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Early metabolic and splanchnic responses to enteral nutrition in postoperative cardiac surgery patients with circulatory compromise

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Abstract *Objectives*: To assess the hemodynamic and metabolic adaptations to enteral nutrition (EN) in patients with hemodynamic compromise.
Design and setting: Prospective study in a university hospital surgical ICU, comparing baseline (fasted) with continuous EN condition.
Patients: Nine patients requiring hemodynamic support by catecholamines (dobutamine and/or norepinephrine) 1 day after cardiac surgery under cardiopulmonary bypass.
Intervention: Isoenergetic EN via a postpyloric tube while catecholamine treatment remained constant. Baseline (fasted) condition was compared to continuous EN condition.
Measurements and main results: Cardiac index (CI), mean arterial pressure (MAP), pulmonary and wedge pressures, indocyanine green (ICG) clearance, gastric tonometry, plasma glucose and insulin, and glucose turnover (6,6$^2H_2$-glucose infusion) were determined repetitively every 60 min during 2 h of baseline fasting condition and 3 h of EN. During EN CI increased (from 2.9 ± 0.5 to 3.3 ± 0.5 l min^{-1} m^{-2}), MAP decreased transiently (from 78 ± 7 to 70 ± 11 mmHg), ICG clearance increased (from 527 ± 396 to 690 ± 548 ml/min), and gastric tonometry remained unchanged, while there were increases in glucose (158 ± 23 to 216 ± 62 mg/dl), insulin (29 ± 23 to 181 ± 200 mU/l), and glucose rate of appearance (2.4 ± 0.2 to 3.3 ± 0.2 mg min^{-1} kg^{-1}).
Conclusions: The introduction of EN in these postoperative patients increased CI and splanchnic blood flow, while the metabolic response indicated that nutrients were utilized. These preliminary results suggest that the hemodynamic response to early EN may be adequate after cardiac surgery even in patients requiring inotropes.

Key words Carbon dioxide · Cardiac surgical procedures · Gastric mucosa · Lactate · Postoperative care · Splanchnic circulation

Introduction

Enteral nutrition (EN) is the currently recommended technique of artificial nutrition in critically ill patients. EN has many advantages over parenteral nutrition, such as maintenance of gut mucosal barrier function [1], reduction in nosocomial infections [2], better tolerance to carbohydrates, and reduced acute-phase response to inflammation or infection [3]. In addition to its metabolic effects, EN activates a series of physiological responses involving the digestive, cardiovascular, respiratory, and immune systems. In healthy subjects the digestion and absorption of nutrients induce typical hemodynamic changes, consisting of an increase in both cardiac output and mesenteric blood flow [4, 5].

EN is well tolerated in most ICU patients in whom the gastrointestinal tract is available as feeding route. In patients with severe circulatory failure, it is commonly thought that EN must be avoided since the systemic and splanchnic hemodynamic responses to nutrition may be altered, precluding a normal utilization of nutrients by the gastrointestinal tract [6]. However, preliminary observations from our ICU suggest that EN can be used in cardiac surgery patients with hemodynamic compromise [7]. In terminal cardiac failure the decreased cardiac output leads to splanchnic hypoperfusion and malnutrition, the so-called cardiac cachexia [8]. In this condition there is preliminary evidence that nutritional support may help improve the patient's clinical condition [9], although the hemodynamic adaptation to artificial nutritional is poorly understood.

We conducted a prospective observational study to assess the early systemic and splanchnic hemodynamic and the metabolic adaptation to EN, in patients actively treated for low cardiac output and hypotension on the day after cardiac surgery.

Materials and methods

The study was conducted according to the principles established in the Declaration of Helsinki and was approved by the Ethics Committee of Lausanne University School of Medicine. Nine patients scheduled for cardiac surgery under cardiopulmonary bypass were enrolled after providing written, preoperative, informed consent. Included were high-risk patients with either reduced left ventricular function or complex valvulopathy, requiring a perioperative pulmonary artery catheter for circulatory monitoring as assessed by the anesthesiologist. Exclusion criteria were: absence of informed consent, age over 75 years, presence of preoperative liver, renal, endocrine or metabolic disorder (in particular diabetes requiring oral or insulin treatment), and cardiogenic shock.

