Enteral Feeding during Circulatory Failure: Myths and Reality

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Introduction

Cardiovascular disease is a major cause of morbidity and mortality in Western countries, being responsible for a large number of admissions to intensive care units (ICU), after acute myocardial infarction, cardiac surgery, or acute cardiomyopathies. Moreover, the prevalence of cardiovascular diseases is high in patients admitted for primary non-cardiac conditions, since a growing proportion of patients is over 65 years, and chronic cardiac heart failure has a prevalence of 30 – 130 individuals per 1000 in this age category [1].

The vast majority of patients undergoing cardiac surgery or with acute myocardial infarction do not require nutritional therapy, as they are able to resume oral feeding within 1 – 2 days. But some patients suffer a more complicated clinical course requiring pharmacological and/or mechanical cardiac support, as well as mechanical ventilation. Such patients are frequently hyper-catabolic, and dependent on artificial nutritional support for many days [2]. Enteral nutrition is considered necessary in acutely ill patients for a variety of metabolic, immune, and practical reasons. Nevertheless, enteral nutrition is commonly considered contraindicated and even hazardous during severe circulatory compromise. The American Society for Parenteral and Enteral Nutrition (ASPEN) 2002 guidelines state that enteral nutrition should be deferred until the patient is hemodynamically stable [2]. Defining stability may be difficult though in patients requiring prolonged inotropic therapy, as well as in patients requiring prolonged mechanical ventricular assistance, such as intra-aortic balloon pump (IABP) [3].

Indeed during cardiogenic shock, the splanchnic circulation is not spared. It may be altered after cardiopulmonary bypass (CPB), exposing the patient to the risk of gastrointestinal complications, particularly bowel ischemia. After cardiac surgery the prevalence of acute mesenteric ischemia varies between 0.5 % [4] and 1.4 % [5], and associated mortality is high ranging between 11 and 27 % [4, 5]. Further, bowel motility is reduced due to a combination of pyloric dysfunction, which is frequent in the critically ill [6], and intestinal atony. But the gut appears functional in many patients, even though bowel sounds are sparse [7], suggesting that enteral nutrition may be possible. This chapter describes under which conditions enteral nutrition is possible, and provides rationale for using specific nutrients and substrates.

Splanchnic Consequences of Feeding and of Circulatory Failure

The normal cardiovascular response to feeding is complex, including an increase in cardiac output, and vasodilation of mesenteric arteries, and a decrease in peripheral resistance. One of the components of this hemodynamic adaptation to feeding is an increase in local oxygen consumption (VO_2) which may decrease oxygen delivery (DO_2) to vital organs. In healthy subjects, enteral feeding induces increases in flow parameters in the superior mesenteric artery and portal vein in both genders [8]: A study enrolling 44 healthy subjects showed splanchnic postprandial hyperemia in response to intraduodenal feeding using Echo-Doppler technology. Postprandially, diastolic blood pressure fell, and flow in the portal vein increased (ns) and mean velocity in the superior mesenteric artery increased significantly. These changes were paralleled by alterations in systemic hemodynamics.

In acutely ill patients with cardiac failure, this response may be worsened by an already insufficient DO_2 to the tissues and organs. During low cardiac output, splanchnic DO_2 is reduced, while splanchnic VO_2 is maintained; therefore, splanchnic oxygen extraction is high [9]. This is one of the factors explaining the high rate of gastrointestinal complications in this category of patients [4, 5]. In patients with chronic heart failure, continuous enteric feeding set at 1.4 – 1.5 times resting energy expenditure, compared with intermittent feeding, has been shown to be well tolerated [10]. The authors concluded that enteral nutrition can be provided safely, except in patients with overt cardiac failure [10]. In another metabolic study in cardiac surgery patients with acute cardiac failure requiring inotropic support [11], the introduction of continuous enteral nutrition set at 110 % of resting energy expenditure caused a 10 % increase in cardiac index and splanchnic blood flow, a 10 % decrease in mean arterial pressure (MAP) in parallel with decreased systemic vascular resistance and unchanged heart rate [11]. Metabolic and endocrine responses indicated that nutrients were utilized as energy substrate: On initiation of enteral nutrition, glucose turnover increased, as did plasma glucose concentrations. These data suggest that careful limited continuous enteral feeding can be administered in patients with acute and chronic circulatory failure.

