Series Editor: Carol Rees Parrish, M.S., R.D., CNSD

Enteral Feeding: Dispelling Myths

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Total enteral nutrition (TEN) is accepted as the preferred method of feeding patients who require nutritional support. Although a majority of patients can be successfully fed with TEN, issues of GI tolerance do arise. The most common of these gastrointestinal concerns include: lack of bowel sounds, initiation and advancement of TEN, interpreting gastric residual volumes, the onset of diarrhea, nausea/vomiting, and osmolality or hypertonicity of formulas. Unfortunately, many current clinical practices are not evidenced-based, leaving tradition, dogma and personal beliefs to prevail. The goal of this article is to provide a review of enteral feeding practices considering gastrointestinal anatomy and physiology and identifying the evidence available (if any) to support traditional approaches to TEN. Suggested guidelines and strategies for overcoming common barriers to effective TEN are provided.

INTRODUCTION

Total enteral nutrition (TEN) is indicated for patients who have a functional GI tract, but are not able to nourish themselves by mouth. TEN is effective when adequate amounts are actually provided to the patient. When compared to parenteral nutrition, it is less expensive, associated with fewer infectious complications, and promotes gut integrity. Unfortunately, there are many issues that arise in the hospital setting that prevent adequate administration of TEN (Table 1).

This article will promote a better understanding of "GI intolerance" by reviewing GI function as it relates to TEN. The article will specifically focus on the most common "intolerance" issues facing clinicians, including:

- Lack of bowel sounds (BS)
- How to initiate and advance TEN
- Interpreting gastric residual volumes (RV)

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- Onset of diarrhea
- Nausea/vomiting/fullness
- Osmolality/hypertonicity of TEN

A brief discussion of feeding post-PEG/Jet-PEG placement is also presented. By combining an understanding of GI anatomy and physiology with clinical assessment and a stepwise treatment approach, TEN related problems can often be successfully managed; a change to parenteral nutrition is rarely necessary. A fully referenced, in-depth review of this topic is also available (2). A review of aspiration in TEN-fed patients appeared in the April 2003 issue of *Practical Gastroenterology* and therefore will not be included.

MYTH 1: BOWEL SOUNDS AND PERISTALSIS GO HAND IN HAND

The decision of whether or not to initiate and/or continue TEN is often based on the presence or absence of bowel sounds (BS). The usual assumption is that BS correlate with peristalsis and, therefore, with the ability to enterally feed. However, there are several problems with this assumption. First, although this practice is often used in the clinical setting, textbooks describe BS in the setting of ileus as varying from hypoactive to non-existent, to high-pitched or hyperactive. Secondly, enteral feeding may stimulate a reflex that results in coordinated propulsive activity and elicit gastrointestinal hormone secretion enhancing bowel motility (3)—"if you feed them, bowel sounds will follow" (the Field of Dreams Approach to BS). Third, if an ileus (and hence lack of peristalsis) were present, gastrointestinal secretions would theoretically build up, ultimately resulting in emesis unless gastric decompression is initiated. Finally, there are no studies that correlate BS with peristalsis or the ability to initiate TEN. On the contrary, many experts feel that TEN can safely be initiated even when BS are not present (4–6). Table 2 provides suggested guidelines for clinical assessment of gastrointestinal function when BS are absent.

MYTH 2: NEVER INCREASE RATE AND STRENGTH AT THE SAME TIME

It is a common belief that TEN needs to be initiated at diluted strength. It is often thought that hypertonic

Table 1
Barriers to TEN delivery in the hospital setting (1)

- Impromptu diagnostic procedures
- Enteral access problems (clogged or pulled tubes)
- Feedings held due to drug-nutrient interactions
- Hemodialysis
- Hypotensive episodes
- Inadvertent hypocaloric TEN orders
- "NPO" at midnight for tests, surgery or procedures
- Physical or occupational therapy
- Transportation off the unit
- Diprivan (propofol)—provides 1.1 lipid calorie/mL infused, therefore, if a patient requires significant dosing, TEN delivery may be limited in order to avoid overfeeding
- **"GI intolerance or dysfunction"**

