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A NARRATIVE REVIEW

Nutritional Support in Severe Acute Pancreatitis

Lindsay Bartholome, MD, FACS[1], Rifat Latifi, MD, FACS, FICS, FKCS[2]

[1] Trauma Medical Director, Banner Desert Medical Center, Mesa, Arizona;
[2] Professor of Surgery, Department of Surgery,
University of Arizona, Tucson, AZ, USA; Trauma and Acute Care Surgery Division,
Arizona Advanced Surgery, Mesa, Arizona

Corresponding Author:

RIFAT LATIFI, MD, FACS, FICS, FKCS

Professor of Surgery, University of Arizona; Arizona Advanced Surgery and
Banner Desert Medical Center, Mesa, Arizona.
E-mail: rlatifi@arizona.edu

Abstract

Background: Severe acute pancreatitis (SAP) develops in up to 20% of patients with acute pancreatitis and carries a mortality of 15–35%, however, mortality may exceed 40% once infected pancreatic necrosis supervenes. The attendant systemic inflammatory response drives a hypercatabolic, hypermetabolic state (resting energy expenditure rising 20–50% above baseline) and precipitates gut-barrier failure, positioning nutritional therapy as a disease-modifying intervention rather than adjunctive care.

Aims: The primary aim of this review is to synthesize the current evidence and guideline recommendations governing nutritional support in SAP. The secondary aim is to translate that evidence into practical, bedside-applicable guidance for the acute care surgeon and surgical intensivist—covering the timing of initiation, the route and adequacy of enteral access, the recognition and prevention of feeding-related complications, and the safe delivery of parenteral nutrition when enteral feeding fails—and to delineate the areas in which the evidence remains insufficient to direct practice.

Methods: A narrative review of PubMed/MEDLINE and the Cochrane Library (2000–2025) was performed, integrating randomized controlled trials, meta-analyses, and the guidelines of ESPEN, ASPEN/SCCM, the AGA, the ACG, EAST, and the AAST.

Results: Meta-analytic data demonstrate that enteral nutrition (EN), versus total parenteral nutrition (TPN), reduces mortality (RR 0.50, 95% CI 0.28–0.91), systemic infection (RR 0.39, 95% CI 0.23–0.65), and multi-organ failure (RR 0.55, 95% CI 0.37–0.81). EN initiated within 24–72 h is uniformly recommended; nasogastric and nasojejunal routes are equivalent. TPN retains a defined role when EN is contraindicated, requiring dextrose infusion below 4–5 mg/kg/min and glycemic targets of 140–180 mg/dL. Probiotics are contraindicated (mortality 16% vs 6%).

Conclusion: Early EN is the evidence-based cornerstone of SAP nutritional management, endorsed across all major guidelines. Route, timing, and access independently determine outcome; TPN is reserved for EN failure under strict metabolic control.

Keywords: Severe acute pancreatitis; enteral nutrition; total parenteral nutrition; ASPEN; SCCM; AAST; ESPEN; feeding tube complications; hyperglycemia; nutritional support; critical care surgery

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Introduction

Acute pancreatitis is among the most frequently encountered gastrointestinal emergencies in surgical and critical care practice, with an annual global incidence of approximately 34 per 100,000 person-years and a steadily rising trend driven by increasing rates of gallstone disease, obesity, alcohol use, and hypertriglyceridemia.[1] The disease imposes a substantial and growing burden on health systems, pancreatitis being among the most common principal gastrointestinal discharge diagnoses in the United States, where it accounts for approximately 300,000 hospital admissions each year and contributes to aggregate gastrointestinal hospitalization costs measured in the tens of billions of dollars.[2] While roughly 80% of cases follow a mild, self-limiting course, up to 20% progress to severe acute pancreatitis (SAP), defined by the revised Atlanta Classification (2012) as pancreatitis complicated by persistent single- or multi-organ failure exceeding 48 hours.[3] SAP carries a mortality of 15–35%, rising sharply beyond 40% once infected pancreatic necrosis develops, and its clinical course is characteristically prolonged, biphasic, and unpredictable.