Gastrointestinal complications, and particularly bowel ischemia, are a serious threat after CPB. Indeed, this type of surgery includes periods of aortic cross clamping and of non-pulsatile blood flow, which affect both systemic and regional perfusion patterns. It predisposes the splanchnic region to inadequate perfusion and increases gut permeability. Splanchnic blood flow does not necessarily decrease during CPB nor after surgery as shown by two trials enrolling 10 patients each. In the first study, splanchnic blood flow was measured using infusion of indocyanine green (ICG) dye and low-dose ethanol from induction of anesthesia through hypothermic CPB and until 4 hours after surgery: Splanchnic blood flow and oxygenation parameters did not change significantly [12]. The second trial confirmed the absence of local or global splanchnic ischemia using intestinal laser Doppler flowmetry, gastric tonometry, and measurements of splanchnic lactate extraction [13]. A mismatch between splanchnic oxygen delivery and demand was seen in the latter trial, particularly during rewarming.

Circulating endotoxin increases during cardiac surgery, and may contribute to cytokine activation, high VO_2, and fever ('postperfusion syndrome') [14]. A trial enrolling 11,202 patients undergoing cardiac surgery requiring CPB with an overall mortality rate of 3 % and a 95 % autopsy rate, showed a 0.49 % incidence of acute mesenteric ischemia [4]. In another trial enrolling 2054 cardiac surgery patients,

postoperative gastrointestinal complications were even more frequent at 1.4 % [5]. Mortality associated with intestinal ischemia is high, at 11 % and above [4, 5], and increases with the need for gastrointestinal surgical intervention (44 % versus 0 % in patients not requiring surgery; $p < 0.01$). Risk factors for complications are duration of CPB and cross-clamp time, intra-aortic balloon pump (IABP) support, the development of post-operative renal failure, and operation type and priority [4, 5].
Cardiac surgery for coronary artery bypass grafting (CABG) using the off-pump technique is also associated with hemodynamic alterations, but there are very few data yet. In our experience, this technique is used preferentially in high risk patients and a complicated postoperative course is, therefore, not infrequent.

Bowel ischemia is favored by the abdominal compartment syndrome (ACS) with intra-abdominal pressures (IAP) > 20 mmHg: This complication does occasionally occur after cardiac surgery but is more frequent after descending thoracic and abdominal aortic surgery [15]. Monitoring of IAP belongs to standards of care after major vascular surgery [16].

In addition, the use of vasoactive drugs exerts unpredictable effects on splanchnic perfusion [17]. While dopexamine seems to improve splanchnic perfusion and gastric mucosal perfusion (as reflected by intramucosal pH [pHi]), all the other vasoactive drugs from dopamine to norepinephrine have unpredictable effects. The balance between the effects on systemic and splanchnic hemodynamics is affected by numerous and complex mechanisms, which explain such unpredictable effects.

Gastrointestinal motility is affected by a series of factors in cardiac surgery patients, and gastric emptying is significantly reduced in the postoperative period [18, 19]. Anesthesia, opioids, mechanical ventilation, vasoactive drugs, and sedatives reduce intestinal and gastric motility; these factors may contribute to difficult enteral feeding.

