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Part II Enteral Feeding: Eradicate Barriers with Root Cause Analysis and Focused Intervention

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Identifying issues that interfere with adequate provision of enteral nutrition (EN) and getting to the root cause of EN "intolerance or complications" increases the likelihood that patients will receive the nutrition intended. Part I of this series discussed two of the most common barriers to EN: the practice of listening to bowel sounds and checking gastric residual volumes as determinants of GI tract function. In Part II, several other barriers to effective EN are discussed, including diarrhea, nausea, vomiting, pain, constipation, and initiation and progression of EN.

Already Published or Upcoming in this Series:

- **Part I Enteral Feeding Barriers: Pesky Bowel Sounds & Gastric Residual Volumes**
- **Part III Jejunal Feeding: The Tail is Wagging the Dog(ma): Dispelling Myths with Physiology, Evidence, and Clinical Experience**
- **Part IV Enteral Feeding: Hydrating the Enterally-Fed Patient—It Isn't Rocket Science.**

CASE

A 40 year old male was admitted with severe odynophagia, dysphagia & "tube feeding intolerance." His recent medical history includes squamous cell carcinoma of the tongue. He is now undergoing chemo and radiation therapy, with a percutaneous endoscopic gastrostomy (PEG) placed prior to starting this therapy. His medical history also includes hypertension, alcohol misuse, and significant smoking. His home EN regimen prior to admission was 6 cans per day of a 1.5cal/mL product, but the patient has only been able to take in 3-4 cans per day. He recently saw an LIP for his inability to tolerate EN and was changed to 2.0cal/mL product; however, he was admitted right after it was delivered to his home and he had yet to

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try it. Upon interviewing the patient, it was evident he was in agony and that it clearly hurt him to answer basic questions–swallowing even his own saliva felt like "swallowing ground glass." Due to the patient's obvious pain, the interview was kept short and consisted of only yes and no questions. The barriers keeping him from consistently taking his EN were: poor pain control and feeling full/nauseated soon after taking his EN. He stated he took his tube feeding over the course of 30 minutes at home — (i.e., was not bolusing the formula in over 5 minutes), and was also not constipated, despite the use of opiates. The primary team was planning on escalating his pain medications. The nutrition support clinician reviewed the patient's medication orders and noted an antiemetic ordered "pro re nada (PRN)," but only one dose had been given to the patient in 3 days. It was recommended to the primary team to schedule patient's antiemetic to every 8 hours vs. prn. Twenty-four hours later, after he had received 3 doses of his antiemetic, the patient was tolerating all of his feedings without complaint.

INTRODUCTION

In this era of high tech medicine, clinical skills may be eclipsed by new technologies, diagnostics, and therapeutic advances. However, basic clinical assessment skills are critical for accurate assessment of the enterally-fed patient. Many issues interfere with patients receiving the full amount of enteral nutrition (EN) ordered (see also Part 1 of this series). Not the least of these issues are patient specific barriers, which are often widely referred to as, "EN intolerance or complications" (Table 1). However, "EN intolerance or complications" is extremely vague and requires further exploration by the clinician in order to effectively intervene. The real problem may be related to the underlying disease state, inadequate or inappropriate medication treatment (such as PRN orders that are never given), or perhaps the wrong medication for the "job." In some cases, the patient may not be able to articulate what is wrong, and it is easy to attribute the patient's symptoms to EN. Simply blaming symptoms on EN may prevent the clinician from identifying the root cause of the barrier, resulting in decreased EN delivery to patients. Part I of this 4 part series reviewed the evidence (or lack thereof) behind the use of bowel sounds as a determinant of GI function and the waning (but persistent) use of gastric residual volumes as a surrogate measurement of EN tolerance. Part II will cover other common GI issues that get in the way of effective EN delivery, including diarrhea, nausea, vomiting, pain, constipation, and initiation and progression of EN. With a better understanding of the GI tract and normal GI function, it is possible to overcome many GI barriers and develop successful EN regimens that actually meet the nutritional needs of our patients.

Table 1. Common Patient Specific Barriers–Often Referred To As "GI Intolerance" or "EN Complications"

- "Abdominal discomfort"
  - Abdominal pressure
  - Fullness
  - Nausea
  - Vomiting
  - Cramping
  - Belching
  - Gas/bloating/distension
  - Dumping
  - Diarrhea
  - Constipation
- Pain/mucositis
- Flow rate advancement "fear"
- Bolusing EN too fast (2-5 minutes)
- Anatomical changes
- Untoward effects of medications
- Active disease process
- Psychosocial
  - Stress
  - Depression
  - Health condition/diagnosis
  - Financial issues, etc.

