Gastrointestinal Fluid Resuscitation of Thermally Injured Patients

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A seminal advance in the care of thermally injured patients was the recognition of the large volumes of fluid required during resuscitation and the relationship of these volumes to burn size and body weight. This discovery resulted in the virtual elimination of acute renal failure as a consequence of burn shock.1,2 Today, the established standard of care focuses on the intravenous route for fluid resuscitation,3 and time to intravenous access was an independent predictor of mortality in a study of children with massive burns by Wolf et al.4 Historically, however, several other methods have been used for delivery of fluids to patients with burn shock, including oral, rectal, and subdermal routes. Several factors led to the preferential use of the intravenous route. It enables the immediate delivery of known quantities of fluid and allows the rapid correction of circulating volume deficits. By contrast, the other routes provide uncertain amounts of fluid at more gradual rates, and vomiting may complicate oral fluid intake. Finally, plasma or albumin solutions can only be given intravenously.

Despite the advantages of intravenous therapy, an increasing focus on preparedness for mass-casualty and austere scenarios has caused us to reexamine the utility of oral, intestinal, and rectal resuscitation of thermally injured patients.5 The detonation of a single nuclear weapon in an urban area would likely generate a large number of thermally injured survivors. The likelihood of there being survivors with burns increases with increasing weapon yield, thus, “burn injury is the most difficult problem to be faced by the military medical community in a nuclear conflict.”6 During the first part of Operation Iraqi Freedom in 2003, the U.S. Army Institute of Surgical Research in collaboration with the American Burn Association and the National Disaster Medical System conducted a daily inventory of burn beds in the United States. This research demonstrated that the number of available burn beds in the United States would be insufficient to handle the number of casualties that would be produced by a single nuclear weapon.7 Even a conventional disaster can generate enough burn casualties to overwhelm the medical system.8 We would speculate that such might have been the case had the World Trade Center towers not collapsed on September 11, 2001.9 Under circumstances such as these, alternatives to physician-directed, intensive care unit-based, intravenous fluid resuscitation of burn patients will be needed.

Concerns regarding potential combat casualties caused by nuclear weapons in the Korean War motivated an examination of oral fluids for burn shock by the 1950 Surgery Study Section of the National Institutes of Health (NIH). Although recognizing the need for further research, the NIH concluded “that the use of oral saline solution be adopted as standard procedure in the treatment of shock due to burns and other serious injuries in the event of large-scale civilian catastrophe.”10 Fifty-six years after the NIH report, significant gaps remain in our knowledge about the ability of the gastrointestinal tract to absorb sufficient quantities of fluid during burn shock.

In addition, Special Operations medics operating under austere conditions may not have access to sufficient volumes of sterile intravenous fluids to permit
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burn resuscitation. Civilian health care providers working in the third world likewise often do not have access to fluids and cannulas. For burn resuscitation in these settings, we have suggested the use of a homemade oral rehydration fluid similar to that recommended for the treatment of diarrhea, which is discussed herein.11,12 How best to perform oral resuscitation under austere conditions remains to be determined. Therefore, the purpose of this article is to review the history and current status of gastrointestinal resuscitation and to suggest clinical guidelines for use.

BACKGROUND

Before the widespread introduction of intravenous cannulas, nonintravenous routes were used more frequently for burn shock resuscitation. In one of the first published descriptions of successful burn care, Haldor Sneve of St. Paul, Minnesota, wrote in 1905:

To assist nature to drive the blood out from the abdomen, give drinks and enemata of cold normal saline solution which add volume to the circulating medium . . . . To counteract the fall in body temperature, use the hot bath and maintain a high temperature in the room. Finally, in order to add more fluid to supply the heart with something to work on use saline transfusion or hypodermoclysis.13

Despite these observations, little progress in this field occurred until Frank Underhill’s report on the Rialto Theater fire of 1921. In this article, the author recognized hemoconcentration (anhydremia), most likely caused by the loss of “fluid of the nature of plasma” from the intravascular space, as a fundamental mechanism of burn shock. Again, he recommended intravenous, rectal, oral, and/or hypodermal resuscitation with saline solution.14 During the first part of World War II, in Great Britain, “shock had been mainly combated by keeping the patients warm and giving them large quantities of fluids orally.”15 At Pearl Harbor, oral fluids in addition to intravenous saline, glucose, plasma, and albumin were used.16 Care for the survivors of the Cocoanut Grove fire of 1942 featured a shift to the use of intravenous plasma as the primary resuscitation fluid.17 Still, it is worth recalling that the landmark article by Reiss et al,1 which described the original Brooke formula for intravenous fluid resuscitation with colloid in 1953, also provided guidance for oral resuscitation:

