

The Evolution of Feeding-Decompression: Automatic Monitoring and Control of Inflow

by Gerald Moss

“Feeding intolerance” frustrates attempts to enterally nourish patients with marginal gut function. Decompression catheters provide proximal aspiration, removing digestive juices and gas in an effort to minimize this iatrogenic complication. We now add automatic “checking for residual” every minute at the enteral feeding site.

Total inflow (feedings plus digestive secretions) is limited to exactly match impaired peristaltic outflow. All air and excess foodstuff are removed by the Moss[®]* tube, with return of the degassed aspirate.

The earliest stage of “feeding intolerance” is fluid accumulation at the enteral feeding site. Full feeding now can be initiated safely in the face of impaired GI function. The potential excess can be removed before distention and vagal reflexes further disrupt the sluggish gut.

Adding monitoring and refeeding to the current feeding-decompression regimens results in earlier enteral nourishment for patients with critically impaired gut function. Their feeding goals can be safely approached and achieved immediately postoperatively.

INTRODUCTION

The first device to efficiently aspirate swallowed air while feeding distally was introduced in 1962 (1). The latest improvement to this regimen is automatic matching of enteral inflow to monitored peristaltic outflow, with return of the degassed aspirate via the feeding channel.

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* Dr. Moss is not affiliated with Moss Tubes, Inc., owner of the Moss[®] trademark.

The guiding principles behind each improvement have always been:

1. Swallowed air is critical to the development of paralytic ileus, and gas is most efficiently intercepted from within a constricted zone of the GI tract; and
2. Stasis at the feeding site triggers the symptom complex of “feeding intolerance.”

The initial aspiration site was the distal esophagus (Figure 1) (2). Trapping and venting air from this constricted site was 12–14× more efficient than gastric aspiration (3).

A separate channel of that device fed into the stomach, necessitating frequent “checks for residual”

to avoid overfeeding and distention. This gastric aspirate was returned manually. We invariably noted that foodstuff spontaneously moved prograde by peristalsis was unimpeded in its digestion and absorption farther downstream.

An unintended consequence of esophageal aspiration was interception of undiluted, swallowed broncho-tracheal secretions, requiring continuous irrigation to maintain patency of the suction lumen. In practice, this dedicated nursing attention could only be obtained at our NIH funded Clinical Research Center (CRC) at tax-payers' expense.

Such diligence was rewarded by total air exclusion, with "degassing" of the gut. The abdomen became scaphoid, and bowel sounds remained absent despite uneventful gastric feeding. Normal bowel sounds returned promptly upon resumption of oral intake.

Postoperative ileus was aborted, and positive nitrogen balances achieved within hours of major resective surgery (1). Serum branched chain amino acids had returned to (and exceeded) basal concentrations within two-to-four hours (4,5). Parallel canine studies showed that the altered metabolic responses included accelerated wound healing and enhanced immune competence (6,7).

The earliest nasogastric device was hand-made, huge (28Fr), and the requisite herculean care precluded wider clinical application. Over the next two decades, manufacturing technology allowed reductions in size to 18 French, and both the aspiration and feeding sites progressively were moved downstream. The aspirate volume markedly increased as the phlegm and other potentially obstructing substances became more dilute, reducing (but not eliminating) the risk of clogging the suction channel. The excess feedings were automatically removed (8).

The current (since 1985) design of the Moss® tube (both the gastrostomy and nasal versions), has the aspiration and delivery channels terminate within the duodenum. Elemental diet is delivered a short distance beyond the aspiration orifices, with little impediment to flow between them (Figure 2). Any excess feeding refluxes freely within the duodenum, to be removed slightly more proximally, and without producing distention or triggering vagal reflexes.