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PATIENT'S SYMPTOMS AS A BARRIER

Diarrhea

Diarrhea is an alteration of the normal balance of absorption to secretion within the bowel. Under normal circumstances, nine to ten liters of endogenous and exogenous fluid are introduced to the GI tract each day (see Part I of this series). Yet, the normal stool volume in adults is only 100-200mL. Diarrhea results from increased water content of stool due to an imbalance in intestinal processes involved in the absorption of ions, organic substrates, and thus water. In osmotic diarrhea, stool output is proportional to the intake of the non-absorbable substrate; stool volume decreases quickly with discontinuation of the offending nutrient/agent. In secretory diarrhea, the epithelial cells' ion transport processes reverse into an active secretory state. The volume of stool output and fluid loss can be very high; however, nutrient absorption often remains intact.

The reported incidence of diarrhea in the hospitalized patient varies greatly from 20% - 70%. This is due to the multiple definitions of diarrhea in the literature, no defined volume or frequency that quantifies diarrhea, subjective diagnoses of diarrhea by both clinicians and patients, and other factors. Lebak identified 33 definitions in the literature, and the definition appeared to be based on the preference of the investigator.[1] It is also of utmost importance to ask patients what their normal stool habits are at home/or pre-illness, so clinicians are not trying to fix something that has been going on long before EN was initiated (although in some cases the problem may still need to be fixed, it is just clearly not the result of EN).

Diarrhea can be both a sign and/or a symptom of an underlying issue, but it is not a disease unto itself. Diarrhea occurs for a variety of reasons in hospitalized patients,[2-8] regardless of whether they are on an oral diet, EN, PN or even NPO.[2,9-11] (Table 2). In the enterally-fed population, diarrhea has long been associated with (and blamed on) the enteral formula and/or delivery method. However, randomized, prospective trials demonstrating EN as a cause of diarrhea, have yet to be done and diarrhea has yet to be causally linked to EN (liquid in ≠ liquid out).[12-15] As far back as 1981, Bloom remarked, "gastrointestinal upset in nasogastrically-fed patients is not always the result of the tube feeding and should not be an accepted consequence".[16] The authors went on to carefully explore diarrhea in EN-fed patients and were able to identify medications as the primary causative agents. In fact, in one study of EN-associated "GI intolerance", diarrhea was observed in 26% (36/137) of patients, while 29% (40/137) exhibited constipation.[17] What is perplexing is that if a patient has diarrhea while on a clear, full, or regular diet, the diet is not typically blamed; hence, why is EN blamed for diarrhea? This assertion is counterintuitive to GI physiology.

Malabsorption

Some clinicians have the misconception that diarrhea equals malabsorption. In fact, the GI tract is so effective in its digestive and absorptive role, >90% of nutrients are completely absorbed within the first 5 feet (150cm) of jejunum in normal subjects.[18,19] A large portion of the GI tract or digestive organ function must be lost to result in malabsorption. Patients with moderately impaired GI tracts are still able to absorb many intact nutrients,[20,21] and even those with a total pancreatectomy are able to utilize greater than 60% of intact protein.[22] Patients fed into the duodenum or jejunum do not routinely require a pre-digested formula, as the digestive capacity of the small bowel is enormous. While malabsorption is certainly on the list of things to consider in those patients who have risk factors, only a small percentage of the EN-fed population malabsorb. In any patient suspected of malabsorbing their EN, a 48-72 hour fecal fat collection (done while the patient actually receives the prescribed formula that they are thought to be malabsorbing) will provide the answer.