A well-cooled solution containing 3 gm of sodium chloride and 1.5 gm of sodium bicarbonate per liter can be administered orally and is well tolerated by most patients . . . . In general, burns that involve less than 20% of the body surface in adults require only a minimum of supportive therapy. Colloid solutions are not necessary, and fluid requirements are satisfactorily met by electrolyte solutions given orally and/or intravenously.1

ORAL BURN RESUSCITATION: CLINICAL REPORTS

After World War II, the enteral route for burn shock resuscitation was deemphasized. In a recent review, Kramer et al49 identified 12 reports on oral resuscitation in burn patients that, although largely anecdotal, provide useful information on the type of fluids used and potential risks and benefits of the approach (Table 1). In 1941, Charles Fox reported oral resuscitation of nine severely injured patients. Four children (TBSA 23–80%) and five adults (19–41%) with full-thickness burns and signs of shock were treated with chilled isotonic sodium lactate, for a total of 100 to 150 ml/kg in 24 hours.18–20 Vomiting was treated by administration of more fluid; in these patients, nasogastric tubes were used to provide continuous fluid delivery. Fluid infusions were adjusted to obtain 1 to 2 liters of urine per day.

In 1949, Moyer reported oral resuscitation of more than 30 severely burned children and adults.21 He found that NaCl alone caused acidosis, which could be prevented with bicarbonate, lactate, or citrate. These buffered saline solutions also were more palatable. He was careful to state that when shock was present, it was necessary to administer intravenous fluid with lactated Ringer’s or plasma until the peripheral collapse was corrected, after which oral resuscitation could be initiated.

In 1956, Markley reported the results of a trial conducted in Peru that evaluated the use of oral resuscitation.22 Treatment was alternated for successive patients between oral isotonic bicarbonated saline and intravenous therapy with plasma and saline. The mean burn size was approximately 40% and 30% in 55 children and 56 adults, respectively. The mean volume of oral saline was 110 ml/kg in the first 24 hours. Mortality at 48 hours was equivalent in the two groups, whereas hematocrit and urine output data suggested effective volume expansion with oral resuscitation. However, urine output and mortality data suggested an inability to fully resuscitate patients with burns greater than 50% TBSA by the oral route.

In 1960, Wilson and Stirman23 reported 142 patients with 15% to 60% TBSA injuries treated with isotonic saline compared with saline plus blood. The mortality rate was greater in the group that received blood. When oral treatment seemed inadequate to maintain the circulation or impractical because of vomiting, patients were administered intravenous lactated Ringer’s solution or plasma until stabilized.
However, 81 of Wilson’s patients were treated exclusively with oral intake, including some patients with a 60% TBSA injury.

Davies reported in 1964 for the British Medical Research Council on the increased use of intravenous plasma for burns that was made available by Britain’s National Blood Transfusion Service. Patients with burns of 40% TBSA or less were resuscitated initially with oral Moyer’s solution administered at two times the normal oral fluid input, independent of injury size. Half of these patients required subsequent intravenous saline or saline plus plasma, which was started when vomiting was excessive or clinical condition deteriorated.

Franke and Kock reported in 1964 on the first clinical use of oral electrolyte solution that also contained glucose for treatment of severe burn injury in children. This group used gastric infusion and an antiemetic to treat 19 of 22 children with 8% to 38% TBSA injuries. Three additional children with larger burns of 35% to 70% received intravenous fluid and gastric infusions, and two of these died from cerebral edema and heart failure.

In 1966, Jackson reported 162 burn cases (113 children and 49 adults) with 10% to 35% TBSA injuries. A total of 75% of these were exclusively treated with oral fluids only, 25% also received IV plasma or LR. Vomiting occurred in 36% of patients.

