# DOWNLOAD PDF PARENTERAL AND ENTERAL NUTRITION IN ADULT PATIENTS REX O. BROWN

## Chapter 1 : Intolerance to enteral feeding in the brain-injured patient : Journal of Neurosurgery

*This American Society for Parenteral and Enteral Nutrition (A.S.P.E.N.) Clinical Guideline summarizes the most current evidence and provides guidelines for the desired blood glucose goal range in hospitalized patients receiving nutrition support, the definition of hypoglycemia, and the rationale for use of diabetesâ€•specific enteral formulas.*

However, validated methods for the treatment of acute hypocalcemia are lacking. The efficacy of a single dose of calcium gluconate using an empiric IV calcium gluconate graduated dosing regimen was evaluated in 37 patients. Patients with an iCa of  Patients with an iCa of Aberration in calcium metabolism is an important but often undetected metabolic problem in the critically Ul patient. Most laboratories report total calcium concentrations that are inaccurate in the critically ill patient, even when the serum albumin concentration is taken into consideration. The intent of this investigation was to evaluate the efficacy of a graduated intravenous IV calcium gluconate regimen for critically ill, multiple-trauma patients. Only trauma intensive care unit patients were potentially enrolled for study. Serum chemistries, arterial blood gas measurements, nutrition markers, and ionized calcium concentration were simultaneously obtained from each patient. The blood was obtained at approximately 3 AM via an indwelling arterial or venous catheter while the patient lay supine in bed. Nutrition assessment measurements were also conducted. Each patient contributed only once to the data pool. The total serum calcium was measured colorimetrically, whereas the serum ionized calcium concentration iCa was determined usiner an ion-selective electrode method. Those who received blood products, calcium or vitamin D therapy, furosemide, or therapeutic doses of heparin within 24 hours before the laboratory measurements were excluded from the analysis. Patients with a history of cancer, bone disease, or parathyroid disease were also excluded. Patients were given enterai nutrition by a small-bore, nasoenteric feeding tube or jejunostomy. Patients who could not receive enterai nutrition received parenteral nutrition. Parenteral nutrition was given via the subclavian or external jugular vein when enterai feeding was contraindicated. The enteral formulas contained 40 mEq of calcium per L and the parenteral nutrition solution contained 5 mEq of calcium gluconate per L. Hypocalcemic patients receiving enterai nutrition received IV calcium gluconate supplementation without additional calcium added to their feeding. Patients who required phosphorus supplementation were given an IV dosage scheme that has been previously shown to not significantly influence serum ionized or total calcium concentrations. We attempted to give the IV calcium and phosphorus infusions sequentially. To ascertain whether any relationship existed between calcium intake upon serum phosphorus concentration changes, patients from each hypocalcemia severity group were further subdivided into those who received phosphorus therapy on the same day as calcium therapy and compared with those who were given calcium therapy without phosphorus supplementation. The study was approved and conducted in accordance with the guidelines established by the University of Tennessee Health Science Center institutional review board. Because all measurements were performed as part of the routine metabolic evaluation of the patient, confidentiality procedures for the patient were maintained, and the data were collected retrospectively, the requirement for informed consent was waived. Only patients treated for hypocalcemia with PV calcium gluconate according to our dosing guidelines cited in the methods were studied. Six patients not given calcium therapy according these guidelines were excluded from the analysis. The majority of the critically ill population was admitted due to a motor vehicle accident or gunshot wound and were ventilator dependent Table I. There was no significant difference between mild and moderate to severe hypocalcemia groups with respect to gender, admission diagnoses, body temperature, white blood cell count, trauma score, blood transfusion, or with presence of head injury, sepsis, vasopressor therapy, or ventilator dependency Table I. One patient received both enteraL and parenteral nutrition transitional feeding during the study. The remaining patients were given parenteral nutrition. Most patients were adequate in body weight for height but had marked serum protein depletion and negative nitrogen balance Table II. The percentage of goal nutrition intake was reflective of a nitrogen balance determination within days of initiating