Nutritional Status of the Cardiac Patient

Malnutrition is present in up to 50 % of patients with severe congestive heart failure [2], exposing these patients to the risk of additional rapid malnutrition in absence of adequate support [20]. Cardiac cachexia is observed in patients with severe and prolonged cardiac failure: It has been recognized as an independent predictor of higher mortality in patients with chronic heart failure [21], while moderate obesity appears to be protective. Cardiac cachexia is also associated with poor outcome after cardiac transplantation, with an increase in 30-day mortality (13 versus 7 % in normal weight recipients) and a doubling of 5-year mortality [22]. A trial involving 5168 patients undergoing CABG [23], showed that the operative mortality was highest among those with both low body mass index (BMI < 20 kg/m^2) and albumin level below 25 g/l. The key role played by cardiac failure is illustrated by the progressive improvement in nutritional status after successful heart transplantation [24].

Surgical and medical cardiac patients share many characteristics: Both have chronic metabolic alterations involving mainly energy, carbohydrate, and lipid metabolism, but also may suffer from acute organ dysfunction due to ischemia. A variable degree of systemic inflammatory response syndrome (SIRS) is present in both categories of patients. Malnutrition worsens cardiac function whatever its initial cause: A trial in rats comparing *ad libitum* chow feeding or restriction to 50 % of this amount for 90 days, showed that malnutrition was associated with a reduction in left ventricular systolic function, and with lower contractility and compliance [25].

Nutritional assessment is challenging. The frequent presence of edema alters the validity of weight and calculated BMI. In cases where an accurate assessment is required, lean body mass determination by anthropometric measurements (skin fold thickness, arm-muscle circumference), or bioimpedance analysis enables an acceptable estimation of total body water. Practically, the clinician should consider actual weight, i.e., the weight just before the acute condition, recent weight loss, and clinical presentation of the patient. An unintentional weight loss of more than 7.5 % of previous normal weight has been shown to be an independent risk factor for mortality in chronic heart failure [21].

While hypermetabolism is not systematic after myocardial infarction, the majority of surgical patients are hypermetabolic and hypercatabolic as a consequence of the acute phase response triggered by surgery and circulating endotoxins [14]. The acute phase with its endocrine and metabolic consequences [3] contributes to the development of hospital malnutrition.

Energy Target and Substrate Requirements

In patients with cardiac failure, the appetite is poor, which contributes to cachexia [26]. Continuous enteric feeding, compared with intermittent feeding, has been shown to minimize VO_2 and myocardial VO_2: Therefore, enteral nutrition can be provided safely from the cardiac function aspect [27]. The combination of oral food and parenteral nutrition to achieve 20 to 30 kcal/kg per day for 2–3 weeks in patients with cardiac cachexia (severe mitral valve disease and congestive heart failure) is also associated with stable hemodynamics, unchanged whole body VO_2 and CO_2 production [28].

The level of energy requirement in critically ill patients is highly variable: Hypermetabolism is frequent, but in the presence of cachexia, the requirements tend to be below the values calculated with prediction equations. In such patients, determination of resting energy expenditure by indirect calorimetry is the only way to precisely determine their true metabolic rate. In our experience, the energy requirements can be set at 25 kcal/kg/day in most cases [11, 29]; lower requirements may be present in patients with severe persistent cardiogenic shock. Protein requirements do not differ from those of other patients and should be set at 1.3–1.5 g/kg/day.

Enteral Feeding Route

In acute conditions and especially in postoperative states, enteral nutrition is disregarded while parenteral nutrition is believed to be the only possible route of feeding. In circulatory compromise, enteral nutrition is indeed considered to be relatively contraindicated, as it may aggravate gut ischemia (steel); low mesenteric blood flow is a risk factor of bowel necrosis.

Our team has repeatedly shown that cautious enteral nutrition can be used even during severe cardiac compromise. Paracetamol (acetaminophen) absorption, which is very similar to that of protein absorption, is maintained in postoperative cardiac surgery patients even in low output states [19]: In a series of 23 patients with hemodynamic failure (cardiac index 2–2.5 $l/m^2/min$), paracetamol jejunal absorption was maintained compared with cardiac surgery controls without cardiac failure (**Fig. 1**).