The patients were anesthetized by a standard technique, including etomidate, fentanyl, and vecuronium for induction, and midazolam and fentanyl for maintenance. Cardiopulmonary bypass was performed under moderate hypothermia (28–32 °C), using a membrane oxygenator and a nonpulsatile flow. At the end of anesthesia patients were transferred to the ICU, where they were managed according to a standardized protocol, including: (a) controlled mechanical ventilation and respiratory weaning; (b) morphine and propofol for sedation and analgesia; (c) fluid resuscitation with normal saline and starch solutions; (d) blood transfusion to maintain hemoglobin concentration at 9.0 g/dl or higher; (e) dobutamine as inotrope after full fluid and blood resuscitation; (f) norepinephrine in continuous infusion to achieve mean arterial pressure of 70 mmHg or higher. Femoral arterial and thermodilution pulmonary arterial catheters (Thermodilution catheter, Abbott, North Chicago, Ill., USA) were used for monitoring and blood sampling. Upon ICU arrival a postpyloric feeding tube was inserted under endoscopic control.

Study design

Patients were studied in the morning of the first postoperative day after a total fasting period of 30 h. Each patient was his own control: data obtained during EN were compared to baseline fasted data.

General procedures

Prior to the study the anthropometric data and the operative risk score [10] were recorded, and biochemical measurements were performed. The experiment lasted 300 min, starting with a baseline period of 120 min. Thereafter a continuous infusion of enteral polymeric diet containing 22 % of calories as protein, 53 % as carbohydrate, and 25 % as fat at 130 % of the measured energy expenditure (see below) was administered for 180 min. The following variables were measured: systemic and pulmonary hemodynamics, ST segment analysis, pulmonary gas exchange, gastric tonometry, indocyanine green (ICG) clearance, and glucose turnover. Hemodynamic measurements included mean arterial pressure (MAP), heart rate, cardiac index (CI) by thermodilution (average of five values using 10 ml glucose at room temperature, regardless of the respiratory cycle), pulmonary artery pressure (PAP), and pulmonary artery wedge (PAWP) pressure. Indexed systemic vascular resistance (SVRI) was computed. The measurements were repeated every 60 min. The infusion of catecholamines remained constant during the study period, and PAWP was maintained constant by fluid infusion. Electrocardiographic monitoring for cardiac arrhythmia and ST segment analysis was recorded continuously (II and V_5, Merlin, Hewlett-Packard, Geneva, Switzerland). Oxygen consumption (VO_2), carbon dioxide production (VCO_2), and energy expenditure were determined using a Deltatrac indirect calorimeter (Datex Instruments, Helsinki, Finland) and the de Weir equation [11]. This measurement was performed by collecting the ventilator expiratory gases or with a ventilated hood in extubated patients. In three patients already extubated who did not tolerate the hood, VO_2 was determined by the Fick method, based on cardiac output by thermodilution and cooxymetry determinations of arterial (SaO_2) and mixed-venous (SvO_2) O_2 saturation [12]. Energy expenditure was then computed by means of the de Weir equation [11], assuming a respiratory quotient of 0.8. Gastric tonometry was performed every 60 min by a Trip NGS Catheter (Tonometrics, Worcester, Mass., USA) using a buffer solution [13], a 238 pH blood gas analyzer (Ciba-Corning, Basel, Switzerland), and the correction factor provided by the manufacturer. The pHi and the arteriomucosal gradient of CO_2 were computed according to standard formula [14]. ICG clearance was determined before (at 90 min) and during EN (at 270 min) [15]. A primed (12 mg bolus) infusion of ICG (Ic-Green, Akorn, Decatur, Ill., USA) was administered at 1.2 mg/min for 30 min. Two arterial blood samples were drawn after 25 and 30 min of infusion. Plasma concentrations were measured with a spectrophotometer at 805 nm (Perkin Elmer Lambda 2, Norwalk, Conn., USA), and the two values were averaged.

After blood collection to determine basal glucose isotope enrichment a primed continuous infusion of $6,6^2H_2$-glucose (prime 6 mg kg^{-1} $mmol^{-1}$ fasting plasma glucose, continuous 60 μg kg^{-1} min^{-1}) was started. After allowing 2 h for tracer equilibration, blood samples were collected at 60-min intervals for calculation of glucose turnover from $6,6^2H_2$-glucose enrichment. In addition, plasma glucose, insulin, free fatty acid, and lactate concentrations were monitored.

