Discussion

Dr. Watts R. Webb (New Orleans, Louisiana): This is a patient at the Charity Hospital in New Orleans burn unit that has just been resuscitated during the past 48 hours with the standard resuscitation program of lactated Ringer's, by the Parkland formula, 4cc/kg/per cent body burn (Slide). He has a 52% body burn. You can see the massive edema that's occurred, not ony externally, but also in his brain, with resulting confusion and anxiety; in his lungs, requiring respiratory support; and his GI tract, so that it is no longer functional.

(Slide) The next slide shows a similar patient with a 53% burn, including an inhalation injury, who has been resuscitated with the solution devised by Dr. Carl Jelenko, which is 240 mEq of sodium and 12½ g of albumin per liter. Note the lack of edema, no endotracheal tube and no gastric tube. Virtually all of these patients—and we have now over 15, including five children, who respond very much the same way—have been resuscitated, with their resuscitation phase over in about 16 hours. We're utilizing only a little more than half the sodium and less than half of the usual water load.

In fact, looking at Dr. Caldwell's statistics, I believe that his hypertonic saline solution had reached the end point of resuscitation at about 16 hours, when the sodium levels reached such a high level.

There has been a very popular myth that there is a generalized capillary permeability secondary to burns. We feel this is only because the mechanical and the oncotic capacities of the capillary system have been exceeded. Most of the plasma proteins have been washed out, reducing the oncotic pressures down in the range of 10 mmHg rather than the usual 20–25.

(Slide) Here is some work by Dr. Jelenko showing the rate of radioactive albumin turnover, or disappearance from the plasma. The normal range shown below is about 5–8% per hour. Note that with Ringer's solution during the first few hours it may be as high as 30% per hour; with hypertonic saline solution, it may be in the range of about 25%. Utilizing the solution, we have described, given at a slow rate, notice that disappearance is in the high normal range, and after the first 24 hours is completely in the normal range. The edema, as you can tell from looking at the patient you just saw, occurs only in the area of the burn, and not throughout the body, and particularly not into the lungs.

(Slide) These are the hemodynamic effects of hemorhagic shock. We are now demonstrating with on-going experiments in scald burns—80% scald in animals—exactly the same phenomenon. You will notice that during hemorrhage there is a tremendous rise in pulmonary resistance, going up some five-fold; systemic resistance goes up double; and then even during the phase of resuscitation there is still a continuing high rise in systemic and pulmonary resistance.

It is our feeling that fluid must be given slowly enough to allow these to adjust, and the resistance to fall, or else to use vasodilating agents, even during the period when the patient has a relatively low blood pressure. With this, esentially all of our patients have been well resuscitated within 16 hours, and returned to normal GI function, and brain and lung function.

So we think there is no pat formula. There are only three principles involved. First, restore the sodium without a water overload; second, maintain normal pulmonary and peripheral vascular resistances, even using vasodilators if needed; and third, maintain a normal oncotic pressure.

Dr. Boyd W. Haynes, Jr. (Richmond, Virginia): This is a prospective study and is characteristic of the work that he has produced. I want only to raise a few cautions, and discuss two points briefly.

First, we have come to think of edema as something bad. I am not at all sure that that is a correct interpretation, although massive edema to the point of producing pulmonary overload obviously is bad. There is a large body of experience using the Evans and the Brook formula which Dr. Caldwell used to compare to the hypertonic solution—a large body of experience which points out that although patients may receive more water, they do well, characteristically well, and they rarely develop pulmonary complications. There does not appear to be any increased necessity for escharotomy, and edema is absorbed and excreted in due course, leaving the patient's wounds dry and healing well.

The other point that's an important issue to raise is: what is happening inside the cell while the hypertonicity is occurring outside? Dr. Caldwell has detailed part of this to you. I have some concerns, however, about electrolyte concentrations within the cell, because there is some data that suggests that major intracellular changes are occurring. This is not exclusively a feature of hypertonic solutions, because lactated Ringer's solution produces some increase in sodium within the cell; but apparently hypertonic solutions may induce four or five times normal quantities, plus significant potassium losses.

Now, whether this is important, and to what extent it is important, is the issue, and I would like Dr. Caldwell to coment on this.

Dr. Basil A. Pruitt, Jr. (Fort Sam Houston, Texas): I speak for avoidance of excess of either volume or salt load. We at the U.S. Army Institute of Surgical Research would agree with Dr. Caldwell that burn patient resuscitation should be affected with the least possible volume, and, in fact, would have given those patients resuscitated with the lactated Ringer's solution no additional free water.

