

Chapter 16

Guidelines for specialized nutritional and metabolic support in the critically-ill patient. Update. Consensus SEMICYUC-SENPE: Neurocritical patient

J. Acosta Escribano^a, I. Herrero Meseguer^b and R. Conejero García-Quijada^c

^aHospital General Universitario. Alicante, Spain. ^bHospital Universitario de Bellvitge. Barcelona, Spain. ^cHospital Universitario San Juan. Alicante, Spain.

Abstract

Neurocritical patients require specialized nutritional support due to their intense catabolism and prolonged fasting. The preferred route of nutrient administration is the gastrointestinal route, especially the gastric route. Alternatives are the transpyloric route or mixed enteral-parenteral nutrition if an effective nutritional volume of more than 60% cannot be obtained.

Total calorie intake ranges from 20-30 kcal/kg/day, depending on the period of the clinical course, with protein intake higher than 20% of total calories (hyperproteic diet). Nutritional support should be initiated early.

The incidence of gastrointestinal complications is generally higher to other critically-ill patients, the most frequent complication being an increase in gastric residual volume. As in other critically-ill patients, glycemia should be closely monitored and maintained below 150 mg/dL.

Nutr Hosp 2011; 26 (Supl. 2):72-75

Key words: *Neurocritical patient. Traumatic brain injury. Early nutrition. Hyperglycemia.*

RECOMENDACIONES PARA EL SOPORTE NUTRICIONAL Y METABÓLICO ESPECIALIZADO DEL PACIENTE CRÍTICO. ACTUALIZACIÓN. CONSENSO SEMICYUC-SENPE: PACIENTE NEUROCRÍTICO

Resumen

El enfermo neurocrítico precisa un soporte nutricional especializado debido a su intenso catabolismo y a un prolongado período de ayuno. La vía de administración nutricional preferente es la gastrointestinal, particularmente la vía gástrica, siendo alternativas la vía transpilórica o la nutrición mixta enteral-parenteral en caso de no obtener un volumen nutricional eficaz superior al 60%.

El aporte calórico total oscila entre 20-30 kcal/kg/día, según el período de evolución clínica en que se encuentre, con un aporte proteico superior al 20% de las calorías totales (hiperproteico). El inicio del aporte nutricional debe ser precoz.

La incidencia de complicaciones gastrointestinales es superior al enfermo crítico en general, siendo el aumento del residuo gástrico el más frecuente. Debe establecerse un estrecho control de la glucemia, manteniéndose por debajo de 150 mg/dl como en el resto de los enfermos críticos.

Nutr Hosp 2011; 26 (Supl. 2):72-75

Palabras clave: *Paciente neurocrítico. Traumatismo craneoencefálico. Nutrición precoz. Hiperglucemia.*

Introduction

Neurocritical patients with brain injury (BI), ischemic or bleeding stroke, or tumor disease, often differ from critically-ill patients in general in several aspects:

– They require drugs and techniques that modify their metabolic status: sedatives, analgesics, barbitu-

rates, muscle relaxation and occasionally hypothermia¹, for at least 5 days, to induce a deep sedation and adequate control of intracranial hypertension.

– BI has a greater incidence in young people and subarachnoid bleeding affects patients between the fourth and sixth decades of life, with adequate nutritional status and, generally, without associated comorbidities. The neurocritical patient with non-subarachnoid vascular disease is generally older, shows a high incidence of metabolic disorders, such as diabetes and hypertriglyceridemia, and the extent of brain recovery is lower, with the resulting longer stay in the ICU^{2,3}.

– Brain injuries cause gastrointestinal complications, particularly delayed gastric emptying, evidenced

Correspondence: J. Acosta Escribano.
Hospital General Universitario.
Alicante, Spain.
E-mail: acostesc@gmail.com

SEMICYUC: Spanish Society of Intensive Care Medicine and Coronary Units.
SENPE: Spanish Society of Parenteral and Enteral Nutrition.

as increased gastric residue (IGR)⁴ in patients receiving enteral nutrition (EN).

– In general, the clinical stabilization period is not long, though the use of vasoactive drugs is common, due to the associated injuries or the need for maintaining an adequate brain perfusion pressure.

– They require long periods of mechanical ventilation related to their low neurological level.