Contributions of Medications

Medications are a common, but often unrecognized, cause of diarrhea in the EN-fed patient. Liquid medications frequently contain sorbitol or other sugar alcohols, which can be very diarrheagenic.[4,5,10,23,24] Sorbitol is a poorly absorbed polyalcohol; 20-50g/day has been shown to cause osmotic diarrhea, although even 5-10g is enough in some patients.[25] For example, one dose of acetaminophen liquid contains 5.47 g of sorbitol/500mg dose; amantadine, 6.4g/100mg;

Table 2. Potential Causes of Diarrhea in the Hospitalized Patients [2,9,10,61]

Osmotic	Secretory
Food/EN	**Constipation**
The following may aggravate diarrhea IF present.	○ Stooling around an impaction
• Lactose	**Bile acid malabsorption**
• Fermentable, Oligo-saccharides, Di-saccharides, Mono-saccharides And Polyols (FODMAPs)	**Infectious etiologies**
• Fiber-containing EN in some patients	• Clostridium Difficile
Medications*	• E. coli
• Antibiotics	• C. jejuni
• Liquid meds w/ sugar alcohols (sorbitol, mannitol, xylitol):	**Reduced mucosal surface area**
○ Tylenol exlixir, Guaifenesin liquid, multivitamin/mineral liquid, KCl elixir, PPI suspension	• Radiation enteritis
• Lactulose	• Intestinal ischemia
• Magnesium supplements	• Short bowel syndrome
• Phosphate	• High out ostomy
○ Neutraphos/ Kphos packets	**Inflammatory Processes**
• Standing orders for stool softeners/ laxatives	• Crohn's disease
Disease processes	• Ulcerative colitis
• Pancreatic insufficiency	• Microscopic colitis
• Small bowel bacterial overgrowth	• Celiac disease
• Diarrhea predominant irritable bowel syndrome	**Dysregulation**
	• Diabetes enteropathy
	• Post-vagotomy syndrome
	• Hyperthyroidism
	Neuroendocrine tumors
	• Carcinoid

*Note: The effects of medications can be additive; hence the more liquid medications a patient is on, the higher the chance of diarrhea.

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and finally, metoclopramide liquid, 3.5g/10mg (therefore, the diarrheagenic effects of liquid Reglan are NOT from its prokinetic effects as it only is effective on the upper gut, not the colon).[26] Liquid medications are also additive in their effect; the more liquid meds, the higher likelihood diarrhea will follow. Diarrhea is often associated with EN in these cases as once enteral access is obtained, medications are frequently changed to liquid form for ease of administration via the feeding tube. Hence diarrhea seems to start at the same time as the EN (Table 2).

Antibiotic-Associated Diarrhea

Antibiotic-associated diarrhea and Clostridium difficile (C. difficile) are frequent causes of diarrhea in the hospitalized patient.[4,6,12,15,27,28] Patients

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receiving EN are at a higher risk for acquiring C. difficile.[8] One study reported EN-fed patients were nine times more likely to develop C. difficile-associated diarrhea than matched non-EN-fed patients (possibly from the hands of health care providers); the risk was even greater when patients were fed postpylorically (delivery below the gastric acid barrier may facilitate the introduction and survival of C. difficile organisms).[8]

Hypoalbuminemia

Although hypoalbuminemia has been cited as a risk factor for EN related diarrhea, no evidence exists to support this notion.[6] Hypoalbuminemia is also associated with sicker patients (ICU, abdominal abscess, etc.), and sicker patients get more infections (hence, more antibiotics), and are in the hospital longer (with even more medications and more infections). These factors are known to precipitate diarrhea. There is no data that patients with hypoalbuminemia absorb less than healthy controls or absorb inadequate amounts.[29]

Osmolality or Hypertonicity

Despite the perception that osmolality (or hypertonicity) is responsible for triggering diarrhea in patients receiving EN, there is no evidence to support this. The GI tract is adept at diluting and digesting food and liquids of various tonicities. When volume is delivered into the stomach, the volume receptors in the stomach respond by adding a secretory volume.[30] After mixing with gastric secretions and saliva in the stomach, chyme leaves the stomach and is further diluted by bile salts, pancreatic enzymes, bicarbonate, and water secreted into the small bowel. Borgstrom demonstrated that a 500mL test meal (625kcal w/ 40% fat, 15% protein, 45% carbohydrate) is diluted to a volume of 1500-2000mL during passage through the duodenum.[18] This process increases the pH and dilutes the solution — "auto-isotonicity" if you will. This is a normal function of the stomach and small bowel. It is incorrect to think that when EN is infused it is the only thing present in the stomach and bowel. One study showed that hypertonic formulas (544mOsm) infused gastrically are nearly isotonic by the time they reach the ligament of treitz (10 inches [25cm] distal from the pylorus),[31] while another found that hypertonic formulas infused at the ligament of treitz are nearly isotonic 14 inches (35cm) distal in the jejunum.[19] Pesola demonstrated a difference in stooling frequency prior to initiation of EN in 39 subjects (5 volunteers, 10 head and neck cancer patients, and 24 ICU patients).[32] However, after initiation of full strength, hypertonic EN (Ensure Plus® – 690mOsm) at 30cal/kg/day by gravity drip or bolus (head and neck patients), no significant difference in diarrhea between groups was found during feeding.[32] Jones et al found no evidence to implicate hypertonicity of EN as an etiology of diarrhea in their study.[15] Finally, Kandil et al continuously infused an average of 275mL/hour (range: 198 to 340mL/hour or 5000 to 8650 kcal/day) of standard, polymeric EN into the duodenum of five healthy volunteers before precipitating diarrhea in their subjects.[33] The authors suspected it was the sheer amount of magnesium that was infused with that volume of EN that precipitated the diarrhea (given how poorly absorbed magnesium is).

