Does GI physiology support the rationale behind switching from oral rehydration solution to intravenous saline preparations in the management of children with acute severe diarrhea?

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While I was on my recent pediatrics rotation at medical school, I must have seen about 20 children present to the emergency department with the manifestations of dehydration owing to acute diarrhea. In line with World Health Organization guidelines, these children’s dehydration status was categorized into three grades: none, mild, and severe, corresponding to dehydration <3%, between 3% and 8%, and >8% of body weight, respectively. It is now well established that the best treatment for the majority of dehydration, especially in the mild category, is to give hypoosmolar oral rehydration solution (5). This is based on elegant gastrointestinal (GI) physiology; the sodium-glucose cotransporter allows for the passive uptake of water in a manner that is dependent on ATP (1). It is not interfered with by the presence of bacterial enterotoxins. Importantly, such treatment is favored over intravenous rehydration, since the former has been clearly shown to effect faster rehydration with fewer complications, thereby reducing morbidity (3). It is also less invasive, an important consideration when managing children.

However, severe dehydration requires intravenous rehydration with saline solutions rather than oral treatments. Why is this? Published guidelines seldom entertain reasons behind their recommendations. Nevertheless, GI physiology provides a plausible explanation for the current medical practice. Fluid absorption from the gut to the interstitium, and from there to the splanchnic circulation, depends on several interactive factors (4). Such factors principally include those related to hemodynamics (e.g., Starling forces) and epithelial cell function (e.g., the activity of surface-membrane active transporters). There is also an interaction between epithelial transport and hemodynamic factors (e.g., paracellular permeability), and there are probably numerous regulatory effects secondary to severe dehydration (e.g., via hormones and the enteric nervous system).

In severe hypovolemia, maximal sympathetic arteriolar vasoconstriction can reduce splanchnic blood flow from 1,500 ml/min to as little as 300 ml/min (6). Such vasoconstriction would reduce capillary hydrostatic pressure. This adaptive circulatory effect helps to preserve perfusion to vital organs, such as the brain and heart, at the expense of other organs, such as the gut and skin.

Intuitively, one might speculate that such a decrease in splanchnic blood flow in severe hypovolemia should cause a resultant decrease in the total amount of enteral fluid absorption. However, it has been shown, in an experimental animal model, that during severe hypovolemia, sodium reabsorption from the ileum increases significantly in a manner probably involving an active ion transport mechanism (2). However, the blood flow to the experimental section of the gut is unknown, and thus there is uncertainty about the extent of hypovolemia that was experienced by the experimental tissue itself. The study is therefore difficult to interpret, since it is well established that the intestinal villi exhibit an oxygen countercurrent exchange mechanism that leads to relative hypoxia of the tip compared with its base (9). It has been shown that small intestinal fluid absorption is impaired in the face of mucosal damage secondary to severe hypovolemia (7). Gut ischemia could limit the activity of secondary active transporters, and, in the study, the extent of gut ischemia was not conclusively known. In addition to the sympathetic nervous system’s adaptive effect on the splanchnic vasculature, this effect would be potentiated by circulating vasoconstrictor hormones, such as angiotensin II and vasopressin, which are present in high concentrations in severe hypovolemia (8).

Clinically, therefore, the predicted consequence of the adaptive effect of disproportionate redistribution of the blood supply to vital organs is that the reduced GI perfusion leads to poor systemic absorption of electrolytes and water. Intravenous rehydration, instead, allows for direct resuscitation of the intravascular compartment. This may be a reason why intravenous rehydration treatment for acute diarrhea should be used only under exceptional circumstances or when the gut sodium-glucose cotransport mechanism is likely to be ineffective, owing to the aforementioned GI ischemia. Future research should be targeted at rigorously determining the evidence behind current clinical practice.

REFERENCES

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