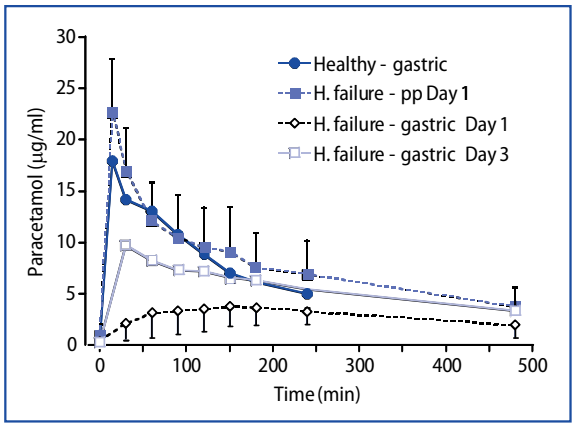

Fig. 1. Paracetamol (acetaminophen) kinetics on days 1 and 3 after cardiac surgery in patients with hemodynamic failure (H. failure) compared to 6 healthy controls having received gastric paracetamol. The figure shows the differences in gastric and postpyloric absorption over time. In the circulatory failure patients, the postpyloric (pp) absorption is already normal on day 1, while gastric absorption is delayed, and recovers slowly by day 3 (data from [19]).

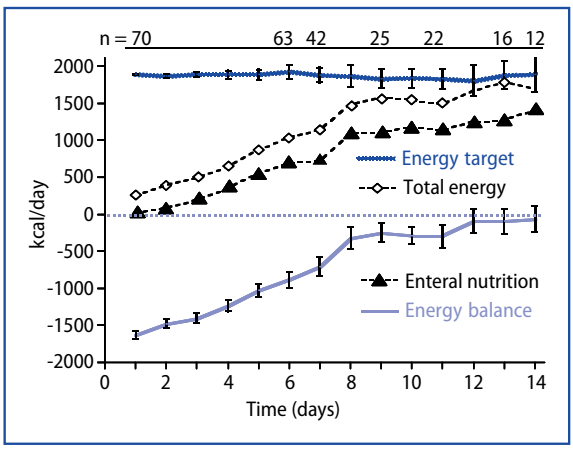

Fig. 2. Evolution of energy delivery and balance in 70 patients with circulatory compromise on pharmacologic and mechanical hemodynamic support [30]. Enteral nutrition, although possible, was not able to cover energy requirements set at 25 kcal/kg.

Such patients can be fed with caution first by the gastric route, or by the jejunal route if the gastric fails, causing large gastric residues. The introduction of enteral nutrition in patients with inotropic support after CPB causes increases in cardiac index and splanchnic blood flow, while metabolic response (endocrine profile) indicates that nutrients are utilized [11]. The data from this trial also suggest that the hemodynamic response to early enteral nutrition is adequate after cardiac surgery. Another recent trial in our ICU, including 70 patients with circulatory compromise, showed that the enteral feeding volume is limited in the presence of severe hemodynamic compromise [30]: As a mean, a maximum of 1000 ml may be delivered by the gastric route, and 1500 ml by the postpyloric route (**Fig. 2**). Among these 70 patients, 17 were dependent on IABP support; analysis of this subset of patients with extremely severe hemodynamic failure showed similar results, enabling the delivery of 15 – 20 kcal/kg/day by the enteral route. Nevertheless, we have repeatedly observed that although enteral nutrition is possible, the total energy delivery should be monitored as the limited feeding volume tolerance results in energy deficits over prolonged periods of nutritional support if the enteral route is used alone. In our prospective observational study [30], enteral nutrition was started at 20 ml/hour, and

increased stepwise, every 12–24 hours according to tolerance. Clinical criteria used to assess feeding tolerance were the volume of gastric residues (< or > 300 ml), the occurrence of abdominal distension, ileus, vomiting, broncho-aspiration of gastric contents, and impossibility to achieve energy target defined as energy delivery < 50 % of target for more than 3 days [30].