– The neurocritical patient of traumatic etiology develops hypermetabolic and hypercatabolic responses, with a severity not clearly related to severity levels as measured by the Glasgow scale (GCS). Thus, the lower coma grades (GCS, 4-5) show a greater energy expenditure than the higher (GCS, 8-11), and these in turn higher than the intermediate (GCS, 6-7)⁵.

– The duration of metabolic response is long, with a peak maximum activity around 2 weeks after admission and a more moderate persistence from the third week⁶.

What are the recommended administration routes in neurocritical patients? How can the requirements be calculated?

Specialized nutritional support in neurocritical patients is essential, due to their hypercatabolism and as generally the period with no oral intake and on mechanical ventilation is longer than 3 days⁷ (Ib). Administration should be performed early⁸⁻¹¹ (Ib), as in all other critically-ill patients, and preferred administration route is the enteral¹² (Ib). A large study in patients with BI evidenced that a cumulative energy deficit in the first 5 days of progress is related to an increased mortality of 30-40% per 10 kcal/kg of cumulative deficit¹³ (Ib). However, there are very few studies comparing early and late EN in neurocritical patients¹⁴.

The semi-seated position, with the head of the neurocritical patient elevated 30°, improves brain distensibility, significantly reduces intracranial pressure^{15,16} (IIb) and the risk of bronchial aspiration¹⁷ (IIa).

Except if there is a formal contraindication or if the volume administered with EN is less than 60% of the scheduled volume, the nutrient supply route in neurocritical patients is the enteral. However, there are not enough studies supporting the advantages of EN in contrast to parenteral nutrition (PN). The use of barbiturates for deep sedation is a factor determining intolerance to EN, so the use of PN is preferred in these cases¹⁸ (IIa).

Monitoring and evaluation of calorie intake should be performed using indirect calorimetry, which allows for calculating the total energy expenditure (TEE), the respiratory quotient, and consumption and use of the different substrates^{19,20} (Ib). When indirect calorimetry is not available, several formulae have been proposed for estimating the TEE, applying a correction factor within 1.2-1.4 of the basal energy expenditure. However, based on the severity and evolutive patient status,

the proposed values for correction factors may underestimate or overestimate calorie needs. Therefore, an adequate calorie intake may be about 20-25 kcal/kg/day in patients with muscle relaxation, and about 25-30 kcal/kg/day in sedated patients. Several factors advise reducing calorie intake, including sedation 20%, analgesia with morphine derivatives 8%, muscle relaxation of 12-28%, treatment with barbiturates of 13-32% and hypothermia or beta blockers 5%²¹ (III).

What substrates should be administered to a neurocritical patient?

Calorie supplies should be given by administration of glucose, with supplies under 5 g/kg/day and fats of 0.7-1.5 g/kg/day. Protein supply is about 1.3-1.5 g/kg/day in the acute phase and 1.3 g/kg/day from the second week. According to the increase in protein needs a calorie intake of protein origin over 20% of the total calorie supplies must be maintained²² (III).

Glutamine is an essential amino acid in stress states²³. Its administration as dipeptide by the parenteral route in critically-ill patients with injuries²⁴ showed a decrease in infectious complications and mortality^{25,26} (Ia). Their use in BI has been limited because of the theoretical risk of causing an increase in intracerebral glutamate values, leading to an increase in neuronal damage, cerebral edema, and increased intracerebral pressure. Two studies have concluded that the use of intravenous glutamine increases glutamate plasma values, without changes in intracellular values of intracerebral glutamine^{27,28} (Ib). A study in neurocritical patients with enteral glutamine²⁹ (IIa) demonstrated a reduction in the infection rate. In conclusion, the use of glutamine has not been shown to be harmful in the neurocritical patient.

With regard to the use of zinc supplements and other trace elements, there are no conclusive studies which demonstrate an improvement in the variables of clinical outcome and degree of brain recovery in neurocritical patients³⁰.

What are the most common complications of nutritional support in neurocritical patients?

Neurocritical patients show a high incidence of gastrointestinal complications, the most common being IGR, conditioned by the brain injury itself^{31,32} and by the drugs necessary for an adequate control of intracranial pressure (analgesics, sedatives and muscle relaxants).