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Table 2
Suggested Guidelines in the Assessment of GI Function when Bowel Sounds are Absent (1)

- Does patient require gastric decompression? If so, is it meaningful? (i.e., is the volume similar to normal secretions above the pylorus or is it a small volume every shift? For more on this issue, see the section on gastric residual volumes) Distinguish severity by differentiating those patients requiring:
  - Low constant suction vs
  - Gravity drainage vs
  - An occasional residual check every 4–6 hours
- Abdominal exam—distended?
- Is the patient nauseated, bloated, feeling full?
- Is the patient passing gas or stool?
- What is the differential diagnosis? Are abdominal issues high on the list? If the above clinical parameters are benign, consider a trial of TEN at low rate of 10–20 mL/hour and observe.

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Table 3
Osmolality of Selected Liquids and Medications (1)

Typical Liquids	*(mOsm/kg)*	*Drug*	*(mOsm/kg)*
TEN formulas	250–710	Acetaminophen elixir	5400
Milk/eggnog	275/695	Diphenoxylate susp.	8800
Gelatin	535	KCl elixir (sugar-free)	3000
Broth	445	Chloral hydrate syrup	4400
Sodas	695	Furosemide (oral)	3938
Popsicles	720	Metoclopramide	8350
Juices	~990	Multivitamin liquid	5700
Ice cream	1150	Na Phosphate	7250
Sherbet	1225	Cimetidine liquid	4035

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TEN formulas (>300 mOsm) cause diarrhea and GI intolerance. The literature, GI physiology, and clinical experience do not support this idea. Initially saliva, gastric and small bowel secretions (such as pancreatic enzymes, bile salts, bicarbonate and water) neutralize TEN ("autoisotonicity") in the first 10 to 45 cm of the small bowel whether gastric or jejunally delivered respectively (7,8). At least two studies have demonstrated that hypertonic formulas do not lead to GI intolerance (9,10).

Furthermore, TEN formulas have a relatively low osmolality compared to many common liquids and medications routinely ordered for patients (Table 3). For example, the osmolality of a clear liquid diet is higher than that of any TEN formula. In addition, the osmolality of frequently used medications is more than four to seven times that of TEN formulas. It is inconsistent to order diluted TEN formula for some patients while others receive "full strength" clear liquid, full liquid or regular diets. The practice of diluting TEN may actually be detrimental as inadequate amounts of nutrition are then delivered to patients who are already nutritionally compromised. An exception to this may be dilution of TEN solutions for patients who have very high fluid requirements. Water can be added to the TEN and delivered at a higher rate to deliver both nutrition and fluid.

The rate at which TEN is initiated and the protocol for advancement vary greatly from institution to institution. Although many guidelines exist, there is no evidence to support any one protocol for TEN initiation or advancement. The typical textbook recommendation for continuous TEN is to begin at 20–50 mL per hour and advance by 10–25 mL every 4–24 hours, yet there are no clinical studies to support this practice. However, several authors have shown that both healthy patients and those with moderately impaired GI function can tolerate TEN initiated at goal flow (based on total calorie requirements) (11,12). When thinking about TEN flow rates and advancement, it is helpful to first put the volume delivered into perspective. For example, 60 mL of TEN is equivalent to 1/4 cup (4 tablespoons) over an *entire hour* if the patient is on a continuous infusion.

General textbook recommendations for bolus or intermittent TEN advancement are to begin with 60-120 mL every 4 hours and advance by 30–60 mL every 8–12 hours. Healthy volunteers have been shown to tolerate intermittent feedings of 500 mL of TEN at a rate of 60 mL *per minute* and 750 mL of TEN at 30 mL *per minute* (13,14). Based on current evidence, the protocols for TEN initiation and advancement at the authors' institution were developed and are listed in Table 4.