Diluting Enteral Formulas to "Treat" Diarrhea

As discussed above, diluting enteral formulas to decrease osmolality in patients with normal anatomy flies in the face of GI physiology, and is without evidence. Researchers have shown that hypertonic formulas are tolerated in both healthy subjects[34] and in those with impaired GI function.[35] Furthermore, the practice of diluting EN can be detrimental to patients as fewer nutrients are provided, and more handling introduces potential contamination with infectious agents. Regardless, with the recent adoption of the ready to hang system, dilution of EN is not possible in the hospitalized setting. Finally, many items commonly provided to our hospitalized patients, including medications, popsicles, fruit juice, soda, and sherbet all have an osmolality much higher than that of EN (Table 3). If high osmolality causes diarrhea, "isotonic" medications, beverages, and oral diets would be needed to prevent diarrhea in all our patients.

There are two circumstances when the dilution of formula may be helpful (primarily in the home setting). With some particularly viscous EN formulas, dilution may be needed. If a highly

Table 3. Osmolality of Selected Liquids [62] and Medications [5,63-65]

Typical Liquids	(mOsm/kg)	Drug	(mOsm/kg)
EN formulas	250-875	Acetaminophen elixir	5400
Milk/eggnog	275/695	Diphenoxylate/atropine susp.	8800
Gelatin	535	KCl elixir (sugar-free)	3000
Broth	445	Cephalexin susp.	1950
7-up/cola	640/750	Lasix (oral)	3938
Popsicles	720	Reglan	8350
Juices	~ 990	Multivitamin liquid	5700
Ice cream	1150	Na Phosphate	7250
Sherbet	1225	Nystatin susp.	3300
Instant breakfast	715	Ergocalciferol Solution	16,100
Prune juice	>1000	Lactulose syrup	3600
Gatorade	330	Barium liquid (w/ flavoring)	148-194
Tea w/ 1 tsp sugar	106	Gastrografin	> 2150
Coffee	83	Ferrous sulfate liquid	4700
Diet soda	50	Sodium phosphate liquid	7250

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viscous EN formula is slow to infuse, adding water can thin the formula and enhance flow through small bore feeding tubes. Also, in some patients with higher fluid requirements, water can be added to the EN formula and the mixture run at a higher infusion rate to provide additional hydration. This will decrease the burden of large, frequent water flushes and decrease caregiver time.

Diarrhea is seen in EN-fed patients for a variety of reasons, but EN is very rarely, if ever, the cause. Risk factors other than the enteral formula should be explored including: medications, infectious etiologies, underlying disease state, GI anatomy, and even constipation (stooling around an impaction) in susceptible individuals.[11] These issues should be addressed and appropriate steps taken before reducing or suspending enteral feeding. Management of diarrhea in EN-fed patients requires a systematic approach to identify and remove risk factors where possible.[6] (Table 4). Ferrie decreased the incidence of diarrhea in critically ill patients from 37% to 24% by careful attention and monitoring of factors known to cause or aggravate diarrhea.[36] Once infectious or other etiologies have been ruled out, anti-diarrheal agents can be initiated to improve patient comfort and protect from skin breakdown. Diarrhea as a symptom does not indicate the need for cessation of EN.