Analytical procedures

Plasma $6,6^2H_2$-glucose was measured by gas chromatography–mass spectroscopy on a Hewlett Packard instrument (GC

Table 1 Anthropometric and clinical chemistry data (*CRP* C-reactive protein, *AST* aspartate aminotransferase, *ALT* alanine aminotransferase, *GGT* γ-glutamyl transferase)

	Mean ± SD	Reference value	SI units
Sex (M/F)	5/4		
Age (years)	64.7 ± 1.9		
Height (cm)	166.6 ± 8.5		
Weight (kg)	73.8 ± 22.4		
CRP (mg/dl)	7.1 ± 2.1	< 1	71 ± 21 mg/l
Albumin (g/dl)	3.2 ± 0.5	33–53	32 ± 5 g/l
AST (U/l)	73 ± 35	14–50	73 ± 35 U/l
ALT (U/l)	26 ± 19	11–60	26 ± 19 U/l
GGT (U/l)	37 ± 44	11–62	37 ± 44 U/l

5890-MS 5971, Hewlett Packard, Palo Alto, Calif., USA). For this, pentacetylglucose derivatives were prepared and analyzed in chemical ionization made with selective monitoring of m/z 331 and 333. Plasma glucose was measured with a Beckman glucose analyzer II (Beckman Instruments, Fullerton, Calif., USA). Glucose rates of appearance and disappearance were calculated with Steele's equations for non-steady-state conditions [16]. Exogenous glucose infusion, when present, was subtracted from the glucose rate of appearance to obtain endogenous glucose production. Plasma lactate was measured with a Yellow Spring lactate analyzer (Yellow Spring Instruments, Yellow Springs, Ohio, USA). Free fatty acids were measured enzymatically using a kit from Wako (Freiburg, Germany). Concentrations of insulin (kit from Biochem Immunosystems, Freiburg, Germany), and cortisol (kit from Diagnostic Products Corporation, Los Angeles, -Calif., USA) were measured by radioimmunoassays. Growth hormone concentrations were determined by a chemiluminescence immunometric assay (Nichols Institute, San Juan Capistrano, Calif., USA). Plasma catecholamines (epinephrine and norepinephrine) were measured by HPLC with electrochemical detection [17].

Statistical analysis

Values are expressed as mean ± SD. The mean values determined every 60 min were compared over time by one-way analysis of variance for repeated measurements. When the effect of time was significant, the values at each time were compared to the value at time 0 (t_0) by Dunett's tests. The level of $p < 0.05$ was considered statistically significant. Statistical analysis was performed using JMP Statistical software version 3.5.1 (SAS Institute, Cary, N.C., USA) [18].

Results

The nine patients were studied 12–16 h postoperatively. Their anthropometric, biochemical, and clinical data are summarized in Tables 1, 2, and 3. Six were extubated, and the other three were on mechanical ventilation; none presented any significant hemorrhage during the study period. All patients required dobutamine infusion to support their hemodynamics (mean 420 μg/min; see Table 3), and four required norepinephrine to maintain a minimum MAP at 70 mmHg (range 6–30 μg/min). At baseline the mean CI was to 2.9 ± 0.5 l min^{-1} m^{-2} (Fig. 1), and lactate levels were within the normal range (Table 4). EN was 1.1 ± 0.25 kcal kg^{-1} h^{-1} and was clinically well tolerated. No gut distention was observed, and no patient complained of an adverse event during the study period.

The hemodynamic response

The hemodynamic response to EN is presented in Table 4. No statistically significant change was observed during baseline examinations (t_0–t_{120}), demonstrating the patients' stable condition in the absence of nutrition. The introduction of EN was associated with a progressive increase in CI that reached statistical significance after 2 h of EN (+10 % at t_{240}). This increase was related to a rise in systolic volume since heart rate remained constant. Meanwhile MAP decreased transiently after the introduction of EN (–11 % at t_{180}), and SVRI decreased (–16 % at t_{180}). PAWP remained constant (range 13 ± 3 to 14 ± 4 mmHg). No change was observed in ST segment analysis. ICG clearance, calculated 3 h after EN initiation, increased significantly versus baseline (+31 %; Table 4). The gradient between gastric mucosa and arterial PCO_2 remained within the normal range and did not change throughout the experimental period (Table 4).