MYTH 3: ALTHOUGH A RESERVOIR, THE STOMACH SHALL HAVE NO RESIDUAL

One of the most common barriers to delivering adequate TEN is concern over gastric residual volumes (RV). Just the words, "residual volume," conjure up the idea that having one is bad. The following beliefs about RV are common:

1. Any type of residual in the stomach is unnatural.
2. Adverse clinical consequences (such as fullness, nausea, vomiting and aspiration) follow an increased gastric residual.

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Table 4
Initiation of TEN at UVAHS (1)

Continuous/Nocturnal feeding
Initiation: Full strength (all products except 2 cal/mL) at 50 mL/hour and increase by 25 mL every eight hours to goal rate. A 2.0 cal/mL product is started at 25 mL/hour (as few patients need 50 mL/hour to meet estimated needs). The final goal rate is dependent on the patient's caloric requirements and GI comfort.

Bolus/Intermittent feeding
Initiation: 125 mL, full strength (regardless of product) every 3 hours for two feedings; increase by 125 mL every 2 feedings to final goal volume per feeding during waking hours.

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One of the primary functions of the stomach is to act as a reservoir; by definition, this means it holds or "stores" things. The only study to date that has tried to evaluate the usefulness of checking RV actually demonstrated that having a gastric residual is normal (15). The study also found a correlation between physical exam and radiographic evidence, but RV did not correlate with either of these.

Although RV are frequently checked in hospitalized patients being fed by TEN, there is no standard definition as to what constitutes a significant RV, how frequently it should be checked, and whether or not it should be returned to the stomach. It is widely assumed that monitoring RV indicates GI tolerance of TEN and may prevent aspiration events if kept below a certain volume. However, evidence for this practice has not been substantiated by prospective, randomized studies.

When discussing the issue of RV, it is helpful to review normal gastric physiology. Approximately 3000–4000 mL of saliva and gastric secretions are produced each day. This is equivalent to an average of 145 mL/hour of fluid passing through the pylorus *in addition to* any food, beverage or TEN provided. Hence, if TEN is running at 100 mL/hr and after 4 hours the RV is 200 mL, this actually means that approximately 780 mL have already passed through the pylorus: [(145 mL secretions/hr + 100 mL TEN/hr) × 4 hrs] – 200 mL residual = 780 mL

If the GI tract were truly "not functioning," the expected residual would be significantly more than the 200 mL RV.

Another under-appreciated factor in truly assessing a RV is the contribution of the "cascade effect." When a patient lies on his or her back (commonplace in the hospital setting) and has a nasogastric feeding tube placed, it is not uncommon for the tip of the feeding tube to settle into the fundus, or non-contractile portion of the stomach. As the spine essentially splits the stomach in half in the supine position, the fundus may fill with TEN until it reaches a volume that is high enough to "cascade" over into the antrum and out the pylorus. Measuring a RV in this case would suggest that emptying may not be optimal, when a simple shifting of the patient to the right side (ever watch a barium swallow ?) will allow the contents to flow out the pylorus.

At the North American Summit on Aspiration in the Critically Ill Patient held in 2002, the available evidence (although scant) surrounding the use of RV was evaluated. The primary reviewer made a recommendation to increase the amount that constitutes a significant RV to 400–500 mL (16). The panel of experts as a whole made the following recommendations in their consensus statement (17):

1. TEN should be held for overt regurgitation or aspiration of gastric contents
2. TEN should be held for RV >500 mL and GI tolerance reassessed
3. RV of 200–500 mL "should prompt careful bedside evaluation" and steps should be taken to minimize aspiration risk
4. RV of <400–500 mL does not ensure tolerance of TEN or prevention of aspiration
5. RV <500 mL should be returned to the patient
6. Clinical assessment should be always be used in combination with RV

Finally, many factors affect gastric emptying (and therefore the assessment of a RV) in hospitalized patients receiving TEN. The most important of these include medications (especially narcotics), effects of illness, post-op ileus, obstruction and hyperglycemia. See Table 5 for suggested guidelines to "treat" a RV.