Table 1. Enteral resuscitation of burn injury: the clinical record

<table>
<thead>
<tr>
<th>First author, date</th>
<th>n</th>
<th>TBSA</th>
<th>Solution</th>
<th>Results/Comments</th>
</tr>
</thead>
<tbody>
<tr>
<td>Fox, 1944</td>
<td>9</td>
<td>19–80%</td>
<td>Isotonic sodium lactate</td>
<td>Less vomiting with sodium lactate than other sodium salts. Hypochloremic on day 2</td>
</tr>
<tr>
<td>Moyer, 1949</td>
<td>30</td>
<td>Not listed</td>
<td>Hypotonic citrated or bicarbonated NaCl</td>
<td>Hypotonic solutions reported to have low incidence of nausea and vomiting. Used intravenous lactated Ringer’s (IV LR) and oral buffered saline</td>
</tr>
<tr>
<td>Markley, 1956</td>
<td>111</td>
<td>&gt;10%</td>
<td>Isotonic bicarbonated NaCl</td>
<td>Vomiting less in oral group, 55% of cases used oral fluids only, rest received some IV LR or plasma</td>
</tr>
<tr>
<td>Wilson, 1960</td>
<td>142</td>
<td>15–65%</td>
<td>0.9% NaCl</td>
<td>Patients denied oral resuscitation if peripheral vascular collapse, vomiting, or gastric dilation</td>
</tr>
<tr>
<td>Davies, 1964</td>
<td>20</td>
<td>10–40%</td>
<td>Moyer’s solution bicarbonated NaCl</td>
<td>Always started with oral resuscitation, but about half of the patients required IV LR because of vomiting or deteriorating shock</td>
</tr>
<tr>
<td>Franke, 1964</td>
<td>22</td>
<td>8–70%</td>
<td>Glucose-HCO$_3$ electrolytes</td>
<td>Used gastric infusion and antiemetics; 19 (86%) received only enteral fluids</td>
</tr>
<tr>
<td>Sorensen, 1965</td>
<td>26</td>
<td>10–45%</td>
<td>Clear fluids and salt tablets</td>
<td>Administered water (100 to 200 ml/kg first 24 hr) and 7.5-g salt tablet per luter</td>
</tr>
<tr>
<td>Jackson, 1966</td>
<td>162</td>
<td>10–35%</td>
<td>Moyer’s solution bicarbonated NaCl</td>
<td>A total of 75% treated with oral fluids only, 25% also received IV plasma or LR. Vomiting occurred in 36% of patients</td>
</tr>
<tr>
<td>Monafo, 1970</td>
<td>7</td>
<td>22–95%</td>
<td>Hypertonic lactated saline-600 mOsm</td>
<td>Combined oral and IV treatment with hypertonic lactated saline</td>
</tr>
<tr>
<td>Ahnfeld, 1975</td>
<td>68</td>
<td>12–34%</td>
<td>Slightly hypertonic glucose electrolyte solution</td>
<td>Tolerance of gastric infusion of 1 liter was 97% of patients in first hour after burn but only 30% at 1.5 to 3 hrs after burn</td>
</tr>
<tr>
<td>Maksimov, 1989</td>
<td>92</td>
<td>10–50%</td>
<td>Isotonic bicarbonated NaCl</td>
<td>Exclusively in 12 patients with moderate burns and to supplement IV therapy in 80 patients</td>
</tr>
<tr>
<td>El-Sonbaty, 1991</td>
<td>20</td>
<td>10–20%</td>
<td>WHO ORS</td>
<td>Control group with LR–Parkland formula had equivalent outcomes</td>
</tr>
</tbody>
</table>

WHO ORT, World Health Organization oral rehydration therapy.

Monafo and Moyer et al advocated crystalloid and colloid solutions, formulas for intravenous fluid resuscitation provided patients required no intravenous support. Eighty percent of these patients received 10% to 60% lactate, and 100 mEq/l of chloride, given orally and lactated saline (300 mEq/l of sodium, 200 mEq/l of potassium, and/or water. To avoid hyponatremia, he administered 7.5-g salt tablets (5 g of NaCl and 2.5 g of NaHCO₃) per liter of fluid. Fluid intake totaled 150 to 200 ml/kg in first 24 hours and 100 to 150 ml/kg in the second 24 hours. Eighty percent of these patients required no intravenous support.

Whereas the Cope and Moore, Evans, and Brooke formulas for intravenous fluid resuscitation provided a combination of colloid and crystalloid solutions, Monafo and Moyer et al advocated crystalloid solutions and ascribed burn shock to extracellular sodium deficiency rather than to plasma volume losses. Thus, oral resuscitation with saline solutions remained part of their armamentarium. This concept of sodium deficiency then led to the development of hypertonic saline resuscitation. In the original article on this topic, Monafo described use of hypertonic lactated saline (300 mEq/l of sodium, 200 mEq/l of lactate, and 100 mEq/l of chloride), given orally and intravenously. Seven patients received 10% to 60% of their fluid needs in the first 24 hours from oral fluids and 60% to 90% of their fluid needs in the second 24 hours.