Transpyloric nutrition is an effective alternative in patients with high IGR³³. Two studies^{34,35} (Ib) evidenced that transpyloric feeding significantly improved the effective volume versus gastric nutrition, and 2 recent publications^{36,37} (Ib) have confirmed that transpyloric versus gastric feeding reduces significantly the incidence of late pneumonia. Administration of mixed,

enteral and parenteral nutritional support, could also be a valid option in case of gastrointestinal complications, with close monitoring requirements to avoid hyperfeeding. However, there are no studies on the use of mixed nutrition in neurocritical patients.

How should glycemia be controlled?

In these patients, hyperglycemia has been related to an increased rate of infectious and non-infectious upper complications, compared to other groups of critically-ill patients. After the brain injury, a number of changes occur in the metabolism, transport and response to insulin, which are dependent on the type of lesion^{38,39}.

The increased blood glucose values increase the infection rate and neurological damage. On the contrary, a dramatic reduction in plasma glucose values causes an increased lactate-pyruvate ratio and brain glutamate, that increases brain damage. The gradient between plasma and brain glucose levels is 0.6-0.7, which leads to recommending larger adjustments in the control of glycemia in neurocritical patients^{40,41}. There is no consensus on the benefit of intensive or conventional therapy with insulin in neurocritical patients. In a large study⁴², no differences in mortality and neurological sequelae were observed between the two groups, though the rate of moderate hypoglycemia rate was higher in the intensive insulin group. Studies evaluating the effect of insulin upon the metabolism and progress variables recommend blood glucose values between 120 and 150 mg/dL, as safety values, in neurocritical patients⁷ (Ib). Lower values may induce decreased extracellular glucose reserve and the subsequent brain energy dysfunction. In contrast, increased glycemia values lead to a worsening of prognostic variables, such as neurological recovery, infection rate, mortality, and hospital stay^{6,7}.

Recommendations

– Due to the severe catabolism state and the unfeasibility of an adequate nutritional supply, neurocritical patients should receive specialized nutritional support in the first three days of their evolution (B).

– High-protein supply is recommended (C).

– Enteral nutrition by transpyloric route is recommended in patients with brain injury since, as compared to the gastric route, it improves the efficacy in enteral supply and reduces the incidence of late pneumonia (B).

– Blood glucose control is recommended as in all other critically-ill patients (A).

– Administration of glutamine dipeptides, intravenously, may be safely used in the neurocritical patient (B).

Conflict of interests

The authors declare that they have participated in activities funded by the pharmaceutical industry for marketing of nutritional products (clinical studies, educational programmes and attendance to scientific events). No pharmaceutical industry has participated in the preparation, discussion, writing, and establishing of evidences in any phase of this article.

References

1. Bullock MR, Chesnut R, Ghajar J, Gordon D, Hartl R, Newell DW et al. Guidelines for the surgical management of traumatic brain injury. *Neurosurgery* 2006; 58: S2-1-62.
2. Latorre JG, Chou SH, Nogueira RG, Singhal AB, Carter BS, Ogilvy CS et al. Effective glycemic control with aggressive hyperglycemia management is associated with improved outcome in aneurismal subarachnoid hemorrhage. *Stroke* 2009; 40: 1644-52.
3. Frontera JA, Fernández A, Claassen J, Schmidt M, Schumacher HC, Wartenberg K et al. Hyperglycemia after SAH: predictors, associated complications, and impact on outcome. *Stroke* 2006; 37: 199-203.
4. Nguyen NQ, Fraser RJ, Bryant LK, Chapman M, Holloway RH. Diminished functional association between proximal and distal gastric motility in critically ill patients. *Intensive Care Med* 2008; 34: 1246-55.
5. Foley N, Marshall S, Pikul J, Salter K, Teasell R. Hypermetabolism following moderate to severe traumatic acute brain injury: a systematic review. *J Neurotrauma* 2008; 25: 1415-31.
6. Haider W, Lackner F, Schlick W, Benzer H, Gerstenbrand F, Irsigler K, et al. Metabolic changes in the course of severe acute brain damage. *Eur J Intensive Care Med* 1975; 1: 19-26.
7. Perel P, Yanagawa T, Bunn F, Roberts I, Wentz R, Pierro A. Nutritional support for head-injured patients. *Cochrane Database Syst Rev* 2006; 4: CD001530.
8. Taylor SJ, Fettes SB, Jewkes C, Nelson RJ. Prospective, randomized, controlled trial to determine the effect of early enhanced enteral nutrition on clinical outcome in mechanically ventilated patients suffering head injury. *Crit Care Med* 1999; 27: 2525-31.
9. Nguyen NQ, Fraser RJ, Bryant LK, Burgstad C, Chapman MJ, Bellon M et al. The impact of delaying enteral feeding on gastric emptying, plasma cholecystokinin, and peptide YY concentrations in critically ill patients. *Crit Care Med* 2008; 36: 1469-74.
10. Kompan L, Vidmar G, Spindler-Vesel A, Pecar J. Is early enteral nutrition a risk factor for gastric intolerance and pneumonia? *Clin Nutr* 2004; 23: 527-32.
11. Kompan L, Kremzar B, Gadzijev E, Prosek M. Effects of early enteral nutrition on intestinal permeability and the development of multiple organ failure after multiple injury. *Intensive Care Med* 1999; 25: 157-61.
12. Doig GS, Heighes PT, Simpson F, Sweetman EA, Davies AR. Early enteral nutrition, provided within 24 h of injury or intensive care unit admission, significantly reduces mortality in critically ill patients: a meta analysis of randomised controlled trials. *Intensive Care Med* 2009; 35: 2018-27.
13. Härtl R, Gerber LM, Ni Q, Ghajar J. Effect of early nutrition on deaths due to severe traumatic brain injury. *J Neurosurg* 2008; 109: 50-6.
14. Minard G, Kudsk KA, Melton S, Patton JH, Tolley EA. Early versus delayed feeding with an immune-enhancing diet in patients with severe head injuries. *JPEN J Parenter Enteral Nutr* 2000; 24: 145-9.
15. Ng I, Lim J, Wong HB. Effect of head posture on cerebral hemodynamics: its influence on intracranial pressure, cerebral perfusion pressure and cerebral oxygenation. *Neurosurgery* 2004; 54: 593-7.