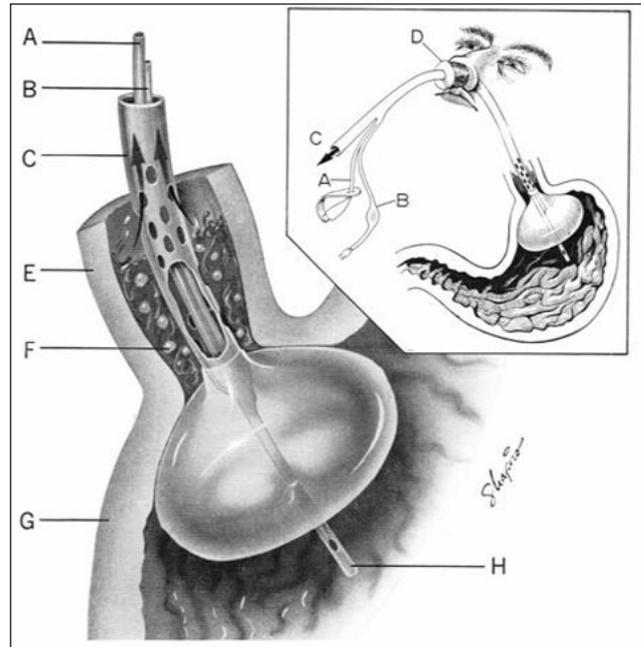


Figure 1. Feed decompression tube. **A**, feeding tube; **B**, balloon inflation tube; **C**, esophageal suction jacket; **D**, sponge retainer; **E**, esophagus; **F**, swallowed air and fluid in esophagus; **G**, stomach; **H**, feeding tube. Progenitor Moss® Tube (circa 1961) Reprinted with permission of the American College of Surgeons. Moss G. Postoperative decompression and feeding. *Surg Gyn Obs*, 1966;122:550-5540.

Approximately 3,000–4,000 mL/day of fluid (saliva, bile, succus, gastric, and pancreatic juices, plus excess feedings) are removed from the proximal duodenum, comparable to a high output duodenal fistula. It should also be noted that these contain secretory globulins that are specific for the patient's gut organisms. It was recommended that this aspirate be returned manually via the feeding channel (8). However, refeeding aspirate was messy, time consuming, and rarely performed outside of a research environment.

The earliest maximum enteral nutrition was tolerated, despite marginal GI activity, with uneventful delivery of 2,000–5,000 kcal of elemental diet the initial 24 hours postoperatively. Gut function rapidly improved, and aspirate became nutrient-free within one-to-two hours of major surgery. By 1987, ~300 kcal/hour of elemental diet was tolerated, for absorption of \$3,000 kcal during the initial postoperative 24 hours.

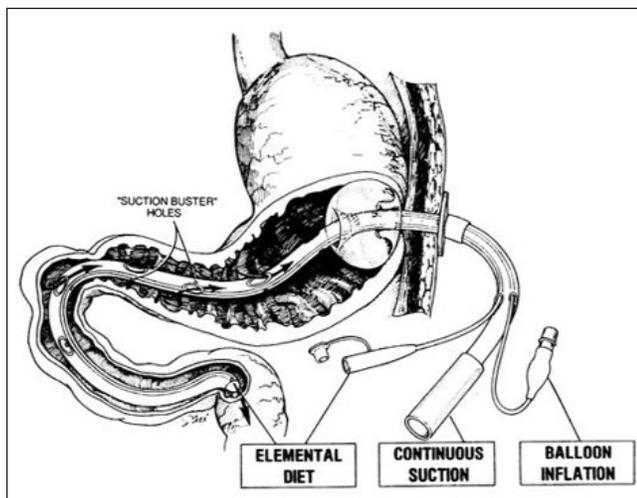


Figure 2. Current Moss Tube (circa 1985) Reprinted with permission from the American Society for Parenteral and Enteral Nutrition. Moss G. Efficient gastroduodenal decompression with simultaneous full enteral nutrition: a new gastrostomy catheter technique. *J Paren Enter Nutrit*, 1984;8:203–207.

We added routine, liberal use of bupivacaine at the time of wound closure. With gut function restored and exploited within hours, the patient became independent of the hospital much quicker.

Postoperative length of stay was reduced to 24 hours for 160 consecutive “open” cholecystectomy patients by 1987, prior to the introduction of laparoscopic surgery. They all declined available narcotics (9). Discharge could be 24–48 hours after conventional colectomy and re-anastomosis, with X-ray confirmation of clinically normal gastro-intestinal motility by five hours postoperatively. Patients routinely had spontaneous bowel movements within 24 hours (10,11).

An automatic device was introduced recently to improve this regimen. Peristaltic outflow at the feeding site is monitored every minute, relative to total inflow (feedings plus secretions). The degassed aspirate is returned. Only potentially accumulating excess is removed immediately, before local distention and vagal reflexes can initiate the symptom complex of “feeding intolerance.”