MYTH 4: NAUSEA, VOMITING OR FULLNESS ARE CONTRAINDICATIONS TO TEN

Some patients receiving TEN may experience nausea, vomiting, or excessive fullness. These symptoms can often be successfully managed, avoiding the switch to parenteral nutrition. The treatment of nausea, vomiting and fullness is similar to that of "treating" a RV (see # 4 through 9 in Table 5). In particular, all too often antiemetics are ordered on a "prn" basis and therefore patients may actually receive very few, if any, doses.

It is not uncommon for patients being fed by a jejunal feeding tube to experience an increase in nausea and vomiting when TEN is initiated. This may be due to a mechanism known as the "ileal brake"(18). If intact nutrients such as fatty acids escape absorption and reach the ileum, a negative feedback mechanism is triggered which, in turn, slows the stomach emptying of nutrients into the small bowel. This may result in nausea, vomiting, fullness, or an increase in gastric residual volumes. These symptoms are usually temporary and generally improve after a period of a few days to a few weeks. Patients with bacterial overgrowth may be at increased risk for this phenomenon. *Temporarily* decreasing the TEN flow rate with more aggressive antiemetic or prokinetic medications, or treatment of bacterial overgrowth with antibiotics may provide symptomatic relief.

MYTH 5: TEN CAUSES DIARRHEA

TEN is often blamed for diarrhea in hospitalized patients. In truth, diarrhea is a common problem in the hospitalized setting, with or without TEN infusion. TEN has yet to be causally linked to the development of diarrhea. Many studies that are available are small and fail to account for other factors that may contribute to diarrhea. Further complicating the issue are the many definitions of diarrhea in the literature.

Diarrhea is often related to medications or other treatments, infectious causes, the underlying disease state, or altered GI anatomy. Diarrhea frequently coincides with the initiation of TEN because many of the medications that had been given by IV are changed to the enteral route at the same time (19,20). Many liquid medications contain sorbitol; patients receiving multiple medications often receive amounts of sorbitol known to cause abdominal symptoms in normal subjects (21). Also, it is not uncommon for patients with diarrhea to receive standing orders for stool softeners

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Table 5
Suggested Guidelines to Treat Elevated Residual Volume (1)

1. Is it a residual? (For example, is it less than the flow rate?)
2. Clinically assess the patient for abdominal distension, fullness, bloating, discomfort
3. Place the patient on his/her right side for 15-20 minutes before checking a RV to avoid the cascade effect
4. Switch to a more calorically dense product to decrease the total volume infused
5. Try a prokinetic agent or antiemetic → review orders for "prn" vs standing vs elixir vs tablets. Typical doses for prokinetics available:
   - Metoclopramide – 5–20 mg qid
   - Erythromycin – 50–200 mg qid
   - Domperidone – 10–30 mg qid
6. Seek transpyloric access of feeding tube
7. Tighten glucose control to less than 200 mg% to avoid gastroparesis from hyperglycemia
8. Consider analgesic alternatives to opiates
9. Consider a proton pump inhibitor in order to decrease sheer volume of endogenous gastric secretions (Ex. omeprazole, lansoprazole, esomeprazole, pantoprazole, rabeprazole). This option is typically used for those patients who will be going home on TEN. It will decrease the sheer volume of gastric secretions and allow discharge without IV hydration in addition to TEN, or decrease it enough that reinfusion through the j-port is manageable.
10. Raise the threshold for what constitutes a residual volume up to 400–500 mL
11. Consider stopping the RV checks if the patient is clinically stable, has no abdominal complaints, or the RV checks have been "acceptable" for 48 hours. There is no need for home TEN patients to check RV's if they were stable in the hospital; no more so than if they were eating by mouth.

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Table 6
Systematic Approach when Addressing Diarrhea in TEN-fed Patients (1)

1. Quantify stool volume—is it really diarrhea?
2. Review medication list (did medications switch from the IV to enteral route when enteral access achieved?)
   Common offenders include:
   - Acetaminophen and theophylline elixir
   - NeutraPhos
   - Lactulose

   Also discontinue any standing orders for stool softeners, laxatives, etc.
3. Check for *C. difficile* or other infectious cause (lactoferrin, leukocytes)
4. Try fiber
   - Few clinical studies
   - Supports the health of colonocytes
5. Once infectious causes are ruled out:
   - Try an anti-diarrheal agent (may need standing order versus " prn")
6. Continue to feed

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or laxatives. Several studies have suggested an association between antibiotics, *C. difficile* and diarrhea in patients being fed by TEN (20,22–24).