The metabolic response

No metabolic change was observed during the baseline period. Mean glucose was 158 ± 23 mg/dl (8.8 ± 1.3 mmol/l), insulin 29 ± 30 mU/l, and free fatty acid

Table 2 Endocrinology and energy metabolism data (VO_2 O_2 uptake, VCO_2 CO_2 production, *EE* energy expenditure)

	60 min	120 min	240 min	300 min
Epinephrine (pg ml^{-1})	–	228 ± 144	–	203 ± 135
Norepinephrine (pg ml^{-1})	–	1128 ± 654	–	1407 ± 242
Cortisol (nmol l^{-1})	–	692 ± 241	–	548 ± 142
Growth hormone (μg l^{-1})	–	4.0 ± 4.0	–	7.0 ± 6.4
VO_2 (ml min^{-1} m^{-2})	209 ± 42	–	237 ± 50	–
VCO_2 (ml min^{-1} m^{-2})	169 ± 34	–	200 ± 46	–
EE (kcal per day)	1425 ± 288	–	1627 ± 355	–

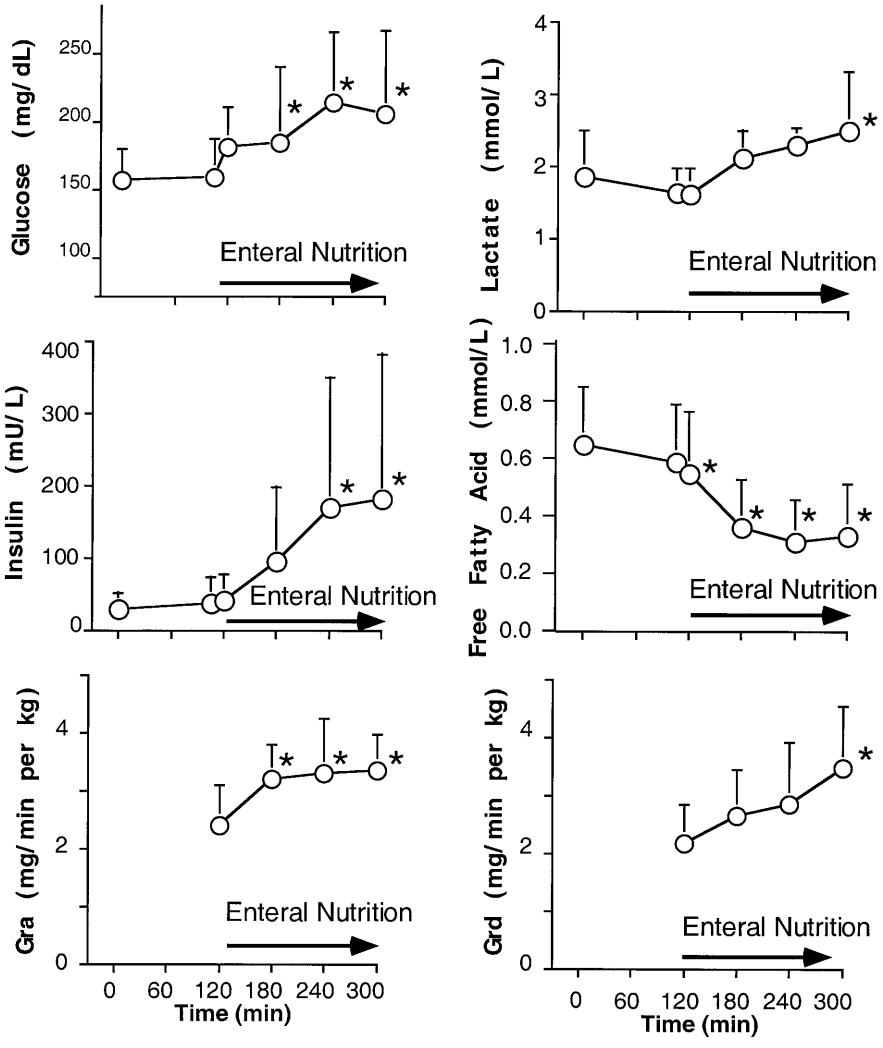

Fig. 1 Metabolic effects of enteral nutrition on metabolic variables and on glucose turnover determined by 6,6$^2H_2$-glucose infusion. *Gra* Glucose rate of appearance; *Grd* glucose rate of disappearance; *p < 0.05 vs. t_0