Nausea, Vomiting, Abdominal (or any) Pain

It is not uncommon for patients in the hospital setting (or any patient with ongoing medical issues) to have nausea, vomiting, or pain. These symptoms often result in inadequate oral intake in patients who are eating. In patients being enterally-fed, these symptoms often cause EN to be held due to a belief that EN is causing the symptoms. In some cases, parenteral nutrition (PN) is initiated. Effective use of medications, such as antiemetics, prokinetics, or analgesia agents, can improve nausea and vomiting, and these modalities should be optimized before surrendering to PN. Of course, the route of medication delivery is an important consideration. For example, oral medications may not be effective if the patient is frequently vomiting. A medication delivered into the stomach will not be utilized if the patient is on gastric suction or is

Table 4. Systematic Approach when Addressing Diarrhea in EN-fed Patients

- First determine patients normal stooling pattern prior to illness.
- Quantify stool volume—determine if it is really diarrhea.
  - Ensure Strict I & O ordered, (Not just "I & O")
- Review medication list for known culprits:
  - Stooling agents, lactulose, Kayexalate, multiple neutral-phos packets, etc.
  - Elixirs or suspensions with sorbitol (not always listed on the ingredient list—may need to contact manufacturer).
- Try to correlate timing of diarrhea in relation to start of new medication(s) or a switch of medications to enteral route once enteral access is obtained.
- Check for *C. difficile* or other infectious etiologies if appropriate.
- In some patients, Fructooligosaccharide (FOS) and fiber-containing EN may precipitate or aggravate diarrhea.
- Avoid liquid medications in those with diarrhea.
  - Multiple liquid medications can have additive effects
- In those suspected of stooling around an impaction, obtain abdominal film for "stool burden."
- Once infectious etiologies (and impaction) are ruled out:
  - Consider an antidiarrheal agent (may need standing order vs. " prn" to be effective)
- Check total hang time of EN (should not exceed 8 hours) (open systems only).
- Give protein modules by bolus vs. adding directly to EN formulas to decrease contamination risk.
- In those patients at risk for pancreatic insufficiency, consider checking fecal elastase in those with formed/semi-formed stools; in those with loose stools, a quantative fecal fat.
- Consider bile acid malabsorption in patients at risk.

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frequently 'venting' a gastric tube to relieve nausea. The timing of medications may also be important in these settings (e.g. ½ hour before meals to maximize efficacy) and, if so, these instructions should be included in the recommendations and orders.

The Curse of "PRN" Medications

PRN drug use, or medications given when the need arises, traditionally meant "as little as possible."[37] PRN orders are routine in hospital, rehabilitation, and nursing home settings and are the default ordering method in many institutions. It has been reported that 35-60% of medication orders are PRN.[38,39] Many patients have suffered at the mercy of these "PRN" orders, as no medication is beneficial if not received by the patient. There is a paucity of data regarding PRN medications and how often they are actually given.[39-41] There are numerous reasons that PRN orders may not be given: patient does not (or cannot) complain of symptoms routinely, patient does not know meds are available to them (let alone know how to pronounce them), nurses do not get to fully assess the patient's symptoms, or nurses just run out of time to give PRN meds. A Cochrane review was unable to find any trials comparing scheduled dosing with giving the same medication only "when needed".[40] One study investigated the non-use of PRN medications in a hospital-affiliated with a large mid-western university and found that 62% were unused (4793 of 7735 PRN orders).[38] Non-use by service category was also

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assessed, revealing that cardiovascular surgery had the highest laxative prescribing rate (almost 100% of patients), yet 89% went unused. The percent of all PRN orders unused ranged from a low of 50% for renal transplant to a high of 81% for ophthalmology. In another study of PRN orders for acute pain management following laryngectomy, 68% of patients met the recommended minimum post-op dosing guidelines for pain, yet none of the patients received the intended dose during a 24 hour period while hospitalized.[41] Of the 13 patients (35%) whose physicians were contacted because of inadequate pain relief, only 8 patients (22%) had their narcotic dose increased appropriately. Finally, in a study of children undergoing various elective surgeries, the authors verified that nurses administered 20% of the non-narcotics available under PRN orders, but only 10% of the available narcotics.[37]

In patients with ongoing symptoms that prevent consistent delivery of EN, it is important to ensure that medications to relieve such symptoms are actually being received by the patient. Always look to doses received, not just ordered. If the patient is not receiving the medication, it is important to find out why — is it being refused? Or, is the medication ordered only as a "PRN"? It may be important to explain to the patient the benefit of the medication, discuss with nursing to determine why it is not being given, or recommend to the primary team that the medication be changed from PRN to scheduled dosing. With some medications, it may be important to go one step further and ensure it is scheduled at specific times. It takes a village to get our patients safely and comfortably EN-fed through a hospitalization.