It is often assumed that diarrhea equals malabsorption. However, given the redundancy and profound efficiency of the GI tract, this assumption is unfounded. The process of digestion and absorption are intricately coordinated by speed of transit of foodstuffs, coordination of pancreatic and bile salt secretion, and the tremendous surface area dedicated to absorption (equivalent to approximately two tennis courts).

It has been shown that a normal GI tract will absorb carbohydrate, fat and protein within the first three to five feet of the small bowel (7,25,26). One study has demonstrated that diarrhea did not precipitate in normal subjects until a rate of ~275 mL *per hour* of standard TEN was fed naso-duodenally (27). Finally, one study reported that even after a total pancreatectomy, patients can digest and absorb more than 60% of intact protein (28).

A careful, systematic and stepwise approach to evaluating diarrhea in the TEN-fed patient will often uncover the underlying cause (Table 6). A change to an elemental or predigested TEN formula is rarely needed, unless GI function is significantly impaired, malabsorption has been documented, and efforts at medication intervention have failed. TEN can usually be continued while the underlying cause is evaluated and treated.

MYTH 6: TEN ORDERED = TEN RECEIVED

In addition to assessing GI related problems that thwart TEN delivery, it is important to monitor the *actual amount* of TEN *received* by the patient. Due to the many barriers listed in Table 1, full TEN delivery is the exception and not the rule. TEN may need to be scheduled around procedures or therapies, or the rate may need to be "padded" to account for unexpected time off the TEN infusion. See Table 7 for strategies used at the authors' institution to decrease the "downtime" off TEN.

Patients in the hospital frequently have their TEN held after midnight for procedures the following day. This can lead to a great deal of time off TEN and result in decreased nutrient provision to the patient. A recent study by McClave of 71 patients undergoing esophagogastroduodenoscopy (EGD) demonstrated that patients receiving TEN up to 4 hours before the procedure did not have increased gastric residuals or an obscured view of the mucosa (29).

A WORD ABOUT POST-PEG OR PEG/J FEEDING… HOW SOON CAN YOU FEED?

Protocols for initiating TEN after PEG placement vary from institution to institution. A recent review indicates general practice is to begin feeding within 6 to 24 hours post PEG placement (30). Waiting an extended period of time to feed post-PEG placement will further increase time to full nutrient provision and possibly increase hospital costs. Numerous studies have shown that TEN can safely be initiated within 4 hours post PEG placement with no increased risk or rate of complications (31). At our institution, the standard protocol for TEN initiation is 6 hours post PEG placement. At that time, TEN is begun at whatever rate that was being

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Table 7
Strategies to Improve Delivery of TEN at UVAHS (1)

- Base flow rates on < 24 hours (as no patient will ever get exactly 24 hours of TEN in the real world)
  - ICU setting = 22 hours
  - Floor beds = 20 hours (16 hours if TEN is held for phenytoin suspension BID-TID)
  - Stable medical ICU patients: the run time is changed to 1500–0900 to allow for planned trips off the unit for procedures (unless on an insulin drip)
- If TEN delivery still falls short of 100% of estimated needs, cyclic TEN (defined as less than 24 hour infusion) is ordered with a defined amount of TEN formula to infuse specified. Examples of such orders include:
  - Run TEN at 75 mL/hour from 1900–0700 for total of 900 mL
  - Start TEN at 1900 and run at 75 mL/hour until 5 cans infused
  - Adjust pump setting to provide a "dose delivery" of TEN (example: start TEN at 1900 and run at 100 mL/hour until 1250 mL infused).
- If jejunally fed, TEN can often run up to the time the patient leaves the unit vs arbitrarily stopping at midnight for procedures such as angiography, modified barium swallows, etc.
- Reevaluate protocols that require TEN to be stopped at midnight for procedures, angiography, modified barium swallows, endoscopy (depending on the procedure of course, etc.)

