Fluid therapy in burns

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Most published accounts of fluid therapy for burns emphasize the advantages of the regime favoured by that author and give details of the scientific evidence for its superiority over all other regimes. Directly, or by implication, alternative methods are condemned or ignored. Clearly this is quite ridiculous because all the well recognized schemes of resuscitation have been validated in clinical practice. The obvious and important differences in these schemes must, therefore, be related to clinical events other than the prevention of burn shock.

However, so varied are the regimes in common use, and so apparently contradictory the statements made in suppport of them, that anyone could be forgiven for concluding that they share no common ground. The main objective of this paper is to show that all regimes of effective fluid therapy in burns are based on certain well established principles and that a common ground of therapy does exist. The secondary objectives are to indicate why particular regimes have become favoured in particular localities and why the random choice of a regime is likely to be dangerous.

Need for salt and water

During the second half of the 19th century, several workers noted the similarity between burns and cholera, and saline infusions were suggested as appropriate treatment. In 1905 Sneve reported the effective administration of 0.9% saline in burn shock and in 1926 Davidson (perhaps better known for introducing tannic acid treatment for burns) reiterated this approach. By 1930, Underhill and others had laid the cornerstone of effective treatment of burn shock, namely that salt and water are the essential requirements.

First principle: To achieve survival, patients with extensive burns need to be given large quantities of fluid which must contain sodium salts.

Quantity of fluid required

The central role of hypovolaemia in the pathogenesis of burn shock was first emphasized by Blalock (1931). From experiments on burned dogs, he suggested that the observed hypotension and oliguria were a consequence of hypovolaemia resulting from fluid loss from the circulation into the burned tissues. As the concept became recognized that a time-limited change in capillary permeability was a characteristic feature of the burn wound, so the idea of devising formulae to predict the likely fluid requirement became widespread. The early formulae were those of Harkins (1942) and Cope & Moore (1947), but that of Evans *et al.* (1952) achieved worldwide usage. This formula indicated that the fluid requirement was related both to the size of the burn and the size of the patient; that the fluid should consist of equal quantities of colloid and non-colloid electrolyte solutions; and that two-thirds of the total requirement should be given in the first 24 hours. The total volume predicted was 3 ml/kg body weight/percent burn.

The Brooke formula (Reiss *et al.* 1953) indicated the same total fluid volume as that of Evans but reduced the proportion of the colloid and increased the infusion rate during the first 8 hours. This tendency to reduce colloid content and infuse the fluid faster was endorsed by Moyer *et al.* (1965) and seemed to reach its zenith in the recommendations of Baxter & Shires (1968), the guidelines of whose regime are: during the first 24 hours no colloid, 4 ml/kg/% burn of Ringer's lactate – one-half of which is given in the first 8 hours (cf. Evans' formula: 2 ml/kg/% burn for the whole of the first 24 hours). In practice, the infusion rate is slowed down once a urine flow of more than 50 ml/hour has been achieved (Baxter 1971).

From these and other well validated regimes, a second principle can be discerned that indicates the magnitude of the fluid requirement.

Second principle: The total volume of salt-containing fluid required to satisfy obligatory burn oedema and make good urine losses is between 2 and 4 ml/kg/% burn, but the actual volume is to some extent dependent upon the type of salt solution used.

Crystalloid versus colloid

Although the treatment of burn shock is quite possible with 0.9% saline, there is convincing evidence (Fox 1970) that a balanced salt solution is preferable. In the USA during the 1960s, Ringer's lactate was used with increasing enthusiasm and the possible advantages of including a colloid in the fluid were dismissed. This attitude was summarized by Moncrief (1973) in his statement: 'Those who continue to use colloid in the first 24 hours post-burn have little going for them other than tradition'.

It seems at least possible that two quite unrelated events encouraged this enthusiasm for the use of Ringer's lactate. In 1968, the National Research Council in the USA issued a statement highlighting the risk of hepatitis in using pooled, whole, human plasma. The statement endorsed the concern that had been felt in the USA for several years about the use of plasma, and concluded: 'The committee recommends that the use of whole, pooled, human plasma be discouraged and even discontinued unless a clear-cut case can be made for its unique requirements'. Although the use of heat-treated albumin solutions was not similarly discouraged, it can be imagined that in a litigation-conscious society the clinician might feel safer if he avoided the use of any blood product if at all possible.

Meanwhile, Shires *et al.* (1964) had reported from Dallas that an acute volume deficit occurred in the extracellular fluid (ECF) space of animals during shock and in man during surgical trauma. It became standard practice to infuse large volumes of Ringer's lactate during surgery and the specific advantages of a crystalloid fluid in the treatment of hypovolaemia seemed to have been established.