(slide) We have, in recent years, modified our resuscitation formula to eliminate administration of additional electrolyte-free water in the first 24 hours postburn, and administer only lactated Ringer's solution in the amounts indicated here. We also decrease salt loading by avoiding further salt administration in the second 24 hours.

We give these fluids in an amount to produce 1 ml of urine per kg/hr in children of 30 kg or less. It would appear from Dr. Caldwell's data that a 30 kg child with an 80% burn would produce approximately 2100 ml of urine in 48 hours, rather than the 1400 with which we would be satisfied; and the need for a greater urinary output is difficult to discern.

We feel, on the basis of both laboratory and clinical studies that sodium dosage and sodium balance, rather than sodium concentration, is an important factor in burn patient resuscitation, and Dr. Caldwell's data would confirm this. That is, despite the administration of a 26% greater load of salt, sodium balance in the hypertonic lactated saline group was only 7% greater at the end of 48 hours. Hypertonic resuscitation fluids, as the authors point out, exert at least part of their volume supportive effect by piracy of intracellular water. And they have identified both advantages—that is, less edema in unburned parts—and disadvantages—a diminished urinary output—associated with such therapy.

The limitations of hypertonic resuscitation appear to be a 15% decrease in intracellular fluid volume and a plasma sodium level of 165–170 mEq/l. With those limits, one can construct monograms, as shown here, and calculate the amount of fluid containing 250 mEq of sodium per liter which will bring about such changes, and necessitate the administration of either electrolyte-free water or less concentrated salt solutions for continued physiologic support.

Such was apparently done in Dr. Caldwell's patients, and one wonders what the sodium concentration in the total fluid load given really was. Calculations using his data indicate that in the 30 kilo patient with an 80% burn it would be 151 mEq/l.—hardly hypertonic.

The authors' physiologic hypotheses are somewhat puzzling, and I ask three questions for clarification. 1) If hypertonic fluids given in the early postburn period result in greater rates of urinary formation, why was urinary output significantly less in the hypertonic treated patients and why was water supplementation necessary?

2) Since following resuscitation the patient is physiologically set to lose water and conserve salt, does the success of hypertonic resuscitation depend on the use of silver nitrate soaks to promote transeschar leaching of the infused sodium load?

3) The lower levels of serum potassium in the hypertonic lactated saline treated group might well be a distinct advantage in patients with extensive burn, in whom serum potassium levels are frequently elevated. Can the authors tell us whether the lower potassium levels

in those patients were the result of the induced alkalosis to which they make reference, or to the kaluresis which has been observed to accompany infusion of hypertonic salt on the solution?

Again, I enjoyed the paper. I think that the authors would agree that the goal of resuscitation [slide] is to maintain vital organ function at the least physiologic cost. They have quite rightly emphasized the importance of limiting the fluid volume administered during resuscitation to decrease later complications in extensively burned patients.

DR. FRED T. CALDWELL, JR. (Closing discussion): I don't know why the potassium is lower. Lactated Ringer's only has 4 mEq of potassium in it. It may have to do with the sustained metabolic alkalosis. I just have to say, I don't know.

As far as silver nitrate being involved in this, none of these patients were treated with topical silver nitrate. They all had Silvadene and/or Silvadene plus cerium, which has no known leaching effect.

The absolute urine volume for the hypertonic group was not greater; but volume for volume of fluid administered, hypertonic saline resulted in more urine formation. This was based on rat experiments which indicated that volume for volume hypertonic fluid given to burned rats produces a sodium diuresis and volume for volume more urine than a comparable volume of hypotonic solution.

We're making a little progress, I really believe, when the Brooke people stand up and say they have deleted the arbitrary 2l of sodium-free water from burn resuscitation. That's real progress.

I don't believe that Dr. Webb's comments require any response. There are two slides in the back of mine I'd just like to show you; you don't have to have albumin in hypertonic lactated saline in order to minimize swelling.

(Slide) This child was given classical hypotonic resuscitation. This is 24 hours postburn—just what you would expect.

(Slide) This child was given hypertonic lactated saline solution. This is how he looks at 24 hours. Neither one of them received any albumin. I am not sure when albumin should be given, but I feel sure it does no good during the first 24 hours postburn.

I agree with Dr. Haynes that some of the most important changes following thermal injury and resulting from resuscitation take place at the subcellular level. We really don't know much about these changes, and bench and clinical research need to be done in this area.