16. Meixensberger J, Baunach S, Amschler J, Dings J, Roosen K. Influence of body position on tissue-pO₂ cerebral perfusion pressure and intracranial pressure in patients with acute brain injury. *Neurol Res* 1997; 19: 249-53.
17. Drakulovic MB, Torres A, Bauer TT, Nicolás JM, Nogué S, Ferrer M. Supine body position as a risk factor for nosocomial pneumonia in mechanically ventilated patients: a randomised trial. *Lancet* 1999; 354: 1851-8.
18. Bochicchio GV, Bochicchio K, Nehman S, Casey C, Andrews P, Scalea TM. Tolerance and efficacy of enteral nutrition in traumatic brain-injured patients induced into barbiturate coma. *JPEN J Parenter Enteral Nutr* 2006; 30: 503-6.
19. Faisy C, Guerot E, Dile JL, Labrousse J, Fagon JY. Assessment of resting energy expenditure in mechanically ventilated patients. *Am J Clin Nutr* 2003; 78: 241-9.
20. Weekes E, Elia M. Observations on the patterns of 24-hour energy expenditure changes in body composition and gastric emptying in head-injured patients receiving nasogastric tube feeding. *JPEN J Parenter Enteral Nutr* 1996; 20: 31-7.
21. McCall M, Jeejeebhoy K, Pencharz P, Moulton R. Effect of neuromuscular blockade on energy expenditure in patients with severe head injury. *JPEN J Parenter Enteral Nutr* 2003; 27: 27-35.
22. Klein S, Kinney J, Jeejeebhoy K, Alpers D, Hellerstein M, Murray M et al. Nutrition support in clinical practice: review of published data and recommendations for future research directions. National Institutes of Health, American Society for parenteral and Enteral Nutrition, and American Society for Clinical Nutrition. *JPEN J Parenter Enteral Nutr* 1997; 21: 133-56.
23. Bonet A, Grau T. Glutamine, an almost essential amino acid in the critically ill patient. *Med Intensiva* 2007; 31: 402-6.
24. Houdijk AP, Rijnsburger ER, Jansen J, Wesdorp RI, Weiss JK, Mc-Camish MA et al. Randomised trial of glutamine-enriched enteral nutrition on infectious morbidity in patients with multiple trauma. *The Lancet* 1998; 352: 772-6.
25. Novak F, Heyland DK, Avenell A, Drover JW, Su X. Glutamine supplementation in serious illness: a systematic review of the evidence. *Crit Care Med* 2002; 30: 2022-9.
26. García-de-Lorenzo A, Zarazaga A, García Luna PP, González Huix F, López Martínez J, Miján A. Clinical evidence for enteral nutritional support with glutamine: a systematic review. *Nutrition* 2003; 19: 805-11.
27. Berg A, Bellander BM, Wanecek M, Gamrin L, Elving A, Rooyackers O et al. Intravenous glutamine supplementation to head trauma patients leaves cerebral glutamate concentration unaffected. *Intensive Care Med* 2006; 32: 1741-6.
28. Berg A, Bellander BM, Wanecek M, Norberg A, Ungerstedt U, Rooyackers O et al. The pattern of amino acid exchange across the brain is unaffected by intravenous glutamine supplementation in head trauma patients. *Clin Nutr* 2008; 27: 816-21.
29. Falcão de Arruda IS, De Aguiar-Nascimento JE. Benefits of early enteral nutrition with glutamine and probiotics in brain injury patients. *Clin Sci (Lond)* 2004; 106: 287-92.