plasma level 0.64 ± 0.2 mmol/l. EN was associated with an increase in plasma glucose (37%), while the free fatty acid level decreased (51%). Insulin plasma levels were high during baseline and increased markedly with EN (+500%; Fig. 1). The glucose rate of appearance increased immediately after the start of EN (37%), while its rate of disappearance increased significant only after 3 h of EN (54%; Fig. 1). Plasma lactate increased with EN (34%). No significant change in epinephrine, norepinephrine, cortisol, or growth hormone occurred over time (Table 2).

Discussion

EN induced significant physiological responses in postoperative cardiac surgery patients under catecholamine support. The hemodynamic response consisted of an increase in cardiac output, decrease in systemic vascular resistance, and increase in splanchnic blood flow, while the metabolic response indicated that nutrients were utilized.

Systemic hemodynamic response

The effect of EN on cardiac output constitutes a major determinant of the hemodynamic response to feeding. In normal subjects EN increases cardiac output, with no concomitant change in systemic pressure [19, 20, 21]. The magnitude of increase in cardiac output depends on several variables, including the composition and the size of the meal: the larger the food intake, the greater the increase in cardiac output [4, 22]. In our patients we observed a small but significant increase in stroke volume and cardiac output and a decrease in MAP and SVRI. These hemodynamic changes were limited, requiring no change in norepinephrine administration. The observation of an increased cardiac output, with unchanged heart rate, indicates that stroke volume

Table 3 Clinical data [*Dob* dobutamine infusion (μg/min), *NE* norepinephrine infusion (μg/min), *LVEF* left ventricle ejection fraction, *CABG* coronary artery bypass graft, *VSD* ventricular septal defect]

Patient no.	Age (years)	Diagnosis	Operation	Catecholamines
1	57	Mitral stenosis + aortic regurgitation	Aortic + mitral valve replacement	Dob 300, NE 6
2	63	Aortic stenosis + congestive cardiomyopathy (LVEF 25%)	Aortic valve replacement	Dob 800
3	70	Coronary disease + ventricular septal defect	3 vessels CABG + VSD closure	Dob 200, NE 7
4	65	Aortic stenosis + mitral regurgitation	Aortic valve replacement + mitral annuloplasty	Dob 200, NE 8
5	55	Coronary disease + congestive cardiomyopathy (LVEF 30%)	3 vessels CABG	Dob 200
6	70	Mitral + aortic regurgitation	Aortic valve replacement + mitral annuloplasty	Dob 300
7	67	Coronary disease + congestive cardiomyopathy (LVEF 30%)	2 vessels CABG + reoperation for hemostasis	Dob 700, NE 25
8	72	Mitral regurgitation	Mitral annuloplasty	Dob 500
9	67	Mitral + aortic regurgitation	Aortic + mitral valve replacement	Dob 600

increased. This can be attributed to a decrease in the afterload of the left ventricle, as evidenced by the decrease in SVRI. An improvement in contractility may also have occurred, a condition difficult to ascertain with a concomitant reduction in afterload. To our knowledge there are no published data on the acute hemodynamic effect of EN during the early postoperative period after cardiac surgery. In patients scheduled for mitral replacement the use of a mixed nutrition (enteral and parenteral) administered 3 weeks preoperatively and 3 weeks postoperatively was associated with an increase in cardiac output and a decrease in SVRI [9].

Splanchnic hemodynamic response

In healthy subjects EN produces a prompt increase in mesenteric blood flow that can reach two to three times fasting value [5, 22] and an increase in splanchnic O_2 consumption [23]. Very similar hemodynamic responses have been reported in healthy subjects after the administration of either a meal in the stomach, or a postpyloric continuous infusion [5]. The latter method of nutrition was used in the present study since delayed gastric emptying is frequent after cardiac surgery [7]. In our critically ill patients EN significantly increased ICG clearance, suggesting an increase in mesenteric blood flow, and

