, T Bongers

Enteral nutrition (EN) is the mainstay of nutrition delivery within intensive care seeking to capitalise on its benefits for the gastrointestinal tract and associated immune system, but this has brought new challenges in delivery to the sick. The hoped for benefit has led to the mistaken belief by some that parenteral nutrition (PN) is no longer required. However, a greater appreciation of the risks of EN delivery in the sick patient combined with improvements in PN formulation and use help explain why PN is not as risky as some have believed. Real outcome benefits have been described with the new glutamine containing PN formulations. PN remains important in the presence of gastrointestinal feed intolerance or failure.

The intensive care unit (ICU) patient presents a number of nutritional challenges. The case mix of patients admitted to intensive care units may range from those admitted electively after major elective surgery to those admitted as emergencies after some surgical catastrophe, major trauma, sepsis, or respiratory failure. The variation in age range and prior health status may be extreme and nowadays ICUs are admitting increasingly more elderly, frail, or malnourished patients whose nutritional reserve may be severely compromised.

RECOGNITION OF PRIOR NUTRITIONAL STATUS

Many patients admitted in emergency may have been suffering an illness and have had poor nutrition before admission to intensive care. The best assessment of prior nutritional state is a detailed history of prior illness and nutritional intake combined with clinical examination of fat and muscle distribution. Body mass index (BMI = weight in kg/height in m²) is useful but weight can be difficult to obtain accurately and may be distorted by resuscitative fluid administration. We know that ICU patients suffering from under-nutrition with a limited nutrition reserve have a poorer outcome and that having a low BMI has been shown to be an independent predictor of excess mortality in multiple organ failure.1

WHAT IS OCCURRING METABOLICALLY?

Increased metabolic requirements through increased protein breakdown and synthesis and changed substrate turnover occur through a range of demands depending on the various clinical situations. The enormous endocrine and cytokine flux of systemic inflammatory response common to sepsis or major trauma will increase basal metabolic rate usually proportional to the degree of insult and this is compounded by the effects of treatments such as adrenergic inotropes. However, what is not realised is that the total energy requirements may only modestly increase in the first few days. Heavy sedation and neuromuscular paralysis used to facilitate total ventilator support will reduce skeletal muscle activity. Detailed metabolic studies in ICU patients have shown that the total energy expenditure in the first week for patients with severe sepsis is 25 kcal/kg/day and for trauma patients 30 kcal/kg/day. What is often not appreciated is that by the second week evidence suggests this may have risen to as much as 40 kcal/kg/day in sepsis and perhaps even 55 kcal/kg/day in some trauma cases. These are not necessarily nutrition targets but illustrate the wide variation in possible requirements.

What is more challenging to accept is how unreliable most predictive estimates of energy requirements are? Detailed metabolic measurements show wide patient variation between and within patients on different days.4 It has proved possible for uncomplicated surgery to suggest energy requirements of 1.0–1.15 times basal metabolic rate (BMR) while major surgery 1.25–1.4×BMR is sufficient. Currently recommendations suggest that 25 kcal/kg/day (105 kJ/kg/day) is a reasonable target intake for ICU patients initially for the first week however if too rigorously adhered to especially in some sepsis and trauma patients may be inadequate in the long run and a target of 30 or 35 kcal/kg/day (125–146 kJ/kg/day) may be more appropriate in subsequent weeks.

As long as over feeding is avoided to prevent excess lipid deposition the steady supply of protein is the most critical nutrient. Sequential studies on the ICU in severely septic patients with peritonitis5 show after an initial gain in body water with resuscitation, there is a large and progressive loss of protein despite full nutritional provision. Two thirds of the protein loss comes from skeletal muscle in the first 10 days, but later more is lost from the viscera. This loss of lean body mass (whole body water and protein) that ranges from 0.5% to 1.0% loss per day is far greater than that attributable to bed rest alone. This occurs in the context of full nutritional provision and not simple starvation and a consistent feature seen is that body fat could be preserved by adequate calorie provision.6 The rapidity and extent of the catabolic

Abbreviations: EN, enteral nutrition; PN, parenteral nutrition; ICU, intensive care unit; NJ, nasojejunal; NG, nasogastric; BMI, body mass index
muscle wasting in the critically ill is astonishing with a reduction in muscle fibre cross sectional area of 3%–4% per day. In the first couple of weeks despite a 35%–50% decline in respiratory and skeletal muscle function there is no loss of cardiac mass or function in critically ill patients however wasting does occur with protracted illness.