In ACS with an IAP > 20 mmHg, enteral nutrition should not be started, or should be discontinued or reduced to 500 ml/24 hours if already initiated.

Two reviews show that enteral nutrition is well tolerated and probably beneficial in most critically ill patients, as it contributes to restoring splanchnic perfusion and immune function [31, 32]. It should however be used with caution in patients during the shock phase [31].

Enteral Access

Enteral nutrition should be initiated by the gastric route in the absence of any contraindication. Alas, gastric feeding may be difficult in patients with cardiogenic shock, due to pyloric dysfunction [18], and reduced gastrointestinal motility. Gaining postpyloric access may solve this problem: Various techniques can be used, including blind manual placement, endoscopic placement, or fluoroscopic positioning. Endoscopic placement of the feeding tubes is considered a safe method of providing enteral nutrition, as shown by a retrospective study including 15 critically ill cardiothoracic surgery patients [33]; no complications of the procedure were observed. Blind placement in the ICU is worth attempting, and various placement techniques and feeding tubes have been advocated. Self-propelled feeding tubes are an alternative [34] although progression is lowest in those patients on the highest norepinephrine doses: Norepinephrine and morphine doses were the most important determinants of feeding tube progression [34].

Our ICU's enteral feeding protocol specifies a prudent increase in energy delivery to target over 4–5 days, with no enteral feeding during the first hours while in unstable shock, feeding being initiated after 24 to 48 hours.

Timing: Preoperative, Early or Conventional Feeding

Early enteral feeding is now supported by level A evidence [35]. According to international guidelines, cardiac surgery patients do not benefit from early enteral feeding [2], nor are they candidates for use of immunomodulating diets [36]. These guidelines require some discussion though.

Preoperative downregulation of the inflammatory response to surgery by an immunomodulating diet is a promising tool [37], as cardiac surgery typically elicits an inflammatory response [14]. The fish oil ω-3 fatty acids have been shown to have beneficial anti-inflammatory properties which make them candidates for nutritional intervention at the various stages of cardiac disease. Preventing such inflammatory responses may require preoperative intervention. A prospective randomized controlled trial enrolling 50 patients aged 70 years or older with poor ventricular function before cardiac surgery, investigated the effect of an oral supplement containing a mixture of immune-enhancing nutrients (arginine, ω-3 fatty acids and nucleotides) [37]. This trial showed that ≥ 5 days of supplementation improved the general immune response (stronger delayed-type hypersensitivity response), and was associ-

ated with a lower infection rate (4/23 vs 12/22, p = 0.013), a reduction in inotropic drug requirements, lower interleukin (IL)-6 concentrations, and a better preservation of renal function. These data suggest that routine preoperative nutritional intervention should be considered in elective cardiac surgery.

The early postoperative period should also be considered accessible to nutritional therapy. A study of 73 cardiothoracic ICU patients also reported the feasibility and good gastrointestinal tolerance of early enteral nutrition [38]. The only information about daily energy supply was that the energy target could be reached with enteral nutrition only in 9 patients (12 %). In our collective of patients with hemodynamic failure [30], more than 1200 kcal per day could be delivered by the enteral route in all the patients requiring artificial nutrition. The patients included in the study suffered critical circulatory failure as shown by their dependence on norepinephrine and other vasoactive drugs for hemodynamic stability.

Enteral, Intravenous, or Combined Nutrition?

The enteral route is the first choice in the majority of acutely ill patients. On the benefice side, continuous enteric feeding minimizes VO_2 and myocardial VO_2 in patients with congestive heart failure: Enteral nutrition is safe for cardiac function [27].

However, there are a few caveats and contraindications to enteral feeding due to the previously mentioned risk of bowel ischemia [4, 5]. Among these contraindications to enteral nutrition is the development of chylothorax after CABG [39], which favors parenteral nutrition in these patients; however, this complication is not an early complication and is usually diagnosed after several days. This complication may also occur in other types of cardiothoracic procedures in adults and children [40]. Most cases respond successfully to conservative treatment consisting of avoiding enteral nutrition (parenteral nutrition) and pleural drainage: The average duration of the lymph leak is 14 days. In some cases (less than 20 % in literature) a low-fat enteral diet can be used as initial treatment.