From the vantage point of a critical care surgeon whose career has centered on nutritional support of the critically ill and injured, SAP is one of the most instructive and demanding nutritional scenarios in all of surgery. The systemic inflammatory response unleashed by pancreatic autodigestion drives a hypercatabolic, hypermetabolic state physiologically indistinguishable from that following major trauma or major emergency surgery, with resting energy expenditure rising 20–50% above baseline, accelerated proteolysis, negative nitrogen balance, and the potential loss of 20–30% of body protein mass within two to three weeks.[4,5] Nutritional support of the critically ill has undergone transformative advances in recent decades, evolving from empirical supplementation into a precision, disease- and disorder-based science with measurable impact on morbidity and mortality.[6] In no disease is this evolution more clearly demonstrated than in SAP, where the metabolic insult is severe, nutritional depletion is rapid, and the consequences of malnutrition—particularly infection—are lethal.

Historically, the principle of "pancreatic rest" drove clinicians to keep SAP patients nil per os (NPO) and to deliver nutrition exclusively through TPN, on the misguided theory that withholding enteral stimulation would quiet pancreatic exocrine secretion and limit autodigestion. Even within that paradigm, as described in a foundational 1991 analysis,[7] meticulous attention to both the route and the composition of nutritional support was recognized as essential. What the pancreatic-rest doctrine neglected, however, was the central role of the gut as an immunological and mechanical barrier organ. Loss of enteral stimulation triggers villous atrophy, disrupts epithelial tight junctions, depletes secretory IgA, and enables translocation of luminal bacteria and endotoxin into the portal circulation—seeding inflamed peripancreatic tissue and converting sterile necrosis into infected necrosis, the principal driver of late mortality.[8] This mechanistic understanding reframed the gut not as an organ to be rested but as one to be protected through early luminal nutrient delivery.

Over the past two decades, high-quality evidence from randomized controlled trials, meta-analyses, and the Cochrane Library—now codified in concordant guidelines from ESPEN,[9,10] ASPEN/SCCM,[11] the AGA,[12] the ACG,[13] EAST,[14] and the AAST[15]—has overturned the pancreatic-rest paradigm completely, establishing early enteral nutrition as the standard of care. In parallel, evolving surgical nutrition research has clarified that the timing of nutritional initiation, the route of delivery, and the adequacy of nutritional access are independent determinants of outcome in surgical patients with acute abdominal pathology, including SAP.[16,17] As a 2025 editorial in European Surgery introducing a special focus on nutritional support across surgical disciplines articulated,[18] effective nutritional intervention enhances immune function, promotes healing, and facilitates recovery, whereas inadequate support heightens the risk of malnutrition, infection, and death. These principles apply with full force to SAP.

Pathophysiology of Nutritional Derangements

SAP triggers sustained hypermetabolism driven by SIRS, with counterregulatory hormone excess (cortisol, glucagon, catecholamines) promoting glycogenolysis, gluconeogenesis, proteolysis, and lipolysis while impairing peripheral glucose uptake.[4] The result is hyperglycemia, negative nitrogen balance, hypoalbuminemia, and rapid depletion of lean body mass.[19] Concurrently, splanchnic hypoperfusion disrupts epithelial tight junctions and depletes mucosal secretory IgA, producing a "leaky gut" through which bacteria and endotoxin translocate into the portal circulation—the principal mechanism seeding sterile necrosis and converting it to infected necrosis, the event most responsible for late

mortality.[8] Early enteral nutrition directly counters this vulnerability by maintaining villous height, stimulating mucosal turnover, promoting bile flow, and preserving commensal flora. This strategy has been associated with reduced mortality, fewer infectious complications, and decreased rates of organ failure, largely through preservation of intestinal mucosal integrity and gut-barrier function.[21,23,24]

The primary aim of this narrative review is to synthesize current evidence and guideline recommendations on optimal nutritional support in SAP. The secondary aim is to translate that evidence into practical, bedside-applicable guidance for the acute care surgeon and the surgical intensivist. To those ends, the review examines the pathophysiologic rationale for early enteral feeding, the comparative evidence for enteral versus parenteral routes, the choice of gastric versus post-pyloric delivery, the complications of both enteral tube feeding and TPN, and the special circumstances of the open abdomen and refractory disease—with particular attention to the metabolic hazards of high-dextrose formulations in metabolically vulnerable patients and the disciplined management that allows parenteral nutrition to be used safely when it is required. It further seeks to make explicit those questions the current evidence base does not answer, so that the residual uncertainty is stated rather than obscured.