Muscle wasting is ultimately a balance between protein synthesis and degradation. Various tissues and organs respond differently and change during the course of an illness. After modest surgery there is a decrease in whole body protein synthesis rather than breakdown. Short term starvation decreases skeletal muscle protein synthesis. With trauma and major surgery both synthesis and degradation increase, the latter being more enhanced. In multiple organ failure increased whole body protein breakdown predominates over increased protein synthesis. To meet this metabolic demand for substrates involved in protein synthesis increased proteolysis occurs, particularly in skeletal muscle. This occurs through the energy demanding ubiquitin-proteasome pathway that requires ATP and is stimulated by fasting, acidosis, trauma, sepsis, and cancer. Glucocorticoids are important in this process not only in regulating and increasing the proteolysis in muscle but also increasing the utilisation of the resulting amino acids in the liver. A combination of insulin and essential amino acids oppose the catabolic effect mediated through mechanisms that inhibit proteolysis in the normal state. In the response to sepsis or injury the signal cascade of TNF and interleukins from activated macrophages and endothelial cells stimulate the ubiquitin-proteasome pathway in muscle probably involving multiple cytokine signals. Normally during prolonged starvation, after the initial mobilisation of amino acids for gluconeogenesis, protein breakdown decreases as increased energy is derived instead from fat metabolism. This important control preserving muscle protein is lost in the face of inflammation and endogenous glucose production continues. The release of amino acids during protein breakdown in skeletal muscle is not in proportion to the constituent amino acids, rather many are synthesised to produce glutamine that is released from muscle to meet the demand for additional amino groups and for added gluconeogenesis.

The provision of high quality (essential and conditionally essential) amino acids along with insulin is central to good nutritional support and is not stored but used in synthesis or metabolised the provision needs to meet on going protein synthetic demand. Importantly, in contrast with the malnourished where protein synthesis can be stimulated by nutrition and catch up for missed feed is therefore possible, in the critically ill where synthesis may already be stimulated the ability to catch up for missed feeding is limited. Currently our best evidence in ICU based upon whole body measurements suggests at least 1.2–1.5 g/kg/day of protein are needed.

There are sound metabolic arguments for nutrition; the challenge comes in finding the safest and most reasonable way to deliver it because patients within intensive care except when recovering are unable to eat normally.

**WHAT IS THE EVIDENCE THAT SUPPORTS THE CURRENT DECISIONS REGARDING THE ROUTE OF NUTRITION TAKEN IN ICU?**

The underlying basis in favour of EN is that a failure to maintain normal oral nutrition is associated with immunological changes and impairment of the gut associated lymphatic system (GALT) that leads to the intestine, through lymphatic drainage, becoming the source of activated cells and proinflammatory stimulants during gut starvation. Other secondary abnormalities such as permeability changes and occasionally even bacterial translocations increase the immune challenge to the GALT but their contribution is probably secondary. This failure of immune defence was originally considered uniquely dependent on lack of enteral (luminal) nutrition. However, significant reversal of these defects has been shown in animal models using intravenous nutrition that specifically has added glutamine.