Gastrointestinal motility is inversely related to the dose of dopamine and norepinephrine as shown by the reduced migration of feeding tubes in patients on high doses of these drugs [34]. Nevertheless enteral energy delivery, although tending to be lower in patients on high norepinephrine and epinephrine doses [30], is not directly related to the dose as shown in **Figure 3**. Enteral feeding remains possible, although resulting in insufficient energy delivery. Interestingly, in our experience, only a few patients need prokinetic agents confirming the data by Kesek et al. [38], which stressed that gastro-intestinal motility, although impaired in some patients, was not the primary cause of enteral feeding failure. Moreover, in our series of 70 consecutive patients, none experienced any serious gastrointestinal complication.

Nevertheless, the most severely ill patients cannot be fed completely by the enteral route. Combined parenteral feeding is a true option in ICU patients staying longer than 5 days to prevent the installation of an energy deficit and to prevent the build up of energy deficits [20, 30, 41]. The combination of parenteral nutrition with oral food to achieve 20 to 30 kcal/kg/day for 2 – 3 weeks in patients with cardiac cachexia from severe mitral valve disease and congestive heart failure was associated with stable hemodynamics, unchanged whole body VO_2 and CO_2 production [28].

The concept of combined enteral and parenteral nutritional therapy opens new perspectives and questions. Should the supplement be a standard parenteral feed

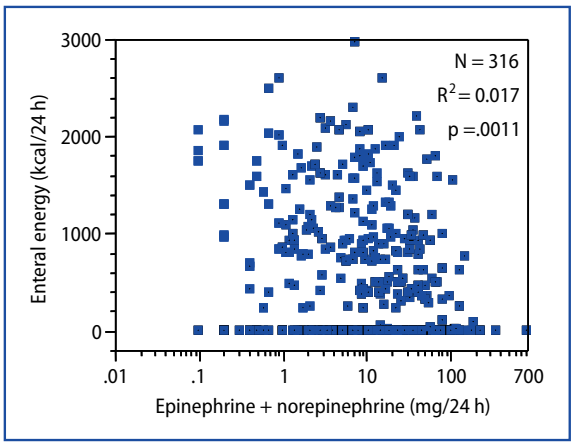

Fig. 3. Scattergram showing the absence of a direct relation between the amount of norepinephrine and epinephrine required for hemodynamic support (log scale) and the energy delivered with enteral feeding. The dose of vasopressor is expressed in mg/24 hours (total amount administered during 24 hours): 100 mg per 24 h is equivalent to 70 μg/min (data from [30]).

aimed at covering energy needs, i.e., provide glucose, amino acids and fat? Should the same types of substrates be delivered by both routes? Should specific nutrients, such as glutamine and antioxidant micronutrients, be delivered intravenously? Should unsaturated fatty acids with anti-inflammatory properties such as ω-3 polyunsaturated fatty acids be delivered intravenously?

Patient Monitoring

The most severe complication after cardiac surgery is splanchnic ischemia with the risk of bowel necrosis, and eventually death [4, 5]. Therefore, the clinical follow up during enteral nutrition includes a careful examination of the abdomen, watching for distension or other signs of sub-ileus. **Table 1** shows commonly encountered problems. Some paraclinical tools can assist the clinician: 1) Monitoring IAP should be the rule after major vascular surgery; any increase in pressure above 20 mmHg puts the gut at risk of ischemia; 2) gastric tonometry and mucosal PCO_2 are helpful; 3) monitoring of arterial blood gases (pH and lactate) can be used to confirm intestinal ischemia; decreasing pH and increasing lactate levels may herald the development of clinically relevant intestinal ischemia, but these are late and non-specific signs.