Methods

A narrative review of the literature was performed using PubMed/MEDLINE and the Cochrane Library. Individual references were verified against PubMed, publisher and DOI records, and, for Cochrane systematic reviews, the Cochrane Library directly. Search and verification focused on terms relating to severe acute pancreatitis, enteral and parenteral nutrition, route of feeding, feeding-tube and parenteral-nutrition complications, gut-barrier function, and critical care nutrition. Coverage emphasized human studies and English-language publications from 2000 through 2024, with priority given to literature published from 2016 onward. Reporting was guided by the Scale for the Assessment of Narrative Review Articles (SANRA), which addresses justification of the review's importance and aims, description of the literature search, referencing, and the presentation of evidence and data.[20]

Guideline documents were retrieved and reviewed in full, including the ESPEN Guideline on Clinical Nutrition in Acute and Chronic Pancreatitis (2020)[9] and the ESPEN Practical Guideline update (2024);[10] the ASPEN/SCCM Guidelines for the Provision of Nutrition Support Therapy in the Adult Critically Ill Patient (2022);[11] the AGA Institute Guideline on Initial Management of Acute Pancreatitis (2018);[12] the ACG Guidelines on Management of Acute Pancreatitis (2024);[13] the EAST Practice Management Guidelines for Nutritional Support of the Trauma Patient (2004);[14] and the AAST Critical Care and Geriatric Trauma Committees clinical consensus document on geriatric nutrition in the surgical patient (2025).[15] The EAST document (2004) is the only formal trauma-specific nutrition practice management guideline and has not been revised since publication; it therefore predates the revised Atlanta Classification, the major parenteral-nutrition timing trials, and every other guideline cited here. It is included for its acute-care-surgery perspective, and its recommendations remain concordant with the more recent documents, but its age should be borne in mind when it is read alongside them. Two Cochrane systematic reviews were retrieved from the Cochrane Library and reviewed in full as primary evidence sources: enteral versus parenteral nutrition for acute pancreatitis[21] and nasogastric versus nasojejunal tube feeding for severe acute pancreatitis.[22] Relevant peer-reviewed publications by the author and colleagues on nutritional support in critically ill and surgical patients were incorporated where directly applicable.[16,6,17]

Results

Across converging randomized controlled trials, meta-analyses, and every major society guideline, the evidence favors early enteral nutrition (EN) over total parenteral nutrition (TPN) in SAP. Pooled Cochrane and subsequent meta-analytic data show that EN, compared with TPN, significantly reduces mortality (RR 0.50, 95% CI 0.28–0.91), systemic infection (RR 0.39, 95% CI 0.23–0.65), multi-organ failure (RR 0.55, 95% CI 0.37–0.81), and the need for surgical intervention[21,23,24] (Table 1). The mechanistic basis is preservation of the intestinal barrier, gut-associated lymphoid tissue, and commensal flora, thereby limiting the bacterial translocation that converts sterile to infected necrosis.[8]

EN initiated within 24–72 hours of admission, once hemodynamic stability is achieved, is recommended by ESPEN, ASPEN/SCCM, the AGA, the ACG, and EAST[9,10,11,12,13,14] (Table 1). Nasogastric and nasojejunal delivery are equivalent in mortality, aspiration, tolerance, and pain,[22,25,26] and the technically simpler nasogastric

route is endorsed as first-line. Caloric targets of 25–35 kcal/kg/day and protein of 1.2–1.5 g/kg/day are consistent across guidelines, with higher protein (up to 2.5 g/kg/day) advised in the highest-acuity and open-abdomen patients.[19] Securing and maintaining enteral access is itself independently prognostic, with lower rates of gastrointestinal complications and mortality.[17]