**SHOULD PATIENTS WITHIN INTENSIVE CARE BE FED ENTERALLY (EN) OR PARENTERAL (PN)?**

In the UK the past 15 years has seen a move away from providing nutrition support intravenously (parenteral nutrition, PN) to providing it nasogastrically or jejunal (enteral nutrition, EN). The reasons reflect changes in our understanding of nutrition and improved delivery systems. Indeed for some patients nowadays weaning off ventilator assistance may mean sitting out in chairs and taking an oral diet.
have been saying.28 This difference was lost when only those
0.27 to 0.97, p = 0.04) confirming what many commentators
benefit in favour of the use of PN (odds ratio, OR 0.51, 95% CI
intention to treat analyses from nine studies comparing EN
approach to investigate the effect of trial quality.27 The
studies in reviews the authors have used a component based
attempt to overcome the criticism of including poor quality
that PN if needed was harmful. A recent systematic review
compared with delayed nutrient intake and infection risk was
not different. A duplicate systematic review publication of the
EN compared with PN question, although presented in more
detail, confirmed there was no difference in outcome, length
of stay, days ventilation, or mortality28 distilling the myth
that PN if needed was harmful. A recent systematic review
from Australia has moved the debate even further. In an
attempt to overcome the criticism of including poor quality
studies in reviews the authors have used a component based
approach to investigate the effect of trial quality.27 The
intention to treat analyses from nine studies comparing EN
and PN were aggregated and showed a significant mortality
benefit in favour of the use of PN (odds ratio, OR 0.51, 95% CI
0.27 to 0.97, p = 0.04) confirming what many commentators
have been saying.28 This difference was lost when only those
studies (and therefore a selected population of patients)
where early enteral feeding was used. The risk of infection
with PN remained but it must be noted that this risk is
insufficient to affect outcome.

Great care should be taken not to over interpret these
reviews either way. The most important fact was that in all
these studies no consideration was taken regarding the
functional state of the gastrointestinal tract as patients could
be randomised to either route of feeding.

**Key points 2**

- Enteral nutrition (EN) as the first choice, is usually
delivered nasogastrically and is justified on cost alone.
- Many patients were given parenteral nutrition (PN) in
the past despite having a fully functional gastrointestinal
tract. However, PN is still needed in 9% of these patients
so cannot be forgotten.
- EN does carry a lower infective risk than PN, RR 0.66
(95% CI 0.56 to 0.79), but at the cost of a non-
significant trend to increased complications RR 1.36
(95% CI 0.96 to 1.83).
- Meta-analysis shows that EN compared with PN is
either risk neutral for mortality risk RR 0.96 (95% CI
0.55 to 1.65) or PN may even offer a lower risk 0.51
(95% CI 0.27 to 0.97)

**WHEN GASTROINTESTINAL FUNCTION IS IN
DOUBT MIGHT PN EVEN BE SAFER THAN EN?**

The only level 1 study that has addressed the EN compared
with PN question of benefit to ICU doctors is a fascinating
“pragmatic” study by John MacFie’s group in Scarborough,
England.29 The study looked at the clinical outcomes of all
562 patients given EN and PN in a major hospital under the
auspices of a specialist gastroenterology nutrition service. EN
was given to 237 patients with a functioning gastrointestinal
tract; PN to 267 with absolute gastrointestinal failure; and
this left a group of 64 where it was unclear if the
 gastrointestinal function was adequate. They randomised
this last cohort of patients to compare EN and PN. They
therefore made sure that the risks were only applied to those
that need them and overcome the problems of all previous
comparisons of EN and PN where opinions might differ as to
the appropriateness of the particular choice of route of
feeding.

There was obviously a noticeable difference in the patients
and the diseases in the first two non-randomised groups that
received either PN or EN. Strikingly there was no difference
in septic morbidity but a higher non-septic complication rate
in the enteral feeding group. These complications were not
minor and significant excess in mortality was seen with EN
emphasising some of the mortality risks associated with EN.
In the non-randomised groups one might think this
increased mortality in the EN patients could be explained
by the difference in patient population was it not that the
exact same relation with higher mortality was seen in the
group of patients where randomisation had occurred.
Inadequate nutritional intake characterised patients receiving
EN. Inclusion of these important data in the most recent
meta-analysis contributes to the evidence that the risk of
enteral feeding on mortality may even exceed that of PN.