Daily monitoring of energy delivery should be part of the clinical management. The initial daily energy target should be set at 25 kcal/kg/day: If this target is not reached within 4 days, combination of enteral feeding with intravenous nutrition should be introduced rapidly to avoid the deleterious effects of negative energy balances [20].

The sequential organ failure assessment (SOFA) score [42] which is the most frequently used organ failure score in Europe, does not include any assessment of gastrointestinal function. Recently an additional gastrointestinal failure (GIF) component was proposed for this score [43]. This score will possibly enable detection of patients at the highest risk of intolerance to feeding, who should not be considered for early enteral nutrition.

Table 1. Common problems encountered during enteral feeding after cardiac surgery and proposed management (adapted from [19])

Problem	Diagnostic tool	Management	Target
Gastroparesis	Gastric residue > 300 ml	Postpyloric feeding Metoclopramide	Residue < 300 ml
Bowel ischemia	Splanchnic acidosis: ⇓ pHi ⇑ arterial lactate Abdominal distension, ⇑ IAP	Improve hemodynamics Gastric decompression (aspiration) Diuretics	Normal pHi (> 7.2) No distension Normal IAP
Abdominal compartment syndrome	⇑ IAP > 15 mmHg	Reduce fluid loading Diuretics Gastric decompression Reduce enteral nutrition to 500 ml/24 h if IAP > 20 mmHg and initiate combined parenteral feeding	IAP < 20 mmHg
Non occlusive bowel necrosis	Abdominal distension	Surgical resection Parenteral nutrition	No distension Resolution of shock
Gastrointestinal bleeding	Blood in nasogatric tube Endoscopic diagnosis	Prophylaxis: anti-H_2 drugs Treatment: proton pump inhibitors Enteral nutrition (?)	No bleeding
Diarrhea	> 5 liquid stools per day	Fiber	< 3 stools/day
Constipation	No stools for more than 5 days	Fiber Neostigmine (continuous or intermittent)	1 stool every 3 days
Acalculous cholecystitis	Abdominal ultrasound Lab: ⇑ alkaline phosphatase non-specific as in other ICU patients	Postpyloric feeding Interventional radiology Surgery	–

IAP: intra-abdominal pressure

Conclusion

The nutritional management of the patient with acute cardiovascular failure has changed over the recent years. These patients are at higher risk of splanchnic ischemia. Enteral nutrition is possible and safe, though requires close clinical supervision, but will invariably result in insufficient energy delivery prompting combined parenteral and enteral approaches. Based on indirect calorimetry data collected over the last 20 years in our investigation unit, the energy requirements can be set at 25 kcal/kg/day [11, 29], resulting in a mean target of 1900 kcal/day in this type of patient. This target should be reached over a period of 3–4 days, which is not possible by the enteral route alone in most cases. As a mean, 1250 kcal/day can be delivered by the gastric route with large inter-patient variability, and up to1500 kcal by the postpyloric route [30] (**Table 2**), which corresponds to 15–20 kcal/kg/day.

Table 2. Energy delivery according to the enteral feeding route in patients with compromised hemodynamic status (adapted from [30])

Route	Energy delivery (kcal/d)	Calculated energy balance (kcal/d)	Number of days (%)
Gastric	1250 ± 650[1,2]	–210 ± 680	276 (38.6 %)
Jejunal	1545 ± 720[1,2]	–170 ± 770	74 (10.3 %)

[1] Difference gastric versus jejunal energy delivery: ns
[2] Difference intravenous versus jejunal or gastric energy delivery: $p = 0.001$

In conclusion [30]: 1) Enteral nutrition is possible during the first postoperative week, and even already after 24 hours, in patients with acute severe circulatory failure under careful abdominal monitoring; 2) enteral nutrition generally results in insufficient energy delivery, stressing the importance of careful monitoring of the total daily energy delivery; and 3) combination with parenteral nutrition should be considered to achieve optimal energy delivery.

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