specialized nutrition support and of the slow initiation characteristics of providing enteraL nutrition. Nitrogen balance also tended to be worse for the moderate to severe hypocalcemiC group, but this difference between groups did not achieve statistical significance Table II due to the variability of the nitrogen balance determinations. Patients had a statistically significant rise in serum iCa and total calcium concentration after the calcium infusion Table III. There was wide variability in response, as evidenced by the pH-corrected change in serum iCa, at each dosage, respectively Figure 1. One patient exhibited mild hypercalcemia, with an iCa of 1. The postdose iCa was done approximately 19 hours after completion of the infusion. In addition, the patient had worsening acidemia, with a pH of 7. About two-thirds of the patients also required IV phosphorus supplementation Table V. The calcium and phosphorus infusions were given separately to avoid calcium-phosphate precipitation. Patients who received combined calcium and phosphorus infusions had significant increases in serum calcium and iCa without a significant change in serum phosphorus concentration Table V. However, patients who received IV calcium supplementation without phosphorus therapy demonstrated a decline in serum phosphorus from 4. The presence of hypocalcemia also may be related to increased mortality and severity of disease. In addition, we used the calcium gluconate salt form for the treatment of hypocalcemia as opposed to calcium chloride used in the animal studies , which contains one-third as much elemental calcium as the chloride salt form. IV calcium administration may also increase digoxin toxicity; however, none of the patients in our study received digoxin therapy. Effective therapy for acute hypocalcemia during critical illness is an enigma due to the lack of any established and proven dosing regimens. For symptomatic patients or those with an ionized serum calcium concentration of Despite conservative dosing, 1 patient experienced mild hypercalcemia at 1. The patient was given 4 g of calcium gluconate for a serum iCa of 0. During alkalemia, hydrogen ion is released from the protein and the calcium ion will bind, reducing its free or ionized concentration. No clinically relevant adverse effects were noted in the medical chart. We have previously shown that our IV phosphorus dosage scheme does not significantly alter serum total or iCa. Serum phosphorus declined in both the mild and moderate to severe hypocalcemia groups when patients were given calcium without phosphorus supplementation Table V. When phosphorus therapy was given sequentially with calcium therapy, the decline in serum phosphorus was attenuated. It is unclear whether the calcium therapy itself caused a fall in serum phosphorus or whether the patients may have been having refeeding issues11,12 or a whether it was due to a combination of both physiologic effects. Further study is warranted to provide more meaningful interpretation of these data. One patient experienced mild hypercalcemia 1. A decline in serum phosphorus concentration was noted for those who received calcium without phosphorus therapy. It is unclear whether the observed fall in serum phosphorus concentration was more reflective of refeeding issues, a causative effect from the calcium infusion, or both. Further study with frequent serial ionized serum calcium and phosphorus determinations and electrocardiographic monitoring appears indicated for patients with moderate to severe hypocalcemia. Accuracy of methods to estimate ionized and "corrected" serum calcium concentrations in critically ill multiple trauma patients receiving specialized nutrition support. 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## Chapter 2 : Vitamin D deficiency in critically ill patients with traumatic injuries

*Manpreet S. Mundi, Erin M. Nystrom, Daniel L. Hurley and M. Molly McMahon, Management of Parenteral Nutrition in Hospitalized Adult Patients, Journal of Parenteral and Enteral Nutrition, 41, 4, (), ().*

Standard enteral formulas meet basic macro- and micronutrient needs; therapeutic enteral formulas meet these basic needs and also contain specific pharmaconutrients that may attenuate hyperinflammatory responses, enhance the immune responses to infection, or improve gastrointestinal tolerance. In this paper, we review principles of how to feed enteral, parenteral, or both and when to feed early versus delayed start patients who are critically ill. We summarize current expert guidelines for nutrition in patients with critical illness, and we present specific clinical evidence on the use of enteral formulas supplemented with anti-inflammatory or immune-modulating nutrients, and gastrointestinal tolerance-promoting nutritional formulas. Finally, we introduce an algorithm to help bedside clinicians make data-driven feeding decisions for patients with critical illness. From supportive nutrition to therapeutic nutrition In patients who are critically ill, there is no doubt that nutritional status and clinical outcome are linked [ 1 ]. ICU patients typically experience catabolic stress and systemic inflammatory response; in turn, these responses alter both the morphology and function of the gastrointestinal GI tract [ 2 ]. Such GI dysfunction, often coupled with inadequate caloric intake, leads many critically ill patients to develop an energy deficit and lose lean body mass [ 5 - 10 ]. ICU patients with poor nutritional status commonly experience immune dysfunction, weakened respiratory muscles and lowered ventilation capacity, and reduced GI tolerance [ 11 , 12 ]. As a result, patients are at risk for a wide range of complications: For such patients, supportive nutrition has long been used as adjunctive care; however, such nutrition is often inadequate, including only fundamental macronutrients to sustain patients through periods of metabolic stress. Feeding an ICU patient now extends beyond choosing the right feeding route, the rate, and the caloric density. Enteral feeds are now formulated with active nutrients that may help reduce oxidative damage to cells and tissues, modulate inflammation, enhance beneficial stress responses, and improve feeding tolerance. In this review, we summarize the most recent data on feeding critically ill patients. We describe practice guide-lines established by US, European and Canadian enteral and parenteral nutrition societies, beginning with recommendations of how to feed enteral, parenteral, or both and when to feed early versus delayed start. We systematically discuss what to feed critically ill patients by reviewing mechanisms of action for specific pharmaconutrients and by concisely summarizing current guide-lines and expert recommendations for feeding various populations in the ICU. We compile clinical evidence on feeding anti-inflammatory, immune-modulating, and GI tolerance-promoting nutritional formulas in specific patient subgroups. Finally, we introduce a straightforward algorithm to help bedside clinicians make feeding decisions for patients with critical illness. Practice guidelines in Europe, Canada, and the US endorse enteral feeding for patients who are critically ill and hemodynamically stable [ 1 , 11 , 15 ]. Enteral nutrition is preferred over parenteral nutrition PN for most ICU patients - an evidence-based practice supported by numerous clinical trials involving a variety of critically ill patient populations, including those with trauma, burns, head injury, major surgery, and acute pancreatitis [ 1 , 16 ]. For ICU patients who are hemodynamically stable and have a functioning GI tract, early enteral feeding within 24 to 48 hours of arrival in the ICU has become a recommended standard of care [ 1 , 11 , 15 ]. Experts identify these early hours as a window of opportunity to provide nutrition that maintains gut barrier function and supports immune responses [ 1 , 11 ]. However, early findings suggest use of early enteral feeding in other vasopressor-dependent patients. In one study, vasopressor-dependent patients who were given enteral feeding within the first 48 hours had a significant survival advantage compared to those whose feeding was delayed; in fact, the sickest patients on multiple vasopressors experienced the greatest benefit [ 17 ]. It should be noted that this finding is based on an observational study. A confirmatory prospective, controlled study is warranted. Even though early enteral nutrition is favored for most ICU patients, caloric and protein needs are often not met by enteral feeding [ 10 ]. Nutritional intake may be