**EN DELIVERY PROBLEMS AND ASPIRATION**

To understand the debate however one must appreciate the
issue of delivery and risk of aspiration for ventilated intensive
care patients as the sicker ones have increasing gastrointestinal
intolerance. A recent study in France of 153 nasogas-
trically fed patients showed that upper digestive intolerance
is a frequent event in the critically ill and associated with pneumonia (43% v 24% p = 0.01), longer ICU stay (23 v 15 days p = 0.007), and increased mortality (41% v 25% p = 0.03).30 Even corrected for illness severity the risk of death was significantly increased RR 1.48 (95% CI 1.04 to 2.10). This increased risk is an important issue that can get
buried in many enteral studies that involve broad populations
of patients with different illness severity. The risks for
intolerance were not surprising because of sedation use (RR
1.78 95% CI 1.17 to 2.71) and catecholamine use (RR 1.81,
95% CI 1.21 to 2.70) both features of genuine intensive care
patients, particularly those with sepsis and shock. The
evidence that using motility agents or nasojejunal (NJ)
feeding rather than nasogastric (NG) feeding significantly changes these risks is lacking. A meta-analysis found that the
evidence that motility agents can affect any aspect of
outcome was wanting.31 A multicentre study from Spain
confirms that NJ feeding does not reduce the incidence of pneumonia.32 In 101 randomised ICU patients there was no
difference in feeding duration, length of stay, or mortality
(NG 43% v NJ 38%). Although the NJ group had lower
gastrointestinal complications there was a similar incidence
of nosocomial pneumonia (NG 40% v NJ 32%). While a study
from Melbourne, Australia33 suggested improved tolerance
with NJ feeding and a low requirement for PN another study in
60 USA medical patients showed that NG fed patients
reached their target goal earlier.34 There was no difference in
aspiration events. These studies are consistent with an earlier
study of 44 patients similarly randomised where small bowel feeding led to a significant increased proportion of the nutrition target achieved (p = 0.05) but definitely no reduction in ventilator pneumonia where the risk with small bowel feeding was RR 1.1 (95% CI 0.96 to 2.44). A rare but often fatal complication of non-occlusive bowel necrosis has been reported in critically ill trauma patients fed into the small bowel. As this serious complication cannot be detected early there is no overwhelming risk free evidence supporting NJ feeding in preference to NG feeding for the reduction in infective risk.

DOES IT MATTER THAT ENTERAL FEEDING MAY NOT DELIVER SUFFICIENT?

Several surveys have shown that the real practice in intensive care is to deliver considerably less than that prescribed ranging between 50% and 60% of target. What is “sufficient” is open to debate and there are some advocates of deliberate underfeeding. However, underfeeding is a concern as it has been suggested that medical ICU patients who received less than 25% of target feed have a higher risk of nosocomial blood stream infections. Feeding earlier should increase delivery but a study on early feeding in medical patients succeeded only in showing that poor nutrition support is common. They studied 150 ventilated, medical ICU patients, in a prospective but unblinded study using unfortunately consecutive rather than random allocation. The patients were fed using the NG or NJ method following a protocol to reach either a target of 25 kcal/kg on day 1 (early feeders) or only 20% of the target until day 5 (late feeders). In contrast with discussion earlier on surgical patients the 75 patients who were early fed had a significantly (p = 0.02) higher incidence of ventilator associated pneumonia 49.3% compared with 30.7%. Not surprisingly the early feeders had a longer ICU stay of 13.6 compared with 9.8 days (p = 0.04). Close inspection however shows that both groups received little nutrition with the percentage of target achieved in the early feeders of only 28% and even less at 7% in the late feeders (p = 0.001). Despite having a higher incidence of ventilator pneumonia and longer ICU stay the early feeders had a non-significant trend to lower hospital mortality 20.7% compared with 26.7% arguing against the view to semi-starve ICU patients. It could be argued that it was the increased risk of aspiration in the early feeding group that increased their morbidity, but that in the long run the greater nutrition improved their outcome?