Constipation

Constipation is a frequent problem in hospitalized patients and is associated with abdominal discomfort, distension, small bowel bacterial overgrowth, poor tolerance of EN, confusion, intestinal obstruction, vomiting, and increased intra-abdominal pressure (which can impact respiratory function).[42,43] Constipation has many possible causes (Table 5). In patients with significant constipation (especially rectal distension), abdominal distension, as well as delayed gastric emptying, can occur due to the recto-esophagogastric reflex.[44] In more than one study, constipation was reported more frequently than diarrhea in patients fed exclusively by EN.[17,45] Another study in cancer patients indicated that symptoms of constipation cause more distress than symptoms of pain.[46] Modern definitions define constipation as a poly-symptomatic disorder including various aspects of disturbed defecation. Despite being such a common problem, constipation

Table 5. Risk Factors for Constipation

Primary	Secondary
• Increased age • Medications (narcotics esp.) • Nursing home residence • Low socioeconomic status • Decreased physical activity • Immobility • Female sex • Diet and lifestyle • Fiber "deficiency" (~ 30% only) • Travel • Pregnancy • Poor bowel habits • Ignoring the urge to defecate	• Structural o Colon cancer o Stricture/obstruction o External compression o Constipation-predominant IBS • Neurologic o Amyotrophic lateral sclerosis o Multiple sclerosis o Dementia o Parkinson's o Spinal cord/traumatic brain injury o Stroke o Paraplegia o Quadriplegia • Endocrine o Hypothyroidism o Diabetes mellitus o Hyperparathyroidism o Pregnancy • Metabolic o Chronic kidney disease • Myopathic o Myotonic dystrophy o Scleroderma o Amyloidosis

Table 6. Assessing Patients for Potential Constipation

- **Pay attention.**
- Obtain relevant history & identify potential risk factors
  - What is patients normal stooling regimen—daily, weekly, etc.?
  - Are stooling agents used at home?
    - If so, which one/s and how often?
    - Example: patient normally takes MiraLAX® BID at home, then gets admitted and is now on bedrest with narcotics and a docusate (Colace®) q pm is ordered…
  - When was patient's last stool? (if patient or family can communicate/remember)
  - Known bowel disorder associated w/ constipation (see risk factors)
  - Watch for drugs added known to cause constipation (narcotics in cancer patients)
- May need follow-up abdominal film in some patients for "stool burden" to ensure colon fully evacuated (especially after multiple admissions for same problem)

is often overlooked.[42,47,48] While constipation in the EN-fed patient has often been referred to as a "complication" of EN, it is not possible for EN to cause constipation. Constipation in any patient is, pure and simple, due to an underlying condition and is often worsened by a lack of attention from the healthcare team to this issue.

One common intervention to "treat" constipation is to use a fiber-containing EN. However, fiber is no panacea.[49] In one study of critically ill patients, constipation was observed as follows: fiber-free EN – the most widely used – (60% constipated), fiber-containing EN (51% constipated), both types used (85% constipated).[43] See Table 6 for suggested guidelines to prevent and treat constipation.

Can Dehydration Cause Constipation?

Another myth that persists today is that dehydration causes constipation.[50] Dr. Lawrence Schiller, a gastroenterologist affiliated with Baylor University Medical Center in Dallas, Texas, with years of clinical experience and numerous publications on the topic of both constipation and diarrhea, explained this common assumption this way:

> "There is no support for this notion. The observation may be valid (dehydration and constipation coexist more than you would expect by chance), but it is not that dehydration causes constipation. More likely some factor leads to both dehydration and constipation. For instance, someone who is very ill may not drink much water, but they also are not eating so the main stimulus for colon motility (gastrocolic reflex–the stimulation of colonic contractions after food ingestion resulting in a bowel movement a short time after eating) is absent. Because the gut mucosa beyond the stomach is so permeable to water, there will always be "enough" intraluminal water for normal function, even if there is a total body water deficit. Electrolyte disorders that may accompany dehydration (e.g., hypercalcemia) may exaggerate constipation, but the water deficit is not the primary driver of the bowel symptoms."