METHODS

The “Monitor-Manager” has previously been described (12). The basic disposable unit consists of a 35 mL plas-

tic chamber, with connecting tubing and an adjustable over-flow initially set at 20–30 mL. A source of programmed, intermittent suction alternately applies and releases vacuum for 30 seconds every minute. One-way valves appropriately direct flow of aspirate into the chamber, and then back to the patient.

A floating ball check valve allows free flow of filtered aspirate into the chamber. When suction to the chamber terminates, a one-way valve allows gravity return of the degassed aspirate via the feeding channel. The floating ball seats to prevent air from following.

If peristaltic activity is adequate to propel all the feedings (plus juices) during this 30 second interval, residual volume is scant, and the rising fluid column fails to reach the overflow level. Only gas bubbles are vented.

If total inflow to the feeding site exceeds peristaltic outflow, the level in the chamber rises to reach the overflow level, and exits into the canister. Only these small volumes are immediately and permanently removed (Figure 3).

DISCUSSION

The physiological disruption of “feeding intolerance” results when nonvolitional nutrition, even minimally, exceeds the patient’s ability to propel and absorb the feedings. Excess foodstuff accumulates at the enteral feeding site in a stagnant pool of pancreatic enzymes. Hydrolysis is followed by imbibition, and the residual volume inexorably increases to maintain iso-osmolality.

Local distention triggers vagal reflexes, which further slow the already sluggish gut. This initiates a vicious cycle of increasing stagnation. Gastrointestinal recovery stalls and reverses, with worsening abdominal distention and general malaise. The patient’s mobility and respiratory mechanics are compromised. A less frequent but lethal complication of inadvertent “overfeeding” is bowel necrosis, with a reported 1:1,000 incidence (13). This probably is also related to another potential of vagal reflexes, that lead to cardio-vascular disruption of splanchnic circulation in susceptible patients.

Miedema, et al at the University of Missouri, studied postoperative patients fed by J-tube who developed “feeding intolerance”(14). This complication was characterized by increased intra-luminal tension by

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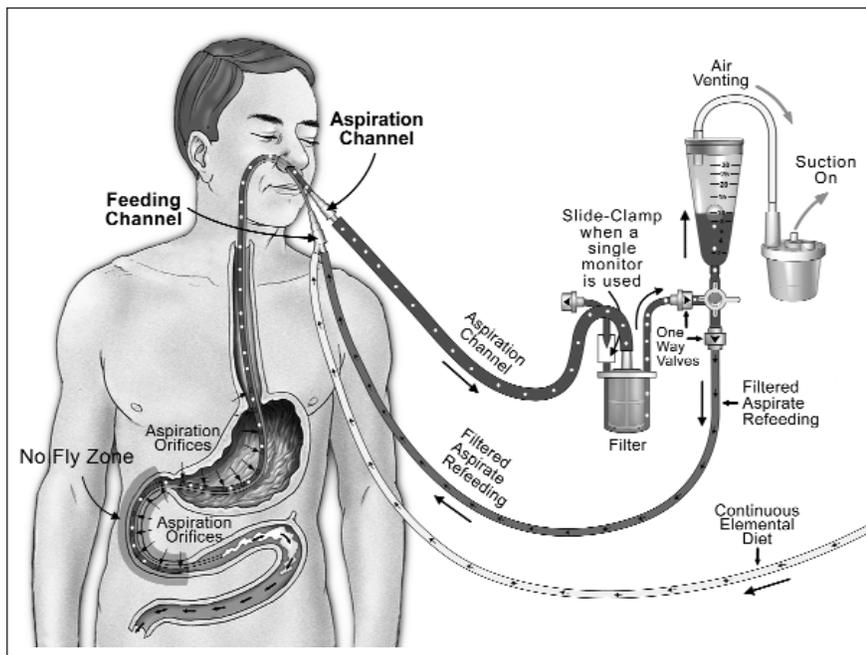


Figure 3. Monitor-Manager Regimen

jejunal manometry. However, pressure elevation developed late, and manometry was inadequate to guide timely clinical intervention.

The jejunum is normally flaccid, virtually empty, and contains volume receptors that respond to minimum local accumulation. (In our experience, the monitored segment usually contained <10 mL of fluid.) Vagal reflexes can initiate a cycle of slowing, to the point of complete paralysis, even before the pressure rise can be recognized externally.