Currently the most reasonable approach is to feed to meet the patient’s energy expenditure with care to avoid overfeeding. With PN overfeeding is far easier while as shown with EN it is virtually impossible. The introduction of guidelines encouraging increased nutrition prescription and use of enteral feeding, often starting earlier has increased nutrition prescription. Many ICUs follow nutrition protocols with PN overfeeding is far easier while as shown with EN it is virtually impossible. The introduction of guidelines encouraging increased nutrition prescription and use of enteral feeding, often starting earlier has increased nutrition prescription. Many ICUs follow nutrition protocols.

Key points 3

- In the malnourished patient unable to tolerate enteral feeding not giving parenteral feed significantly increases the mortality risk threefold, RR 3.0 (95% CI 1.09 to 8.56).
- The degree of underfeeding is a concern as it has been suggested that medical ICU patients who received less than 25% of target feed have a higher risk of nosocomial blood stream infections.
- Survival from intensive care was improved when an evidence based guideline for nutrition was followed and more nutrition was delivered more consistently. This was achieved by earlier introduction and more complete EN delivery without any decline in the use of PN alone or in supplementation.
- One of the real landmark outcome studies in recent years showed the great benefits of glycaemic control in patients receiving early enteral or parenteral nutrition.
The study confirmed there was no evidence that the addition of PN added any risks or caused harm. A recent meta-analysis suggested there is no evidence that supplemental PN is a risk in ICU as long as overfeeding is avoided but starting it early and concurrently in the non-malnourished is not yet warranted. Whether starting PN early in those patients unable to start early enteral feeding is beneficial as hypothesised by the recent Australian review is speculative and needs to be formally tested. Perhaps what is more important is to take a serious approach to nutrition and metabolic control overall. One of the real landmark outcome studies in recent years showed the great benefits of glycaemic control in patients receiving early EN or PN. Closely applied best evidence nutrition protocols were central to this process. The long term outcome was related to the tight glycaemic control achieved with insulin, and not by underfeeding. We know that taking a different approach and underfeeding by omitting lipids and delivering hypocaloric parenteral feeds neither prevents hyperglycaemia nor its infectious complications.

**WHY IS THERE PERHAPS LESS RISK WITH MODERN PN WITHIN INTENSIVE CARE PRACTICE?**

Reflecting on its origins and the state of nutrition support in the 1960s by just one of the pioneers will enlighten those interested on how far we have come. Such considerable experience over the past decades has highlighted the potential clinical complications of TPN and established the practical guidelines to minimise their impact. As the risks of indiscriminate and inappropriate use of PN are recognised they can be prevented by not overfeeding, and conscientious monitoring. Careful sterile line placement will minimise infections and some of the immunosuppression can be attenuated by supplementation of glutamine. Carefully respecting guidelines will go a long way to reduce the rate of complication but lack of consistent practice means that audit studies still regularly show over or underfeeding and shortcomings in PN prescription. It is therefore not surprising that some people carry a misguided interpretation that PN in itself rather than how it is used or misused in intensive care carries an excess mortality in patients where EN is difficult or impossible. It should not be forgotten that intensive care is potentially one of the safest environments for risky procedures as the high staff presence and experience should anticipate and prevent complications. For instance line infections have a serious association with mortality outside intensive care but recent evidence from intensive care shows that nowadays it is no longer possible to show an attributable causal link between intravenous catheter line infections and mortality. This is presumably because our practice of line management is greatly improved and any infective line is only a symptom of the patient’s illness and not a significant independent contributor to mortality. The relation between sepsis and nutrition in ICU and how it affects outcome has recently been reviewed.

The progressive development in formulation has improved PN used in the critically ill. The debate that intravenous fat emulsions have a detrimental effect on lung function in sepsis has been explored and shown that when infused at the normal recommended rates used in continuous all-in-one formulations to have no deleterious effect in septic patients. It seems it requires excessively rapid infusions inconsistent with good clinical practice to show any harmful effect. This is a good argument why all-in-one PN preparations should be universally available to intensive care.