Table 4 Hemodynamic data (*HR* heart rate, *CI* cardiac index, *MAP* mean arterial pressure, *SVRI* systemic vascular resistance index, *PAP* pulmonary arterial pressure, *PAWP* pulmonary wedge pressure, $PaCO_2$ arterial PCO_2, PCO_2 gradient between gastric tonometry and arterial PCO_2, *pHi* tonometric pH, SaO_2 arterial O_2 saturation, SvO_2 mixed-venous O_2 saturation, *ICG Cl* clearance of the indocyanin green

	Fasted			Enteral nutrition		
Time (min)	0 min	60 min	120 min	180 min	240 min	300 min
HR (bpm)	99 ± 13	98 ± 13	101 ± 14	98 ± 14	98 ± 13	100 ± 16
CI (l min^{-1} m^{-2})	2.9 ± 0.5	3.0 ± 0.4	3.0 ± 0.4	3.1 ± 0.3	3.2 ± 0.5*	3.3 ± 0.5*
MAP (mmHg)	78 ± 7	79 ± 7	77 ± 9	71 ± 9*	73 ± 7	78 ± 11
PAP (mmHg)	25 ± 7	25 ± 6	24 ± 8	24 ± 8	27 ± 1	25 ± 9
SVRI (dyne s^{-1} cm^{-5})	2176 ± 378	2131 ± 313	2114 ± 362	1838 ± 278*	1836 ± 261*	1978 ± 162*
PAWP (mmHg)	13 ± 3	14 ± 2	13 ± 3	13 ± 4	14 ± 4	14 ± 5
$PaCO_2$ (mmHg)	42 ± 4	42 ± 4	43 ± 4	41 ± 4	42 ± 5	42 ± 5
PCO_2 (mmHg)	6 ± 1	6 ± 1	6 ± 1	6 ± 1	6 ± 1	
pHa	7.39 ± 0.04	7.35 ± 0.04	7.35 ± 0.04	7.35 ± 0.4	7.34 ± 0.04	7.35 ± 0.04
SaO_2	0.97 ± 0.01	0.97 ± 0.01	0.96 ± 0.01	0.97 ± 0.01	0.96 ± 0.01	0.96 ± 0.01
SvO_2	0.68 ± 0.06	0.68 ± 0.06	0.69 ± 0.04	0.70 ± 0.06	0.69 ± 0.05	0.68 ± 0.06
ICG Cl (ml/min)	–	–	527 ± 396	–	–	690 ± 548*

*$p < 0.05$ vs. t_0

did not affect gastric tonometry values. The latter were in the normal range, suggesting that gastric mucosal blood flow matched the demand during the fasted and the absorptive conditions. This observation contrasts with findings in other studies indicating that cardiac surgery under cardiopulmonary bypass may induce alterations in gastric tonometry during the first postoperative hours, even in patients with satisfactory systemic hemodynamics [24, 25]. Likewise, dobutamine may increase both global and splanchnic blood flow, concomitant with a decrease in gastric pHi after cardiac surgery [26]. Of note, these studies focused on the first postoperative hours. By contract, the present study was performed on the first postoperative day, while the inflammatory response and the circulatory alterations may have receded.

The two methods used to assess splanchnic hemodynamics indicate that mesenteric blood flow increased, and that perfusion remained adequate during EN. Since mesenteric blood flow accounts for 10–15 % of cardiac output under fasted conditions, a marked splanchnic vasodilatation is likely to induce a large increase in the fraction of cardiac output going to the digestive system and may contribute to the decrease in global systemic vascular resistance.

To interpret the results of the present study, the methods used to assess splanchnic hemodynamics and their limitations must be considered. ICG clearance and gastric tonometry were used to assess splanchnic blood flow indirectly. ICG clearance depends on both liver blood flow and liver dye extraction. The measurement of liver extraction requires catheterizing the suprahepatic veins, an invasive procedure that was not performed in this observational study. We relied on ICG clearance to assess changes in mesenteric blood flow, based on the following assumptions. First, there was no change in liver extraction, as shown in ICU patients operated on for coronary artery bypass grafting, in whom ICG liver extraction was similar to normal subjects [15]. Second, there was no increase in hepatic arterial blood flow during EN. This supposition is consistent with the literature on healthy subjects [27] and conforms to the concept of hepatic artery "buffer response" [28]. Thus, if all these assumptions are correct, the increase in ICG clearance could indicate an increase in splanchnic blood flow.