“Clinical judgment” alone has proven inadequate to optimally and safely feed immediately postoperative patients. The physician is faced with a clinical dilemma. Those with marginal bowel function usually are most in need of immediate nutrition. Yet they risk severe respiratory and other complications from inadvertent “overfeeding.”

Prospective controlled clinical studies have failed to demonstrate benefit from the usual regimens of early enteral postoperative feeding, when guided by clinical judgment alone. Physicians at Memorial Sloan Kettering Cancer Center studied 100 paired abdominal cancer patients (15). Half received jejunal feeding following resective surgery, and their recovery was impaired, not

improved. One of their fed patient developed bowel necrosis. They concluded that immediately postoperative enteral feeding was contraindicated.

An Ottawa group had a similar study, with similar dire results (16). However, they also measured postoperative physical activity and pulmonary function, to understand the mechanism by which enteral feeding exerted its detrimental effect.

These functions were impaired for all postoperative patients. However, the fed patients had more severely compromised mobility and respiratory mechanics, which could be attributed to their greater degree of abdominal distention.

Aerophagia plays a major role in the development of abdominal distention and paralytic ileus. Wangenstein and Rea studied experimental small bowel obstruction, transecting and oversewing the terminal ileum (17). Even this extreme challenge could be tolerated indefinitely, if they simultaneously vented all swallowed air from a cervical esophagostomy. The dogs could be adequately hydrated by clysis at that time (but not nourished), and they eventually died of starvation. The longest survivor died without distention, two months after the initiation of total distal intestinal obstruction.

The physician should be forewarned that immediate absorption of full nutrition markedly alters patient metabolism, albeit beneficially. The feeding serves as a “glucose tolerance test,” superimposed on the acute stress of surgery.

The main constituent of an elemental diet is carbohydrate. Immediate feeding after experimental bowel resection caused glucose oxidation to double, and plasma insulin levels to triple, relative to the responses of unfed canine controls (18). We reported a similar response by otherwise healthy, young cholecystectomy patients, with markedly elevated plasma insulin levels but without glycosuria.

Older (i.e., >35 years of age), non-diabetic patients usually required titration with 5-20 “units” of supple-

mental insulin in response to glycosuria. Diabetics that were regularly controlled with ~30 units of insulin/day, initially required ~300 units of insulin. All patients reverted to their pre-operative glucose metabolic patterns within 24 hours after cholecystectomy or colectomy. This short-term postoperative approach to glucose control parallels similar advances in the Intensive Care Unit, where glucose monitoring and supplemental insulin titration have become standard.

Addition of this monitor-manager regimen, per se, is intrinsically safe. Nothing is delivered to the patient in net fashion. A small volume of aspirate is returned under gravity to essentially the same location from which it had been aspirated seconds earlier. Only air bubbles and incipient accumulating excess liquid are vented.

Our current results have duplicated the patterns achieved with manual "refeeding" over 40 years earlier, but with minimum added workload. Full enteral nutrition has been initiated within minutes of major operation, avoiding "feeding intolerance" and with scant wastage of body fluids.

Digestive juices contain secretory globulins, specific for the patient's own enteric organisms. Future studies will show whether routinely returning the previously discarded aspirate protects against nosocomial infections following operations, or reduces length of hospital stay and cost. This approach may offer an alternative to prophylactic antibiotics, with concomitant detrimental effects on emergence of resistant bacteria.

The current feeding-decompression designs (circa 1985) still appear to be useful, and should not be discarded. As with all enteral devices, the aspiration channel is prone to clogging with particulates and/or mucus. Adding the monitor-manager regimen does not increase the nursing workload to maintain patency, but it does promptly identify and focus attention on this problem. The time and effort required to maintain patency are well rewarded by improved patient outcome.

CONCLUSION

Postoperative paralytic ileus is an avoidable complication (19). Both the swallowed air and excess feedings can be removed by the current feeding-decompression catheters alone, but with wastage of large volumes of

digestive secretions. The latest advance is a practical, automatic regimen to provide optimum nutrition within minutes of surgery, without discarding enteric fluids. Overfeeding is avoided safely by automatically limiting inflow to match monitored peristaltic outflow. ■

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