The most significant formulation change in recent years to have entered ICU practice is the inclusion of glutamine to overcome a conditional deficiency. This reflects different requirements in the critically ill that are not met by conventional formulations that had compromises made for pharmaceutical reasons. Existing formulations that may be adequate to maintain a patient long term while receiving home PN have been proved inadequate for the changed requirements of the critically ill. The first double blind randomised outcome study comparing a standard PN with a glutamine enhanced PN within intensive care patients with gut failure as part of multi-organ failure showed a significantly improved six month survival (24 of 42 compared with 14 of 42, p = 0.049). Subsequent data showed that glutamine recipients have a significantly lower incidence of catheter related infections (p = 0.026), but overall only a non-significant and modest reduction in early acquired infections as the opportunity for new infections is so high in these patients. More importantly though there was a later reduction in infection and the survival of patients with candida infections was improved. There was a clear link between duration of glutamine and survival with a reduced intensive care mortality from multiple organ failure in those patients remaining in ICU for a longer period and requiring a minimum of five days of parenteral feed (p = 0.05) and a

### Key references


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**Nutrition in the ICU: simple rules**

- Feed the malnourished and plan for those soon to be.
- Start some enteral feeding if safe and as soon as practical.
- Use simple standard complete regimens, decisions over volumes of fluid dominate.
- Modest targets based on patient size bands are easier to achieve (25 kcal/kg/day with 1.5 g protein/kg/day).
- Use protocols, monitor delivery, note deficits, and act to meet targets.
- Use parenteral to complement or replace if delivery fails.
- Give sufficient insulin for glycaemic control using established protocols.
- Do not exacerbate glutamine deficiency, include in all PN preparations.
- Don’t miss feeding because it is harder to catch up.
Infective complications and morbidity resulting in reduced feeding alone before the stress event significantly reduces intensive care perioperative nutrition where especially pre-hospital response in the lungs of patients developing acute inflammation with antioxidants moderated the severity of the initial inflammatory response to activate defence mechanisms against infection, to direct cell movement, and induce endogenous cytoprotective mechanisms must be balanced by the collateral damage that occurs to various tissues.

Some studies have comprised single additional substrates such as glutamine or ornithine-alpha-keto-glutarate while other formulations include a fixed dose mixture of substrates. The evidence base in humans for the various additional substrates is variable and for the more complex mixtures at the fixed doses used within these formulations or “cocktails” is simply lacking.

Immune nutrient cocktails
Apart from one unblinded study using an immune nutrient cocktail that showed an improved ICU survival in septic patients evidence for benefit is lacking. Taking the two largest studies that contain hospital mortality data collectively the same immune nutrient feed produces a significant excess mortality OR 1.45 (CI 1.06 to 2.11) p = 0.02. This is disturbing because it suggests that more harm than good may occur. As benefits were said to occur in sub-sets of patients, it could be that some patients might benefit while others suffer harm. Mortality risk and benefits should be looked at using the decision to feed (intention to treat) as one does in practice.

A number of systematic reviews have tried to put sense to the poor evidence but given the heterogeneity of the feeds and the patients studied then for the various additional substrates it is not necessarily a standard ICU enteral feed. We do not know how applicable these results are to patients with more severe ARDS than in the study group or those early at risk of ARDS. The underlying genetic status of the patients may not only determine the severity of their ARDS but also their response to lipid modification.

In recent years small animal models have re-examined the appropriateness of enteral feeding in the very ill and septic subject. In these EN was associated with a worse mortality and was less effective at augmenting muscle and liver protein synthesis compared with PN. Interim analysis of a recent randomised multicentre study in Italy showed that the ICU mortality of patients with severe sepsis given EN was significantly higher than those for PN (44% v 14.3% p = 0.039).

Conclusion
A number of simple nutrition rules are clear. ICU patients staying more than a few days will need nutrition support and if malnourished they may need it sooner. Enteral feeding should be encouraged using simple feeding protocols and started early if safe to do so. However, it should not be forced if gastrointestinal intolerance is evident. We should use PN cautiously and follow guidelines of best practice. It is too easy to overfeed. It is reserved for those with verified gastrointestinal intolerance—that is, either in the context of gastrointestinal failure for total PN or gastrointestinal intolerance for supplemental PN. There is at present no evidence for “early” PN. We should ensure tight glycaemic control with insulin and use glutamine containing PN formulations in all-in-one mixtures.