TPN retains a defined role when EN is contraindicated—such as refractory shock, high-output fistula, and peritonitis—or simply not tolerated, but early TPN in patients who could receive EN confers no benefit and increases infectious complications, ICU stay, and ventilation.[27] When indicated, TPN is delivered safely through disciplined metabolic control—dextrose infusion below 4–5 mg/kg/min, glycemic targets of 140–180 mg/dL, and strict aseptic catheter care.[28,29,30] Enteral glutamine is no longer recommended, and probiotics are contraindicated, having increased mortality (16% vs 6%) in a landmark trial.[5,31] The principal studies and guidelines underpinning these findings are summarized in Table 1.

Discussion

We have presented in this review the evidence that early enteral nutrition is the therapeutic cornerstone of nutritional management in SAP.[9,10,11,12,13,15,14] There is no need for reflexive NPO orders and TPN-first management, in favor of an approach grounded in targeting the specific derangements of pancreatic critical illness: hypercatabolism, gut-barrier failure, insulin resistance, and susceptibility to secondary infection.

Enteral Nutrition: Evidence and Guideline Concordance

The evidence favoring EN over TPN is robust and consistent (Table 1). A Cochrane systematic review, reinforced by subsequent meta-analyses, demonstrated significant reductions in mortality, systemic infection, and multi-organ failure with EN,[21,23,24] a conclusion echoed in contemporary reviews of nutrition therapy specific to SAP and to current management standards.[32,33] The question of timing is paramount: early nutrition confers measurable benefit but requires hemodynamic stability as a prerequisite,[16] so EN should commence within 24–72 hours of admission once resuscitation is underway—not after waiting for normalization of amylase, lipase, or inflammatory markers, none of which predict feeding tolerance. All major guidelines are concordant on early EN as first-line therapy, with TPN reserved for EN failure or contraindication.[9,10,11,12,13] Securing enteral access is not a secondary concern but a primary therapeutic act; a propensity-matched analysis showed that malnourished elderly patients who received surgical nutritional access had significantly lower gastrointestinal complication rates.[17]

Route of Delivery: Nasogastric versus Nasojejunal

The traditional preference for nasojejunal feeding rested on the concern that intragastric nutrients stimulate pancreatic exocrine secretion, but clinical evidence has consistently failed to support this.[22,25,26] Cochrane and additional systematic reviews found no significant differences between nasogastric and nasojejunal feeding in mortality, aspiration, tolerance, exacerbation, or pain. Because nasogastric placement is a bedside procedure enabling feeding within hours—whereas post-pyloric placement requires fluoroscopy, endoscopy, or electromagnetic guidance and delays initiation—the nasogastric route is endorsed as first-line, with post-pyloric feeding reserved for high aspiration risk or gastric intolerance.[9,10,11,12]

Complications of Enteral Feeding Tubes

Initiating EN begins rather than ends the clinician's responsibility. Tube malposition (1–3% of insertions) may cause aspiration pneumonitis, pneumothorax, or pulmonary hemorrhage, mandating radiographic confirmation before feeding. Aspiration is the most consequential ongoing complication, elevated in SAP by opioid-induced gastroparesis and impaired airway reflexes; preventive measures include head-of-bed elevation to 30–45 degrees, gastric residual monitoring, prokinetics, and transition to post-pyloric access when residuals persist. Bacterial sinusitis (5–20% with transnasal tubes beyond 5–7 days), diarrhea, tube occlusion, and oropharyngeal discomfort require active surveillance, because delays in diagnosis deprive the patient of a proven therapeutic intervention.