hampered by setting target levels too low, interruption of feeding for procedures, issues of airway management, and poor tolerance of feedings [ 8 , 18 , 19 ]. To enhance use of enteral feeding in ICU patients, several feeding strategies have been proposed recently: PN is necessary in critically ill patients who do not have an intact GI tract, but current guidelines do not agree on when to initiate PN [ 5 ]. For patients who are intolerant or have other contraindications to enteral feeding, European guidelines recommend starting PN within 24 to 48 hours if the patient is not expected to be on oral nutrition within 3 days [ 24 ]. US guidelines hesitate to recommend PN on admission to the ICU; standard care intravenous fluids is recommended first, with PN reserved and initiated only after 7 days in well-nourished patients [ 1 ]. Canadian guide-lines state that PN should not be used in any patient with an intact GI tract [ 25 ]. When enteral feeding alone is inadequate, some experts are calling for use of PN and enteral nutrition together to meet energy and protein targets [ 5 , 7 , 26 , 27 ]. Combination regimens are justified by observations that actual enteral intake typically meets only half of prescribed calories in ICU patients [ 6 , 8 - 10 ]. However, clinical evidence for combination feeding remains unclear. Two recent randomized trials have helped clarify this subject. Those in the late-initiation group also had significantly fewer ICU infections, shorter duration of mechanical ventilation and a shorter course of renal replacement therapy. Several aspects of the study limit generalizability of the findings to all ICU populations. First, patients with chronic malnutrition were not included in the study. Third, patients in the trial received a low protein delivery median of 0. Finally, the trial examined a low mortality-risk patient group with an average ICU mortality of 6. Accounting for the aforementioned limitations, The EPaNIC trial is unquestionably a key contribution to the literature on supplemental PN use in critical care. We believe the key conclusion is that early aggressive calorie delivery via PN does not appear to be beneficial in low mortality risk, non-chronically malnourished patients. Additional trials on the use of enteral nutrition with supplemental PN have recently been completed or are underway. Choosing which enteral formulation to feed For most ICU patients, the next decision is what enteral formula to feed. Critically ill patients are a heterogeneous population, so no one-size-fits-all nutritional formula should be expected [ 30 , 31 ]. Feeding formulas to consider are anti-inflammatory, immune-modulating, GI tolerance-promoting, and standard enteral nutrition. What are the key functional pharmaconutrients in enteral formulas? Guidelines from professional nutrition societies around the world identify certain populations of patients who can benefit from formulations with specific pharmaconutrients [ 1 , 11 , 15 , 25 ]. To help guide such choices, the following section reviews functional pharmaconutrients and their roles in critically ill patients. Anti-inflammatory enteral nutrients Critical illness and injury are characterized by oxidative stress and excessive inflammation, harmful processes that damage cells and impair function of vital organs. Extreme inflammation - as in patients with systemic inflammatory response syndrome SIRS , sepsis, acute lung injury ALI , or acute respiratory distress syndrome ARDS - often progresses to multiple organ dysfunction syndrome and even death. Feeding formulas with specific pharmaconutrients can help o set tissue damage and moderate inflammation. Dietary antioxidants vitamins A, C, and E and selenium play important roles in reducing potential for tissue damage by stabilizing free radicals in cells, while dietary fish oil and borage oil blunt out-of-control inflammatory responses by modulating synthesis of pro- and anti-inflammatory mediators [ 31 - 33 ]. Fish oil and borage oil Dietary intake of certain oils alters the fatty acid composition in membranes of cells involved in immune inflammatory responses, that is, neutrophils and macrophages. Certain cell membrane fatty acids for example, arachidonic acid AA serve as precursors to inflammatory eicosinoids and leukotriene mediators, while other fatty acids eicosapentaenoic acid EPA , docosohexaenoic acid DHA , gamma linolenic acid GLA are metabolized to form less pro-inflammatory mediators [ 31 - 34 ]. In addition, DHA and EPA are precursors of resolvins and protectins, which help resolve inflammation and reduce tissue injury [ 35 ]. While healthy humans have desaturase enzymes that convert alpha-linolenic acid to EPA and DHA, such conversion is negligible in individuals who are critically ill.