

**Figure 4.** Small bowel vs gastric feedings, nutrition efficiency.

−0.82 days; 95% CI, −1.29 to −0.34;  $P = .0007$ ). Hospital LOS and mortality were not significantly different. These differences in outcome from the separate routes of feeding largely reflect findings from older studies and may diminish in the future with improvements in glycemic control, protocolized medical management, and new lipid emulsions.

**Question:** Is the clinical evidence of contractility (bowel sounds, flatus) required prior to initiating EN in critically ill adult patients?

**B3. Based on expert consensus, we suggest that, in the majority of MICU and SICU patient populations, while GI contractility factors should be evaluated when initiating EN, overt signs of contractility should not be required prior to initiation of EN.**

**Rationale:** The literature supports the concept that bowel sounds and evidence of bowel function (ie, passing flatus or stool) are not required for initiation of EN. GI dysfunction in the ICU setting occurs in 30%–70% of patients, depending on the diagnosis, premorbid condition, ventilation mode, medications, and metabolic state.<sup>70</sup>

Proposed mechanisms of ICU and postoperative GI dysfunction are related to mucosal barrier disruption, altered motility, atrophy of the mucosa, and reduced mass of GALT. GI intolerance has been variably defined (eg, absence or abnormal bowel sounds, vomiting, bowel dilatation, diarrhea, GI bleeding, high gastric residual volumes [GRVs]) and appears to occur in up to 50% of patients on mechanical ventilation. Bowel sounds are indicative only of contractility and do not necessarily relate to mucosal integrity, barrier function, or absorptive capacity.

The argument for initiating EN regardless of the extent of audible bowel sounds is based on studies (most of which involve critically ill surgical patients) reporting the feasibility and safety of EN within the initial 36–48 hours of admission to the ICU.

Nonetheless, reduced or absent bowel sounds may reflect greater disease severity and worsened prognosis. Patients with normal bowel sounds have been shown to have lower ICU mortality than those with hypoactive or absent bowel sounds

(11.3% vs 22.6% vs 36.0%, respectively).<sup>71</sup> ICU LOS has been shown to increase with greater number of symptoms of GI intolerance (2.9 days when asymptomatic vs up to 16.8 days with 4 symptoms of intolerance).<sup>72</sup> Not surprising, success of EN delivery is reduced with a greater number of symptoms of GI intolerance. A greater number of signs of intolerance may warrant increased vigilance as EN is started and may necessitate further clinical evaluation.

**Question:** What is the preferred level of infusion of EN within the GI tract for critically ill patients? How does the level of infusion of EN affect patient outcomes?

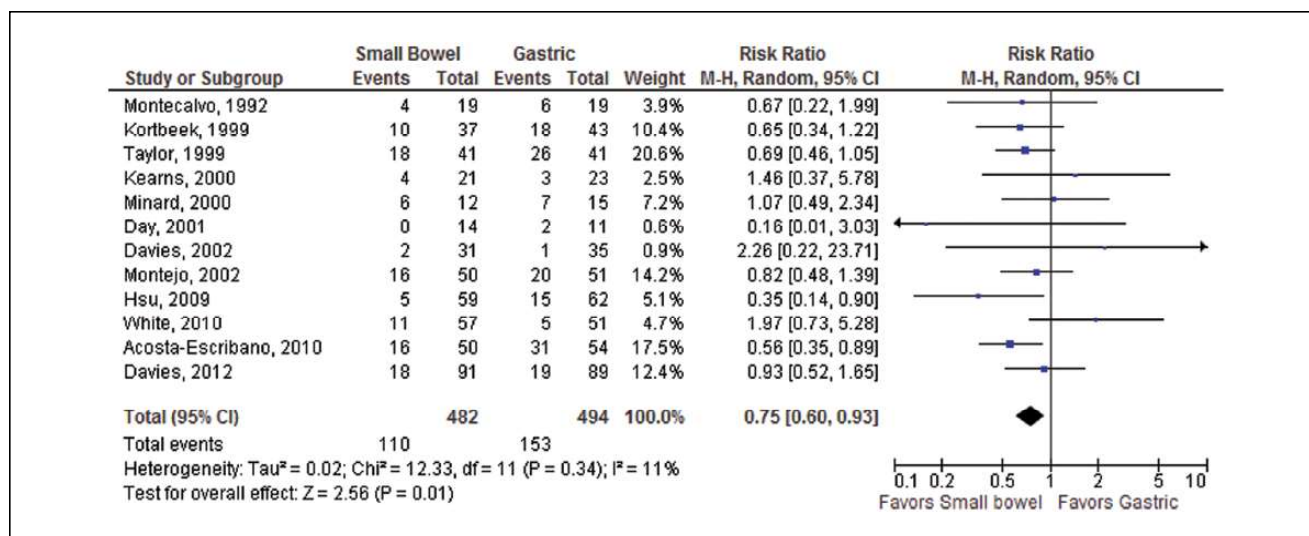
**B4a. We recommend that the level of infusion be diverted lower in the GI tract in those critically ill patients at high risk for aspiration (see section D4) or those who have shown intolerance to gastric EN.**