OTHER FACTORS GETTING IN THE WAY

Initiation & Progression

Initiation and advancement of EN varies among facilities (see Table 7 for one institution's EN initiation protocol). There are no prospective randomized studies to determine the optimal rate to initiate feeding or how quickly to advance. Recommendations for initiation of continuous EN generally start at 20-50mL/hour, and advance by 10-25mL every 4-24 hours. Intermittent or bolus feedings protocols generally start at 120mL every 4

Table 7. UVAHS Protocol for Initiation and Advancement of EN

Continuous Feeding	**Medicine NST:** Unless 2.0cal/mL product, or rate would exceed refeeding goal: Begin at 50mL/hour x 4 hours; advance by 20mL every 4 hours until goal rate is reached. Also have the option to begin feeding at goal rate. • If a 2.0cal/mL is used, EN is started at 25-30mL/hr. **Surgical NST:** 20-25mL/hour and advance by the same amount every 4 hours to goal.
Bolus (Intermittent) Feeding	125mL x 1 feeding; if tolerated advance by 125mL every feeding until goal is reached

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hours, and advance by 30-60mL every 8-12 hours.[51] The results of a recent survey of dietitians in the United Kingdom (n = 606), demonstrated that 65% of respondents reported most commonly using a start rate of 24–49mL/hour, with 50-74mL/hour being the next most common initiation range.[52] A significant association between the number of years in clinical practice and start rate was found--with those having more clinical experience using a higher start rate.

Extremely slow protocols for EN advancement can lead to decreased nutrition provided to patients. When one considers the actual amount of EN that is provided at a typical flow rate (for example, 60mL/hour equals ¼ cup delivered over an hour), these advancement protocols seem very conservative. Over the years, various researchers have demonstrated that rates anywhere from 87mL/hour[34] to 100-150mL/hour.[53] are generally well tolerated. In fact, in two small studies (6-9 subjects), Heitkemper et al demonstrated that subjects tolerated gastrically infused full strength, hypertonic EN at rates of 30-60mL/minute (yes, mL/minute) up to a total of 500mL and 750mL.[54,55] This translates into 500-750mL being infused over 8-25 minutes. Only at a rate of 85mL/minute did subjects experience GI discomfort.[54]

Although data are sparse on initiating patients at goal flow rates, in addition to the studies above, Taylor et al. compared two different EN starting regimens in 82 head-injured patients.[56] The two groups were either started at a goal rate (90mL/hour) or with a starter regimen of 15mL/hour advancing every 8 hours as tolerated to 30, 60, and then 90mL/hour based on energy requirements. The 90mL/hour group (treatment) included both small bowel and gastrically-fed patients; the starter group enlisted only gastrically-fed patients. There were no significant differences in infectious complications or pneumonias (including aspiration pneumonia).

At discharge, patients going home on pump feedings from University of Virginia Health System (UVAHS) are advised that they can advance their EN rate by 5-10mL/hour every three days or so, until they are running the set number of cans over the number of hours that suits them (or until further advancement is not tolerated). In general, 120-150mL/hour is an acceptable target as long as the patient is "comfortable." Demonstrating just how much 120mL (1/2 cup) is to patients (using a cup available at bedside) may be a helpful visual. The exception to these instructions are those patients on insulin — coordination with their endocrine team is necessary to adjust insulin as the feeding regimen is changed.

Calculating Run Time for Patients on Continuous Feeding

Because of the many barriers to EN and the lost feeding time that results, patients often do not receive the prescribed goal nutrition. One approach to improve the amount of nutrition delivered is to base flow rate calculations on a less than 24-hour time period.[57-59] For example, at UVAHS, the calculations of goal flow rate for continuously fed patients are calculated based on 22 hours/day for ICU and 20-21 hours for floor patients. The EN orders are then entered as continuous, but at the padded rate to account for the expected EN

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downtime. EN rates are then modified as needed, depending on the actual "dose received" in the days that follow.

SUMMARY

EN is a safe and effective way to nourish patients unable to eat enough on their own. Many barriers exist in the hospital setting that impede successful EN delivery to patients; however, many of these obstacles are based on the unsupported perception that EN causes GI symptoms. Part two of this series specifically addresses diarrhea, osmolality, infusion rates, nausea, vomiting, and pain as barriers to successful EN, and provides alternative approaches to maximize nutrient delivery in the enterally-fed patient. ■

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