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given the night before PEG placement. If the patient has yet to receive TEN, then the protocols listed in Table 4 are instituted based on the regimen selected.

There is little information available on feeding initiation after a PEG with jejunal extension (PEG-J or JET-PEG) or a direct percutaneous endoscopic jejunostomy (PEJ) has been placed. In the review series above, the same author indicates that TEN is also generally initiated within 6–24 hours after placement of PEG-J or PEJ tubes (32). In our institution, initiation of TEN via PEG-J or PEJ tubes begins upon arrival back to the floor (no waiting period).

SUMMARY

TEN is the preferred route for the provision of nutrition support. For TEN to be effective, it must actually be received and tolerated by the patient. When perceived problems arise, a stepwise approach that considers the anatomy and function of the GI tract is necessary to distinguish true intolerance from traditional practices and beliefs. Interventions that evaluate the underlying cause of the problem will provide long term solutions and allow adequate nutrition to be delivered to the patient. This article provides a review and update for practitioners involved in enteral feeding and offers suggestions to reassess TEN delivery practices in an effort to avoid some of the pitfalls encountered in the clinical setting. ■

References

1. Parrish CR, Krenitsky J, McCray S. *University of Virginia Health System Nutrition Support Traineeship Syllabus, 2003*. University of Virginia Medical Center Nutrition Services Department, Charlottesville, VA. Email: Linda Niven at ltn6m@virginia.edu for details.
2. Parrish CR. Enteral Feeding: The Art and the Science. *Nutr Clin Pract,* 2003;18(1):76-85.
3. Luckey A, et al. Mechanisms and treatment of postoperative ileus. *Arch Surg,* 2003; 138: 206-214.
4. Zaloga GP. Enteral nutrition in hospitalized patients: a consensus summary. In: *Enteral Nutrition Support for the 1990's: Innovations in Nutrition, Technology, and Techniques; Report of the Twelfth Ross Roundtable on Medical Issues*. Columbus, OH: Ross Laboratories; 1992, 47.
5. Heyland DK, Cook DJ, Winder B, et al: Do critically ill patients tolerate early, intragastric nutrition? *Clin Intensive Care,* 1996;7:68-73.
6. Charney P. Enteral Nutrition. Indications, options and formulations. In Gottschlich MM (ed). *The Science and Practice of Nutrition Support*. Kendal/Hunt Publishing: Dubuque IA; 2001, 141-166.
7. Miller LJ, Malagelada JR, Go VLW. Postprandial duodenal function in man. *Gut,* 1978; 19: 699-706.
8. Hecketsweiler P, Vidon N, Emonts P, et al. Absorption of elemental and complex nutritional solutions during a continuous jejunal perfusion in man. *Digestion,* 1979; 19: 213-217.
9. Zarling EJ, Parmar JR, Mobarhan S, et al. Effect of enteral formula infusion rate, osmolality, and chemical composition upon clinical tolerance and carbohydrate absorption in normal subjects. *J Parenter Enteral Nutr,* 1986;10:588-590.
10. Rees RPG, Keohane PP, Grimble GK, et al. Tolerance of elemental diet administered without starter regimen. *Br Med J,* 1985;290:1869-1870.
11. Rees RPG, Hare WR, Grimble GK, et al: Do patients with moderately impaired gastrointestinal function requiring enteral nutrition need a predigested nitrogen source? A prospective crossover controlled trial. *Gut,* 1992;33:877-881.
12. Taylor SJ, Fettes SB, Jewkes C, et al: Prospective, randomized, controlled trial to determine the effect of early enhanced enteral