The Open Abdomen and Abdominal Catastrophe

A subset of SAP patients—those with abdominal compartment syndrome, necrosis requiring operative debridement, or enteroatmospheric fistula—present the added challenge of the open abdomen. A comprehensive expert review identified that most failures of primary fascial closure and fistula formation are preventable through effective temporary abdominal closure coupled

with appropriate resuscitation and nutritional support.[34] Protein losses through open abdomens are profound (an estimated 1–2 g per 100 mL of peritoneal exudate), so protein targets should be revised upward to 1.5–2.0 g/kg/day, and combined enteral plus supplemental parenteral nutrition is often the most pragmatic approach.

Total Parenteral Nutrition: Role, Timing, and Safe Delivery

When EN is not feasible, TPN retains a major and legitimate role, and its safe delivery depends on disciplined metabolic management rather than avoidance. Absolute indications include bowel obstruction, high-output proximal fistula, mesenteric ischemia, abdominal compartment syndrome, refractory hemodynamic instability, and refractory EN intolerance. The landmark EPaNIC trial established that early TPN in patients who can receive EN provides no benefit and increases infectious complications, ICU duration, and ventilation;[27] TPN should therefore not be initiated within the first 7 days if EN is feasible. When TPN is indicated, it remains an essential and effective therapy: the convergence of stress-induced insulin resistance, potential islet-cell dysfunction in necrotizing disease, and glucose-dense formulations makes glycemic control the central determinant of whether TPN helps or harms. The prescribing principles are straightforward—keep the dextrose infusion rate below 4–5 mg/kg/min, balance non-protein calories with lipid, target glucose of 140–180 mg/dL with continuous insulin infusion, and apply strict aseptic catheter care.[28,29,30] Applied with this rigor, TPN can be delivered safely and prevents the complications historically attributed to it.

Glutamine, Immunonutrition, Probiotics, and Oral Refeeding

A critical review of enteral glutamine noted that two major multicenter RCTs (REDOXS and METAPLUS) not only failed to show benefit but suggested harm in multi-organ failure;[5] enteral glutamine is therefore no longer recommended, and intravenous glutamine may be considered only when TPN is required and EN is not feasible.[10] Immunonutrition formulas have not shown consistent mortality benefit, and probiotics are contraindicated based on the PROPATRIA trial, which found significantly increased mortality (16% vs 6%).[31] Early oral refeeding is supported in moderately severe disease once pain is controlled, the patient is alert, and ileus has resolved.

Limitations

Several limitations of the evidence base must be acknowledged. Most RCTs in this field are small, single-center, and methodologically heterogeneous, and many predate the revised Atlanta Classification and the current step-up approach to necrosectomy. The optimal caloric dose in the early acute phase remains incompletely defined, and personalized approaches guided by indirect calorimetry and metabolic phenotyping are promising but not yet integrated into routine guideline recommendations.

Conclusion

Severe acute pancreatitis demands early, evidence-based, and precisely executed nutritional intervention. Early enteral nutrition—initiated within 24–72 hours of admission via the nasogastric route—is the standard of care, endorsed by ESPEN, ASPEN/SCCM, the AGA, the ACG, EAST, and the AAST.[9,10,11,12,13,15,14] This approach reduces mortality, infectious complications, and organ failure through gut-barrier preservation. The timing of initiation, the route and adequacy of enteral access, and the management of feeding-related complications are independent determinants of clinical outcome.[16,6,17] TPN retains an important role when EN is not feasible and, delivered with meticulous attention to dextrose load, glycemic control, and catheter care, is safe and effective. In SAP, nutrition is not supportive care—it is, alongside resuscitation and source control, one of the pillars of management upon which patient survival depends.