In 1975, Ahnefeld et al reported on the use of Liquidsorb (J. Pfrimmer) for the treatment of 68 burn patients with 12% to 34% TBSA injuries. Liquidsorb, which has been described as a slightly hypertonic 370 mOsm electrolyte "lemonade," contains 4 g of glucose, 100 mg of ascorbic acid, and 12 mg of nicotinamide per liter of bicarbonated saline. Although only 4 of 58 patients who arrived at the hospital within 1 hour of injury could not drink the solution, 16 of 23 patients who arrived more than 2 hours after their injuries were not able to drink because of "sickness, vomiting, centralization of the circulation, and unconsciousness." Therefore, oral resuscitation must be started early, ideally within the first hour after injury. Ten subsequent burn patients with 12% to 26% injuries also were treated with Liquidsorb and, after receiving it via gastric tube in 50-ml increments, demonstrated good absorption of 1 liter in the first hour and 750 ml in the second hour. This approach was well tolerated, except for patients who had evidence of shock.

El-Sonbaty in Egypt reported oral resuscitation of 20 children with 10% to 20% TBSA burns using the World Health Organization’s (WHO’s) oral rehydration therapy (ORT) solution. The volume and rate of oral hydration was identical to standard Parkland formula for intravenous administration. El-Sonbaty reported oral hydration to be as effective as intravenous lactated Ringer’s, which also was administered at Parkland rate.

**ORAL TREATMENT OF SEVERE DIARRHEA**

Data on oral burn resuscitation are limited and come largely from an older literature. However, a substantial body of literature exists on the use of ORT for the treatment of severe diarrhea secondary to cholera and other diseases. In much of the underdeveloped world, the intravenous route is considered the standard of care for fluid resuscitation for only the most severely affected patients. In addition, in most places afflicted with cholera, cannulas and sterile solutions are in short supply or are unavailable, and families often deliver the medical care. This shortage led to development of ORT solutions made from clean water, sodium, chloride, potassium, base (bicarbonate or citrate), and glucose.

In 1975, the WHO ORT solution became widely available (Table 1). ORT was the principal intervention responsible for reducing annual deaths worldwide from diarrhea in children younger than 5 years of age from 5 million to approximately 2 million. Although simple, ORT “was heralded as one of the great medical achievements of the 20th century.” The failure of ORT (requiring intravenous rehydration) is unusual, occurring in approximately 4% of children with diarrhea. In 2003, a new WHO ORT solution, with reduced osmolarity, replaced the 1975 formula. This change was motivated by the observation that the 1975 formula did not reduce the stool output or duration of diarrhea. The new solution, with a lower sodium concentration, is more palatable and may be more appropriate for noncholera diarrhea than the 1975 solution. The stool in cholera has a very high sodium concentration (100–135 mEq/l). Thus, some authors expressed concern that the new solution may be associated with hyponatremia when used to treat cholera. In a Cochrane metaanalysis, hyponatremia (<130 mEq/l) was more common during treatment of cholera with the new formula, although symptomatic hyponatremia was not observed.

To maximize sodium uptake, glucose is an essential component of ORT solutions. Although nutrient-independent salt absorption (Na/H and Cl/HCO₃ exchanges) are impaired by cholera, the enterocyte’s...
sodium-glucose cotransporter (SGLT1) is not. Intestinal transmembrane transport of one glucose molecule is coupled to two sodium ions, resulting in an osmotically driven influx of more than 200 water molecules.\textsuperscript{34,39,40} By using rice powder or other glucose polymers, it is possible to increase the delivery of carbohydrate to the small intestine without increasing the osmolarity of the solution to levels that increase diarrheal losses.\textsuperscript{41} Other molecules have been shown to enhance water absorption by the gastrointestinal tract. Ramakrishna et al\textsuperscript{42} conducted a trial in India in which amylase-resistant starch (high-amylose maize starch) was added to ORT solution. This starch is not broken down until it reaches the colon, where it is fermented into short-chain fatty acids and absorbed with transporters coupled to sodium. L-arginine, the nitric oxide precursor, stimulated water and electrolyte absorption from the small bowel. This may be related to nitric oxide-induced vasodilation.\textsuperscript{43} Amino acids, including glycine, alanine, and glutamine, enhance sodium and water absorption from the small bowel by different sodium coupled transporters. Thus, amino acids have been proposed for optimization of ORT solutions.\textsuperscript{44–47}