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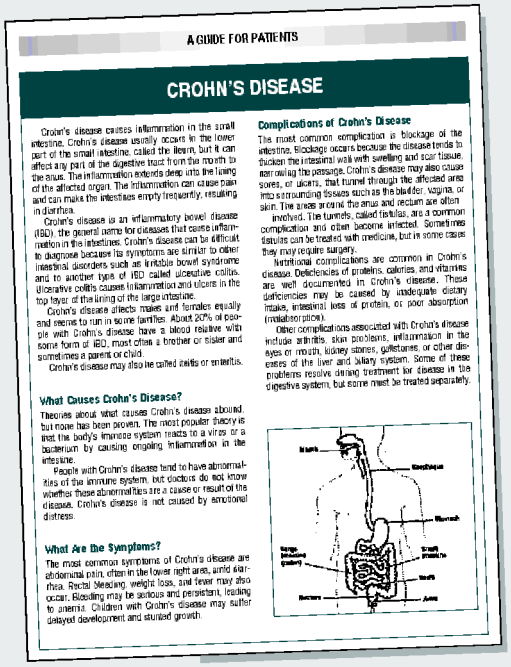
nutrition on clinical outcome in mechanically ventilated patients suffering head injury. *Crit Care Med,* 1999; 27: 2525-2531.
13. Heitkemper M, Hanson R, Hansen B: Effects of rate and volume of tube feeding in normal subjects. *Commun Nurs Res,* 1977;10:71-89.
14. Heitkemper ME, Martin DL, Hansen BC, et al: Rate and volume of intermittent enteral feeding. *J Parenter Enteral Nutr,* 1981;5:125-129.
15. McClave SA, Snider HL, Lowen CC, et al: Use of residual volume as a marker for enteral feeding intolerance: retrospective blinded comparison with physical examination and radiographic findings. *J Parenter Enteral Nutr,* 1992;16:99-105.
16. McClave SA: Clinical use of gastric residual volumes as a monitor for patients on enteral feeding. *J Parenter Enteral Nutr,* 2002;26:S43-50.
17. McClave SA, DeMeo MT, DeLegge MH, et al: North American Summit on Aspiration in the Critically Ill Patient: Consensus Statement. *J Parenter Enteral Nutr,* 2002; 26: S80-85.
18. Van Citters GW, Lin HC. The ileal brake: A fifteen-year progress report. *Curr Gastroenterol Rep,* 1999;1:404-409.
19. Heimberger DC, Sockwell DG, Geels WJ: Diarrhea with enteral feeding: prospective reappraisal of putative causes. *Nutrition,* 10:392-396.
20. Edes TE, Walk BE, Austin JL: Diarrhea in tube-fed patients: Feeding formula not necessarily the cause. *Am J Med,* 1990;88:91-93.
21. Shelly MS. Iatrogenic diarrhea caused by sorbitol. *J Family Pract,* 1993;36:95-96.
22. Keohane PP, Attrill H, Love M, et al: Relation between osmolality of diet and gastrointestinal side effects in enteral nutrition. *Br Med J,* 1984;288:678-680.
23. Heimburger DC, Geels WJ, Bilbrey J, et al: Small peptide and whole protein enteral feedings on serum proteins and diarrhea in critically ill patients: a randomized trial. *J Parenter Enteral Nutr,* 1997;21:162-167.
24. Bliss DZ, Johnson S, Savik K, et al: Acquisition of Clostridium difficile and Clostridium difficile-associated diarrhea in hospitalized patients receiving tube feeding. *Ann Intern Med,* 1998;129:1012-1019.
25. Borgstrom B, Dahlquist G, Lundh G, et al: Studies of intestinal digestion and absorption in the human. *J Clin Invest,* 1957; 36:1521-1536.
26. Raimundo AH, Rogers J, Filden P, et al: Influence of intraduodenal infusion of polymeric enteral diets on human pancreatic enzyme function. *Gastroenterology,* 1990; 98: 427 (Abstract).
27. Kandil HE, Opper FH, Switzer BR, et al: Marked resistance of normal subjects to tube feeding-induced diarrhea: The role of magnesium. *Am J Clin Nutr,* 1993;57:73-80.
28. Steinhardt HJ, Wolf A, Jokober B, et al: Nitrogen absorption in pancreatectomized patients: Protein versus protein hydrolysate as substrate. *J Clin Lab Med,* 1989;113:162-167.
29. McClave S, et al. When should enteral nutrition support be stopped prior to a procedure or diagnostic test? *JPEN,* 2001;25(1):S14.
30. Vanek VW. Ins and outs of enteral access: Part 2—Long term access—esophagostomy and gastrostomy. *NCP,* 2003;18:50-74.
31. Stein J, Schulte-Bockholt A, Sabin M, Keymling. A randomized prospective trial of immediate vs. next day feeding after percutaneous endoscopic gastrostomy in intensive care patients. *Intensive Care Med,* 2002:28:1656-1660.
32. Vanek VW. Ins and outs of enteral access. Part 3: Long term access—jejunostomy. *NCP,* 2003;18:201-220.