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Table 1. Evidence Summary of Major References

Ref (Year)	Author / Source	Type of Study	N (patients / trials)	Main Outcome	Clinical Utility
3 (2013)	Banks et al.; Atlanta Working Group	International consensus classification	n/a	Revised Atlanta Classification defining SAP by persistent organ failure >48 h.	Diagnostic framework; defines the SAP population.
8 (2014)	Wu, Sankaran, Plank, Windsor, Petrov	Meta-analysis	AP cohorts	Quantified gut-barrier dysfunction as a driver of infected necrosis.	Mechanistic rationale for early EN.
9,10 (2020/24)	Arvanitakis et al. (ESPEN)	Clinical practice guideline	n/a	EN first-line within 24–72 h; NG acceptable; IV glutamine only if TPN required.	Definitive, most comprehensive guideline reference.
11 (2022)	Compher et al. (ASPEN/SCCM)	GRADE-based guideline	n/a	EN within 24–48 h; avoid TPN in first 7 days if EN feasible.	Most rigorous critical-care nutrition guideline.
12 (2018)	Crockett et al. (AGA)	Clinical practice guideline	n/a	EN over TPN; nasogastric route preferred.	US gastroenterology guideline.
13 (2024)	Tenner et al. (ACG)	Clinical practice guideline	n/a	EN within 48 h when oral intake not tolerated.	Current US ACG guideline.
14 (2004)	Jacobs et al. (EAST)	Practice management guideline	n/a	Early EN within 72 h preferred in the severely injured.	Trauma/acute-care surgery perspective.
15 (2025)	Farrell et al. (AAST)	Clinical consensus document	n/a	Best-practice guidance on nutrition in the surgical/geriatric patient.	Recent multidisciplinary consensus.
17 (2021)	Gogna et al.	Propensity score-matched cohort (NIS)	4,311 pts	Surgical nutritional access reduced GI complications in malnourished elderly.	Shows independent prognostic value of enteral access.
19 (2025)	González-Castillo et al.	Systematic review	n/a	Protein 1.5–2.5 g/kg/day; simplified energy targets when calorimetry unavailable.	Practical targets for the highest-acuity patients.
21 (2010)	Al-Omran et al. (Cochrane)	Systematic review / meta-analysis of RCTs	8 RCTs	EN vs TPN reduced mortality (RR 0.50), systemic infection (RR 0.39), organ failure (RR 0.55).	Foundational Cochrane evidence favoring EN.
22 (2020)	Dutta et al. (Cochrane)	Systematic review / meta-analysis of RCTs	5 RCTs	No difference between NG and NJ feeding in mortality, aspiration, or tolerance.	Supports first-line nasogastric feeding.
23 (2008)	Petrov et al.	Meta-analysis of RCTs	RCTs in SAP	EN reduced mortality and infectious complications vs TPN.	Early confirmatory meta-analysis.
24 (2018)	Wu, Li, Sun	Meta-analysis of RCTs	11 studies / 562 pts	EN reduced mortality and infection vs TPN; no heterogeneity.	Contemporary quantitative confirmation.
25 (2013)	Chang et al.	Meta-analysis of RCTs	6 RCTs	NG feeding equivalent to NJ in safety and tolerance.	Corroborates NG-first approach.
26 (2023)	Carlin-Ronquillo et al.	Systematic review of RCTs	4 RCTs / 192 pts	NG feeding as efficacious and safe as NJ.	Most recent RCT-level synthesis.
27 (2011)	Casaer et al. (EPaNIC)	Large multicenter RCT	4,640 pts	Early TPN increased infection, ICU stay, and ventilation vs late initiation.	Defines timing; supports withholding early TPN when EN feasible.
29 (2001)	Van den Berghe et al.	RCT	1,548 pts	Intensive insulin therapy reduced mortality in the surgical ICU.	Basis for glycemic control during nutrition support.
30 (2009)	NICE-SUGAR Investigators	Large multicenter RCT	6,104 pts	Refined glycemic target to 140–180 mg/dL.	Current standard glucose target under TPN.
31 (2008)	Besselink et al. (PROPATRIA)	Multicenter RCT	296 pts	Probiotics increased mortality (16% vs 6%) in predicted SAP.	Establishes probiotics as contraindicated in SAP.

AP, acute pancreatitis; EN, enteral nutrition; TPN, total parenteral nutrition; NG, nasogastric; NJ, nasojejunal; RR, relative risk; NIS, National Inpatient Sample; RCT, randomized controlled trial.

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