Further research on oral fluids for plasma-volume expansion has been performed by NASA. Prolonged space flight is associated with plasma volume losses and cardiovascular deconditioning, such that reentry into a gravitational field causes “gravitational reentry syndrome,” which may cause orthostatic symptoms or syncope during critical mission events. To counteract this, Greenleaf et al\textsuperscript{48} evaluated several oral fluids of varying sodium content and osmolarity. They determined that a nearly isotonic electrolyte solution was most effective at expanding plasma volume. This resulted in AstroAde, which contains 164 mEq/l of sodium (Table 2). By contrast, commercially available sports drinks such as Gatorade® are low in sodium (20 mEq/l), reflecting the low sodium content of sweat (30–60 mEq/l) and the desire to produce a palatable commercial product.\textsuperscript{49}

**GASTROINTESTINAL FUNCTION IN BURN SHOCK**

On the basis of the extensive experiences reviewed earlier, it is apparent that oral replacement is highly effective for treatment of the large-volume losses in life-threatening diarrhea. The extent to which this can be adapted to burn shock resuscitation is not evident. Burn shock, in contrast to infectious diarrhea, is associated with marked changes in blood flow to the gastrointestinal tract. Asch et al\textsuperscript{50} in a canine model documented decreases in directly measured regional blood flow to the small intestine using radioactive microspheres, with intestinal perfusion being partially restored by intravenous fluid resuscitation.

### Table 2. Oral hydration solutions compared with intravenous solutions

<table>
<thead>
<tr>
<th>Formula</th>
<th>Carbohydrate mM (wt/vol)</th>
<th>Na⁺</th>
<th>Cl⁻</th>
<th>K⁺</th>
<th>Buffer</th>
<th>Osmolarity (mOsM)</th>
<th>Use</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Oral solutions</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>WHO ORS (1975)</td>
<td>111 (2.0)</td>
<td>90</td>
<td>80</td>
<td>20</td>
<td>30</td>
<td>331</td>
<td>Diarrhea</td>
</tr>
<tr>
<td>WHO ORS (2002)</td>
<td>75</td>
<td>75</td>
<td>65</td>
<td>20</td>
<td>10</td>
<td>245</td>
<td>Diarrhea</td>
</tr>
<tr>
<td>Gatorade</td>
<td>250 (4.5)</td>
<td>20</td>
<td>20</td>
<td>3</td>
<td>3</td>
<td>280</td>
<td>Sports</td>
</tr>
<tr>
<td>Pedialyte</td>
<td>139 (2.5)</td>
<td>45</td>
<td>35</td>
<td>20</td>
<td>30</td>
<td>250</td>
<td>Dehydration</td>
</tr>
<tr>
<td>Rehydralyte</td>
<td>139 (2.5)</td>
<td>75</td>
<td>65</td>
<td>20</td>
<td>30</td>
<td>325</td>
<td>Dehydration</td>
</tr>
<tr>
<td>Fox’s Na Lactate</td>
<td>0</td>
<td>161</td>
<td>0</td>
<td>0</td>
<td>161</td>
<td>321</td>
<td>Burn</td>
</tr>
<tr>
<td>Moyer’s Citrated NaCl</td>
<td>0</td>
<td>85</td>
<td>63</td>
<td>0</td>
<td>29</td>
<td>160</td>
<td>Burn</td>
</tr>
<tr>
<td>Monafo’s HLS</td>
<td>0</td>
<td>300</td>
<td>200</td>
<td>0</td>
<td>100</td>
<td>600</td>
<td>Burn</td>
</tr>
<tr>
<td>Liquidsorb</td>
<td>222 (4.4)</td>
<td>60</td>
<td>44</td>
<td>4</td>
<td>28</td>
<td>370</td>
<td>Burn</td>
</tr>
<tr>
<td>Jiang’s Burn Drink</td>
<td>252 (5.0)</td>
<td>48</td>
<td>28</td>
<td>0</td>
<td>20</td>
<td>344</td>
<td>Burn</td>
</tr>
<tr>
<td>Ricelyte</td>
<td>0</td>
<td>50</td>
<td>45</td>
<td>25</td>
<td>34</td>
<td>200</td>
<td>Dehydration</td>
</tr>
<tr>
<td>AstroAde (NASA)</td>
<td>0</td>
<td>164</td>
<td>76</td>
<td>0</td>
<td>40</td>
<td>253</td>
<td>Plasma volume expansion</td>
</tr>
<tr>
<td><strong>Intravenous solutions</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Lactated Ringer’s</td>
<td>0</td>
<td>130</td>
<td>109</td>
<td>4</td>
<td>28</td>
<td>270</td>
<td></td>
</tr>
<tr>
<td>0.9% NaCl</td>
<td>0</td>
<td>154</td>
<td>154</td>
<td>0</td>
<td>0</td>
<td>308</td>
<td></td>
</tr>
<tr>
<td>Plasmalyte-R</td>
<td>0</td>
<td>140</td>
<td>98</td>
<td>5</td>
<td>50</td>
<td>294</td>
<td></td>
</tr>
</tbody>
</table>