30. Young B, Ott L, Kasarskis E, Rapp R, Moles K, Dempsey RJ, et al. Zinc supplementation is associated with improved neurologic recovery rate and visceral protein levels of patients with severe closed injury. *J Neurotrauma* 1996; 13: 25-34.
31. Kao CH, ChangLai SP, Chieng PU, Yen TC. Gastric emptying in head-injured patients. *Am J Gastroenterol* 1998; 93: 1108-12.
32. Acosta Escribano JA, Carrasco Moreno R, Fernández Vivas M, Navarro Polo JN, Más Serrano P, Sánchez Payá J et al. Gastric enteral intolerance in mechanically ventilated patients with traumatic cerebral lesion. *Nutr Hosp* 2001; 16: 262-7.
33. Graham TW, Zadrozny DB, Harrington T. The benefits of early jejuna hyperalimentation in the head-injured patient. *Neurosurgery* 1989; 25: 729-35.
34. Montejo JC, Grau T, Acosta J, Ruiz-Santana S, Planas M, García-De-Lorenzo A et al. Multicenter, prospective, randomized, single-blind study comparing the efficacy and gastrointestinal complications of early jejunal feeding with early gastric feeding in critically ill patient. *Crit Care Med* 2002; 30: 796-800.
35. Neumann DA, Delege MH. Gastric versus small-bowel tube feeding in the intensive care unit: a prospective comparison of efficacy. *Crit Care Med* 2002; 30: 1436-8.
36. Hsu CW, Sun SF, Lin SL, Kang SP, Chu KA, Lin CH, et al. Duodenal versus gastric feeding in medical intensive care unit patients: a prospective, randomized, clinical study. *Crit Care Med* 2009; 37: 1866-72.
37. Acosta-Escribano J, Fernández-Vivas M, Grau Carmona T, Caturla-Such J, García-Martínez M, Menéndez-Mainer A, et al. Gastric versus transpyloric feeding in severe traumatic brain injury: a prospective, randomized trial. *Intensive Care Med* 2010; 36: 1532-9.
38. Bilotta F, Spinelli A, Giovannini F, Doronzio A, Delfini R, Rosa G. The effect of intensive insulin therapy on infection rate, vasospasm, neurologic outcome, and mortality in neurointensive care unit after intracranial aneurysm clipping in patients with acute subarachnoid hemorrhage: a randomized prospective pilot trial. *J Neurosurg Anesthesiol* 2007; 19: 156-60.
39. Vespa P, Boonyaputthikul R, McArthur DL, Miller C, Etchepare M, Bergsneider M et al. Intensive insulin therapy reduces microdialysis glucose values without altering glucose utilization or improving the lactate/pyruvate ratio after traumatic brain injury. *Crit Care Med* 2006; 34: 850-6.
40. Rovlias A, Kotsou S. The influence of hyperglycemia on neurological outcome in patients with severe head injury. *Neurosurgery* 2000; 46: 335-42.
41. Jeremitsky E, Omert LA, Dunham CM, Wiberger J, Rodríguez A. The impact of hyperglycemia on patients with severe brain injury. *J Trauma* 2005; 58: 47-50.
42. Green DM, O'Phelan KH, Bassin SL, Chang CW, Stern TS, Asai SM. Intensive versus conventional insulin therapy in critically ill neurologic patients. *Neurocrit Care* 2010; 13: 299-306.