**[Quality of Evidence: Moderate to High]**

**B4b. Based on expert consensus we suggest that, in most critically ill patients, it is acceptable to initiate EN in the stomach.**

**Rationale:** Initiating EN therapy in the stomach is technically easier and may decrease the time to initiation of EN. The choice of level of infusion within the GI tract (ie, whether the tip of the feeding tube is in the stomach, different segments of the duodenum [D1, D2, D3, or D4], or the jejunum) may be determined by patient selection within ICU practitioners' institutional framework (ease and feasibility of placing small bowel enteral access devices, institutional policies, and protocols).

In the largest multicenter RCT to compare gastric versus small bowel EN in critically ill patients, Davies et al found no difference in clinical outcomes between groups, including LOS, mortality, nutrient delivery, and incidence of pneumonia.<sup>73</sup> Aggregating the data from the RCTs that met our inclusion criteria, 6 trials reported on improved nutrient delivery with small bowel feedings (WMD = 11.06%; 95% CI, 5.82–16.30%;  $P < .00001$ ) (Figure 4),<sup>73–78</sup> and 12 trials demonstrated a reduced risk of pneumonia compared with gastric EN (RR = 0.75; 95%



**Figure 5.** Gastric vs small bowel feedings, pneumonia.

CI, 0.60–0.93;  $P = .01$ ) (Figure 5).<sup>73–84</sup> Although small bowel EN decreases the risk of pneumonia, there is no difference in mortality or LOS between small bowel and gastric EN. Therefore, if timely obtention of small bowel enteral access device is not feasible, early EN via the gastric route may provide more benefit than delaying feeding initiation while awaiting small bowel access.<sup>73</sup>

*Question:* Is EN safe during periods of hemodynamic instability in adult critically ill patients?

**B5. Based on expert consensus, we suggest that in the setting of hemodynamic compromise or instability, EN should be withheld until the patient is fully resuscitated and/or stable. Initiation/reinitiation of EN may be considered with caution in patients undergoing withdrawal of vasopressor support.**

**Rationale:** At the height of critical illness, EN is being provided to patients who are prone to GI dysmotility, sepsis, and hypotension and thus are at increased risk for subclinical ischemia/reperfusion injuries involving the intestinal microcirculation. Ischemic bowel is a very rare complication associated with EN.<sup>85</sup> In a retrospective review of patients requiring stable low doses of vasopressors, those patients receiving early delivery of EN had lower ICU mortality (22.5% vs 28.3%;  $P = .03$ ) and hospital mortality (34% vs 44%;  $P < .001$ ) than those receiving late EN, respectively. The beneficial effect of early EN was more evident in patients treated with multiple vasopressors (OR, 0.36; 95% CI, 0.15–0.85). When adjustments were made for confounding by matching for propensity score, early EN was associated with decreased hospital mortality.<sup>86</sup>

While EN may be provided with caution to patients on chronic, stable low doses of vasopressors,<sup>76</sup> EN should be withheld in patients who are hypotensive (mean arterial blood pressure  $<50$  mm Hg), in patients for whom catecholamine agents (eg, norepinephrine, phenylephrine, epinephrine, dopamine) are being initiated, or in patients for whom escalating doses are required to maintain hemodynamic stability.

For patients on vasopressor therapy receiving EN, any signs of intolerance (abdominal distention, increasing nasogastric [NG] tube output or GRVs, decreased passage of stool and flatus, hypoactive bowel sounds, increasing metabolic acidosis and/or base deficit) should be closely scrutinized as possible early signs of gut ischemia, and EN should be held until symptoms and interventions stabilize.

## C. Dosing of EN

*Question:* What population of patients in the ICU setting does not require nutrition support therapy over the first week of hospitalization?

**C1. Based on expert consensus, we suggest that patients who are at low nutrition risk with normal baseline nutrition status and low disease severity (eg, NRS 2002  $\leq 3$  or NUTRIC score  $\leq 5$ ) who cannot maintain volitional intake do not require specialized nutrition therapy over the first week of hospitalization in the ICU.**

**Rationale:** Patients admitted to the ICU are a heterogeneous group with varying degrees of nutrition risk and disease severity. Occasionally, patients with low nutrition risk, normal