An index of gastrointestinal hypoperfusion is the pCO₂ gap (PiCO₂–PaCO₂, where PiCO₂ is measured by gastric tonometry). Decreased pCO₂ at 6 hours after burn was associated with increased mortality in human burn patients.51

The clinical relevance of these observations has been debated, mainly with respect to the timing of enteral nutrition. At the U.S. Army Institute of Surgical Research (USAISR, the U.S. Army Burn Center), vomiting secondary to gastric ileus during burn shock is prevented by the placement of a nasogastric tube to suction for all patients with burns of 20% TBSA or greater. Postburn gastrointestinal ileus may significantly limit net absorption of tube feeds for several days postburn.52 Thus, USAISR surgeons typically have initiated enteral feeding at the close of the first 48 hours after burn, once return of bowel function and resolution of ileus have occurred.53 Others have argued that early enteral feeding prevents ileus, protects the gut, and maintains gut barrier function. At the University of Texas Medical Branch at Galveston, burn patients and some trauma patients are started on enteral feeding within 2 hours of admission. Recently, Brown et al54 reported a retrospective review of experience with postburn vomiting. Eighteen patients (16.4%) vomited significantly enough to prevent initiation of enteral feeding during the first 48 hours after burn. Patients with vomiting had larger burn size and older age. It is remarkable that little consensus exists to date on when best to initiate enteral feeding in patients with major burns.

RECOMMENDATIONS FOR ORAL RESUSCITATION

Insufficient data exist to support definitive guidelines on the timing and method of oral burn shock resuscitation. Intravenous resuscitation remains the standard of care for burn shock, as advocated in Advanced Burn Life Support (ABLS) and similar courses.3 However, oral resuscitation should be considered when intravenous fluid therapy will be delayed, and the patient is conscious and without apparent gastrointestinal injury. It is important that water without electrolytes should not be administered in significant quantities due to the risk of inducing hyponatremia. Oral resuscitation could be used alone in many patients and supplemented or replaced by intravenous resuscitation in those patients with extensive injuries. Enteral resuscitation may be more effective when started within the first hour after injury and may be more effective in children than in adults. Fluid can be delivered in small sips by drinking or can be delivered using a spoon. A fixed volume of one cup or 250 ml can be programmed for consumption every 15 minutes. If this dose is well tolerated, consumption can be increased to six cups per hour. If not tolerated, infusion through a nasogastric tube may be effective. Fox18 did not consider vomiting a contraindication to enteral resuscitation. We are unaware of any reports on aspiration with oral resuscitation of burns. Table 2 provides a summary of the various oral rehydration fluids that have been used for burn shock and other indications. In addition, Table 3 provides recipes for homemade oral resuscitation fluids. The first one, based on clean water, has been recommended by international organizations.55 When available, however, the WHO ORT fluids should be used in lieu of homemade solutions. Several studies have demonstrated that considerable error may occur when homemade solutions are used.56 We based the other solutions in Table 3 on fluids that may be available locally during a disaster. Note that oral rehydration fluids can be made from commercial Gatorade with salt added (Table 3, row 3) or lactated Ringer’s solution with sugar added (Table 3, row 4). One of the authors (L.C.C.) recalls the use of oral lactated Ringer’s solution (without sugar) by soldiers as self-treatment for heat exhaustion on a battalion objective during war in Panama, Operation Just Cause, 1989.