A GUIDE FOR PATIENTS

The more that a patient knows about his or her problem, the easier it is for the patient to cooperate with you and the more effective can be the prescribed treatment. Each "Guide" is on a different subject among the digestive diseases. You may cut out the "Guide" and photocopy as many reprints as you wish for distribution to your patients. You may want to include your name and address. The information in "A Guide for Patients" has been prepared by the National Digestive Diseases Information Clearing House, a service of the National Institute of Diabetes and Digestive and Kidney Diseases, National Institutes of Health, U.S. Public Health Service. The material it contains has been carefully reviewed by NDDIC for scientific accuracy and content.


A GUIDE FOR PATIENTS

CROHN'S DISEASE

Crohn's disease causes inflammation in the small intestine. Crohn's disease usually occurs in the lower part of the small intestine, called the ileum, but it can affect any part of the digestive tract from the mouth to the anus. The inflammation extends deep into the lining of the affected organ. The inflammation can cause pain and can make the intestines empty frequently, resulting in diarrhea.

Crohn's disease is an inflammatory bowel disease (IBD): the general name for diseases that cause inflammation in the intestines. Crohn's disease can be difficult to diagnose because its symptoms are similar to other intestinal disorders such as irritable bowel syndrome and to another type of IBD called ulcerative colitis. Ulcerative colitis causes inflammation and ulcers in the top layer of the lining of the large intestine.

Crohn's disease affects males and females equally and seems to run in some families. About 20% of people with Crohn's disease have a blood relative with some form of IBD, most often a brother or sister and sometimes a parent or child.

Crohn's disease may also be called ileitis or enteritis.

What Causes Crohn's Disease?

Theories about what causes Crohn's disease abound, but none has been proven. The most popular theory is that the body's immune system reacts to a virus or a bacterium by causing ongoing inflammation in the intestine.

People with Crohn's disease tend to have abnormalities of the immune system, but doctors do not know whether these abnormalities are a cause or result of the disease. Crohn's disease is not caused by emotional distress.

What Are the Symptoms?

The most common symptoms of Crohn's disease are abdominal pain, often in the lower right area, and diarrhea. Rectal bleeding, weight loss, and fever may also occur. Bleeding may be serious and persistent, leading to anemia. Children with Crohn's disease may suffer delayed development and stunted growth.

Complications of Crohn's Disease

The most common complication is blockage of the intestine. Blockage occurs because the disease tends to thicken the intestinal wall with swelling and scar tissue, narrowing the passage. Crohn's disease may also cause sores, or ulcers, that tunnel through the affected area into surrounding tissues such as the bladder, vagina, or skin. The areas around the anus and rectum are often involved. The tunnels, called fistulas, are a common complication and often become infected. Sometimes fistulas can be treated with medicine, but in some cases they may require surgery.

Nutritional complications are common in Crohn's disease. Deficiencies of proteins, calories, and vitamins are well documented in Crohn's disease. These deficiencies may be caused by inadequate dietary intake, intestinal loss of protein, or poor absorption (malabsorption).

Other complications associated with Crohn's disease include arthritis, skin problems, inflammation in the eyes or mouth, kidney stones, gallstones, or other diseases of the liver and biliary system. Some of these problems resolve during treatment for disease in the digestive system, but some must be treated separately.

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