The second method used to assess regional blood flow was gastric tonometry. This measurement can detect hypoperfusion in mucosa of the digestive tract, as demonstrated in experimental [14] and clinical studies [29]. There is recent evidence that this method remains valid during postpyloric feeding [30]. Based on this indirect approach, we found no evidence of hypoperfusion of the gastric mucosa during EN.

Plasma lactate concentrations increased during EN. This finding may be related to a metabolic mechanism since glucose administration increases lactate production via a stimulation of the glycolytic pathways [31]. Indeed, in the present study isotopic data indicate that glucose rate of disappearance was markedly enhanced. Although the occurrence of dysoxia, related to insufficient increase in cardiac and splanchnic blood flow during EN, cannot be ruled out in patients after cardiac surgery, it is a less likely explanation since cardiac output and ICG clearance increased throughout the study. This observation highlights the multiplicity of mechanisms responsible for lactate increase in ICU patients.

Considering various aspects of the integrated response to EN, i.e., normal fasted cardiac output and SvO_2, as well as the observation of increased cardiac output and ICG clearance, associated with unchanged SvO_2 and tonometric PCO_2 during EN, the most likely interpretation of our results is that an appropriate systemic and splanchnic hemodynamic response to nutrition occurred. However, other factors cannot be ruled out since there was no control group.

Metabolic response

EN increased plasma glucose concentration and glucose rate of appearance. An increase in glucose rate of appearance of $1\ mg\ kg^{-1}\ min^{-1}$ corresponds roughly to one-half the dose of glucose administered enterally. This indicates that a substantial portion of the glucose administered was absorbed. Glucose rate of disappearance, i.e., the amount of glucose that was utilized (oxidized or stored), also increased with a delay of 2 h, a finding consistent with the plateau in plasma glucose observed at that time. Insulin concentrations increased concomitantly to glycemia, an expected response. Likewise, the decrease in free fatty acid concentration can be explained by the effect of insulin on lipid metabolism.

Indirect calorimetry to target the amount of energy administered was not always possible. In some patients energy expenditure was computed from O_2 consumption, a less accurate method.

Clinical implications

Patients with acute circulatory failure may not be able to adapt to the physiological stress induced by EN. Such concern has led to the widespread attitude to restrict EN in cardiac patients treated with large doses of inotropes or vasopressors, in whom enteral feeding may provoke enteral distention and ischemia [6]. Very few studies have assessed the hemodynamic effect of artificial nutrition in patients suffering from circulatory failure, a relevant question considering the potential benefits of EN in critically ill patients. There is limited

evidence that prudent nutritional support is beneficial in patients suffering from cardiac failure, particularly those with cachexia [9]

To our knowledge, this is the first study assessing acute adaptation to isoenergetic EN in critically ill patients suffering hemodynamic compromise. A first point is to determine whether the nutrition was absorbed and metabolized. Indeed, a delay in gastric emptying has been reported after cardiac surgery [7] while the absorption of paracetamol, a semiquantitative probe for intestinal absorption, was normal in patients after cardiac surgery when administered via a postpyloric tube. Accordingly, the latter route was used in our patients, in whom the metabolic response to EN indicates that nutrients were absorbed, and suggests that their utilization was adequate.

Another potential benefit of EN resides in its ability to vasodilate the splanchnic vascular bed while maintaining the balance between blood flow and metabolism. This aspect could be particularly beneficial in cardiac patients who exhibit preponderant splanchnic vasoconstriction during cardiac failure [32]. It should be underlined that our results are valid only for postoperative cardiac surgery patients requiring inotropes, not for other populations such as patients in cardiogenic shock or with organ failure. Our findings may thus not apply to patients with more severe circulatory compromise or with metabolic failure. Further studies are necessary to determine the effect of prolonged EN on patients' clinical outcome.

Conclusions

In a short-term observational study we found that postoperative patients requiring inotropic and/or vasopressor therapy after cardiac surgery had increased CI and SVRI 3 h after the initiation of isoenergetic enteral nutrition. During the observation period of 3 h there was no evidence of digestive ischemia based on gastric tonometry and ICG clearance. These findings suggest that the circulatory response to EN is adequate, even in patients requiring inotropes therapy, and deserves further studies.

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