RESUSCITATION BY PROCTOCLYSIS

In addition to the orogastric route, patients with burn shock also may be resuscitated by proctoclysis, that is, the rectal infusion of saline solution. As mentioned previously, proctoclysis was performed by both Sneve13 and Underhill14 before intravenous therapy became widely available. An early description of proctoclysis was

<table>
<thead>
<tr>
<th>Base Ingredient</th>
<th>Volume</th>
<th>Sugar</th>
<th>Salt*</th>
<th>Baking soda</th>
</tr>
</thead>
<tbody>
<tr>
<td>Clean water</td>
<td>1 liter</td>
<td>8 tsp</td>
<td>1/2 tsp</td>
<td>1/2 tsp</td>
</tr>
<tr>
<td>Gatorade</td>
<td>Quart bottle</td>
<td>No addition</td>
<td>Add 1/4 tsp</td>
<td>Add 1/4 tsp</td>
</tr>
<tr>
<td>Lactated Ringer’s</td>
<td>1 liter</td>
<td>8 tsp sugar or glucose</td>
<td>No addition</td>
<td>No addition</td>
</tr>
</tbody>
</table>


*In the absence of baking soda, double the salt dose.
provided by John M. Murphy of Chicago in 1913, leading to the term, “Murphy’s Drip”.

The saline solution is made by adding one dram [1.77 g] each of sodium chlorid and calcium chlorid [sic] to each pint [473 mL] of hot water. The solution must be kept at a temperature of 100° to 101°F. The average quantity given is 1.5 to 2 pints every two hours... The average twenty-four-hour quantity is 18 pints, or 1.5 pints every two hours... In a child of eleven years as much as 30 pints were given in twenty-four hours.

Proctoclysis is rarely used in the West today, although it has been used by surgeons in Africa without ready access to sterile intravenous fluids and cannulas (Ronald Tolls, MD, personal communication, 1994). A compelling case report describes the use of ORT solution for proctoclysis in the care of a patient with life-threatening hemorrhagic shock from gastritis during a trek in Nepal. One liter of double-strength solution followed by 2 liters of standard-strength solution were given over the course of 3 hours.

Proctoclysis with tap water or normal saline solution recently has been used for the palliative care of terminally ill patients for 15 ± 8 days. The infusion rate was approximately 250 ml/hr. Proctoclysis currently is under study in a porcine model of thermal injury at the University of Texas Medical Branch at Galveston, Texas.

We mention in passing that intraperitoneal fluid therapy has been used for patients with limited intravenous access. Because of the risk of abdominal compartment syndrome during the resuscitation of burn patients and because of the need for insertion of peritoneal catheters under sterile conditions, this route seems unlikely to be of use during clinical burn care.

**FUTURE RESEARCH**

Clearly, research is needed in large animal models and in burn patients. Several questions remain to be answered:

- What is the ideal solution for oral resuscitation of burn shock?
- What is the effect of glucose and amino acids on intestinal absorption during burn shock?
- How much volume can be delivered during burn shock via oral and rectal routes?
- Does gastrointestinal resuscitation produce equivalent outcomes with respect to end-organ and systemic perfusion as does intravenous resuscitation?
- What is the effect of changes in regional blood flow to the gastrointestinal tract on absorption?
- What is the effect of burn shock on gastric motility?
- Can gastric motility be pharmacologically enhanced during burn shock?
- Is there an upper limit to the burn size of patients who can be successfully resuscitated by the gastrointestinal route?
- What is the effect of extremes of age on the success of gastrointestinal resuscitation?

To this end, Kramer and colleagues have performed initial studies of duodenal infusion of the 1975 WHO ORT solution in swine with 40% TBSA burns. Changes in hematocrit, hemodynamics, and plasma volume were similar in enterally and intravenously resuscitated animals. The “triple-lumen” technique was used to estimate intestinal absorption rates. A total of 93% of infused solution was absorbed; absorption rates increased from 77 ml/hr to 296 ml/hr during the course of the 4-hour experiment.

**CONCLUSION**

Historical experience and preliminary large animal research indicate that gastrointestinal routes are feasible for the fluid resuscitation of many patients with burn shock. This is particularly true during mass-casualty and third-world situations in which cannulas and sterile fluids are in short supply. Extensive research with oral resuscitation for patients with cholera has led to the development of inexpensive solutions and effective delivery regimens. More research is needed to define fully the best approach to gastrointestinal resuscitation in burn patients.

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**REFERENCES**


