The intestinal factor in multiple organ failure and shock

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AUTOINFECTION with common enteric microorganisms is now recognized to be a leading cause of late morbidity and mortality after trauma, critical surgical illnesses, and shock. Indeed, bacterial translocation from the gut—a process whereby indigenous intestinal microflora relocate extraluminally—may be responsible for the multiple organ failure syndrome. There is a number of factors that could promote bacterial translocation in critically ill patients. Reduction in mesenteric blood flow greatly diminishes the efficiency of the intestinal mucosal barrier; total parenteral nutrition (TPN) promotes bacterial translocation from the gut; in rats TPN causes a marked reduction in the amount of intestinal secretory immunoglobulin A, the principal defense against attachment of intestinal bacteria to mucosal cells. In addition, replacement of enteral feeding by TPN causes a substantial reduction in size of the functional intestinal mucosa, which is largely dependent on the enteral route for its own nutrition and trophism. Malnutrition leads to an absolute decrease in intestinal mucin. Impaired capacity to maintain mucosal mucin content may be a factor in reducing intestinal resistance to enteric infection and pancreatic proteases. The possible concurrence of reduced intestinal blood flow and TPN in the critically ill could thus enhance the role of the “intestinal factor” in the pathogenesis of multiple organ failure and the ultimate mortality in the intensive care unit (ICU).

Antemortem necrosis of the epithelium and the tip of the intestinal villi with gastroduodenal erosions has been described for some time in dogs when arterial blood pressure is lowered to 35 to 40 mm Hg for 3 to 4 hours. Similarly, ischemic necrosis of the intestinal mucosa with gastroduodenal erosions has been recognized at autopsy in patients who died after congestive heart failure, burn, hemorrhage, and sepsis. Endoscopic examination has enabled physicians to observe the occurrence of gastroduodenal erosions within 5 hours of injury in more than 80% of patients with severe burn. Experimental evidence has shown that early responses to ischemia, enterocyte membrane disruption and pancreatic elastase interference with brush-border protective glycoproteins, expose the underlying intracellular structures to the digestive action of trypsin.

The use of an elemental diet in the prophylaxis of experimental ischemic enteropathy and shock was conceived with the object to reduce, by dietary means, the concentration of potentially noxious physiologic constituents of the intestinal chyme before shock. Indeed, the prefeeding of an elemental diet, containing most nutrients in their simple molecular form, has been found to minimize the gastrointestinal lesions in laboratory animals subjected to severe hypovolemia or burn, and, in accordance with the role of the “intestinal factor” in the pathogenesis of shock, survival has been improved. This type of protection is mostly prophylactic because a majority of the control animals die in shock within a few hours. In addition, better use of predigested nutrients after postischemic drop in brush-border digestive enzymes may favor the recovery of survivors.

In the clinical situation the degree of ischemia-anoxia—hence the time-sequence and severity of morbid events in the gastrointestinal mucosa—may vary over a wide range. Obviously, the potential benefit of feeding an elemental diet enterally in ICU patients will greatly depend on proper timing. When hemorrhagic necrosis has developed in the intestinal mucosa, the systemic adverse effect of the intestinal factor, such as multiple organ failure, could hardly be reversed by a treatment that is by definition designed to prevent the onset and the evolution of ischemic enteropathy. This probably is the reason that when elemental feeding was initiated 4 to 6
days after diagnosis of sepsis and hypermetabolism, it had no effect on the incidence of multiple organ failure and mortality. In all situations it is important to recognize that the feeding of hypertonic solutions into the small intestine may severely damage fragile intestinal epithelium. Due attention must be taken to ensure that the feeding solution be isotonic, particularly in the initial phase of treatment. Proper monitoring of diet osmolarity might have prevented the occurrence of osmotic diarrhea reported in one study.

Recent advances in low-risk infusion delivery systems designed for continuous enteral alimentation have enhanced the use of elemental diets in a low-cost nutritional management of the critically ill. In addition, proper timing and monitoring of this diet may provide an interesting new prophylactic measure against the development of intestinal complications, multiple organ failure, and shock.

REFERENCES