Multiple choice questions (true (T)/false (F); answers at end of references)
A. Regarding nutritional status and nutritional requirements
1. The nutritional status has an effect on outcome (morbidity and mortality) in the critically ill.
2. If critically ill patients are severely under fed (<25% of target feed) they have a higher risk of nosocomial bloodstream infections.
3. All patients on intensive care have a similar energy expenditures of about 25 kcal/kg/day through their illness and are therefore fed using the same feeding regimen.
4. Over feeding is not a problem with patients on intensive care as the energy requirements are significantly increased.
5. Protein loss does not normally occur in critically ill patients and if present suggests starvation.

B. What is regarded as “best” nutrition in the critically ill and how should it be delivered?
1. Similar to starvation protein breakdown decreases when energy requirements are increasingly met by fat metabolism.

Can enteral feed modify or worsen specific disease processes?
From a background of detailed animal work a novel enteral feed containing a changed lipid profile and increased antioxidants moderated the severity of the initial inflammatory response in the lungs of patients developing acute respiratory distress syndrome (ARDS). Feeds with increased antioxidants alone have shown little discernible benefit. The combination of eicosapentaeenoic acid (EPA; fish oil), γ-linolenic acid (GLA; borage oil) had been shown to modulate membrane phospholipid composition with a shift towards a less inflammatory eicosanoid pattern.

Patients were recruited with firm pathological diagnosis, bronchoalveolar lavage was used to show a reduction in lung inflammation, and outcome measures showed improved parameters of gas exchange, reduction in duration of ventilation, decreased ICU stay, and decreased new organ failures. Subsequent analysis showed that the feed reduced the alveolar inflammatory response and their mediators to account for the improvements in gas exchange. However, the control group received a commercial high n-6 fat feed and not necessarily a standard ICU enteral feed. We do not know how applicable these results are to patients with more severe ARDS than in the study group or those early at risk of ARDS. The underlying genetic status of the patients may not only determine the severity of their ARDS but also their response to lipid modification.
(2) Increased proteolysis occurs (particularly skeletal muscle) in the critically ill to meet the increased metabolic demand for protein synthesis.

(3) Provision of high quality (essential and conditionally essential) amino acids along with insulin is central to good nutrition as amino acids are not stored but used in synthesis or metabolised during critical illness.

(4) Enteral feeding carries a lower risk of infections and is therefore superior to parenteral nutrition.

(5) Despite improved delivery systems and better quality of enteral feed there is still a number of patients who require parenteral nutrition because of gastrointestinal failure.

C. Regarding enteral nutrition use in the critically ill

(1) Upper digestive intolerance is a frequent event in the nasogastrically fed critically ill patient and is associated with pneumonia, longer ICU stay, and increased mortality.

(2) Upper digestive intolerance increases with sedation and cattacholamine use.

(3) Nasogastric feeding can cause non-occlusive bowel necrosis, a life threatening complication.

(4) Nasojejunal feeding is associated with fewer aspiration complications compared with nasogastric feeding.

(5) Several surveys suggest, that under feeding is common practice on intensive care when feeding enterally.

D. Further aspects of nutrition in intensive care

(1) Survival from intensive care improves when an evidence-based guideline for nutrition is followed.

(2) Glycemic control in patients receiving early enteral or parenteral nutrition in mandatory.

(3) There is good evidence supporting the use of immune nutrient cocktails in intensive care.

(4) A novel enteral feed containing a combination of eicosapentaenoic acid (EPA; fish oil) and γ-linolenic acid (GLA; borage oil) shows a reduction in lung inflammation.

(5) Strong evidence suggests that including glutamine within parenteral nutrition is preferred in the critically ill.

References


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**ANSWERS**

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