In 1967 a warning shot was fired by Moore & Shires in an editorial entitled 'Moderation': 'The objective of care is restoration to normal physiology and normal function of organs ... This can never be accomplished by inundation'.

Enthusiasm for crystalloid solution administration in almost every surgical situation continued, however, and Roth *et al.* (1969) in Los Angeles, concerned at the vast infusion regimes becoming popular (intraoperative fluid loads had doubled whilst sodium loads had increased by a factor of 11), critically examined the case for a deficit. Their results showed serious errors in the animal model and methods of measurement used by Shires *et al.* (1964). They concluded that there was no ECF deficit in dogs following haemorrhagic shock or in patients undergoing even major surgery, and *The Lancet* (1969) commented: 'Now, in the light of the work of the Los Angeles group, there seem to be good reasons for recession of the high tide of salt water'.

In the event, the tide has been a long time going out. Studies by Moyer and others in the 1960s clearly showed that in hypovolaemic states only one-quarter of an administered crystalloid solution will remain in the circulation, whilst three-quarters will be distributed to the extravascular interstitial space (Moyer & Butcher 1967). In contrast, plasma administered to the hypovolaemic subject remains in the circulation. Following a major burn, the volume shift of plasma into the damaged tissues to form burn oedema represents a large new plasma space created by the injury and in equilibrium with the original plasma volume. Resuscitation can be viewed, therefore, as an attempt to expand the plasma volume enough to permit the coexistence of this new space with the normal plasma volume. Because the burn oedema is contained in a leaky sac, in that some fluid leaves the body via the surface of the burn, additional fluid is required to make good this exudative loss.

Attempts to fill the plasma volume by giving isotonic crystalloid solutions will succeed only at the expense of producing interstitial oedema in the unburned portions of the body, whereas administration of a colloid solution will be accompanied by minimal non-burn oedema. This difference has been demonstrated beyond doubt in several clinical trials. Hall & Sørensen (1978), in a 5-year prospective randomized clinical trial of dextran 70 in saline versus Ringer lactate solution, showed that the group of patients who received colloid maintained normal haematocrit during the resuscitation whereas those in the crystalloid group, who received approximately twice as much fluid, demonstrated marked haemoconcentration and developed massive generalized oedema.

More recently, Mason (1980) presented a simple though elegant computer program which demonstrated the enormous non-burn oedema to be expected when crystalloid solutions are used. He said: 'Only experience will disclose the merit, if any, of deliberately restricting edema load in the normal interstitium, but if such restriction is desirable, this set of equations will provide a guide to flexible control of resuscitation using electrolyte solutions alone in smaller burns and larger patients and high fractions of plasma in the resuscitation of large burns, children, and those in whom any excessive volume load is an unwarranted risk'.

In addition to the generalized non-burn oedema that results from crystalloid resuscitation, the exclusion of colloid from the replacement fluid would seem to prolong the period of plasma volume deficit post-burn. Studies at the US Army Institute of Surgical Research (subsequent to the period when Moncrief was its Director) have thrown further light on the role of colloid

solutions in the treatment of burn shock. In a comparison of crystalloid and colloid regimes, echocardiographic measurements of left ventricular function in burned patients showed that cardiac output had not returned to normal at 24 hours post-burn in the crystalloid group, whereas in the colloid group cardiac output had returned to normal between 12 and 16 hours post burn (Dorethy *et al.* 1977).

A further comparative study at that Institute (Goodwin *et al.* 1980) showed that effective resuscitation was associated with smaller volumes of colloid-containing electrolyte solutions than when crystalloid solutions alone were used but that in both groups of patients similar increases in lung water were observed. The rather surprising suggestion was then made that if a colloid solution was given in the quantity that would be appropriate for a crystalloid solution, a further increase in lung water might result. Thus, it was implied, 'excessive' colloid therapy is more dangerous than 'routine' crystalloid therapy. A truism perhaps, but also an example of the need to appreciate the particular properties of the fluids chosen to effect resuscitation – a point elaborated later.

It should be noted that in these studies (and in many other American studies), the 'colloid' group of patients received a less concentrated plasma protein solution than would be 'traditional' in the UK. Nonetheless, even these 'watered-down' colloid regimes showed significant differences from the crystalloid regimes and although the rediscoverers of these phenomena are not prepared to ascribe to them any clinical advantage, the weight of evidence cannot be denied.

Third principle: Compared with the use of an isotonic salt solution, the use of a similar fluid containing a suitable colloid is associated with less generalized oedema, a reduced total fluid volume requirement and a reduced period of plasma volume deficit.

How much salt?

Monafo, having recognized that resuscitation with an isotonic salt solution could so easily become inundation, explored another approach to the provision of minimal fluid loading (Monafo *et al.* 1973). He carried to its logical conclusion in clinical practice the concept of the animal experimental work of Fox (1971), which had quantified the central role of the sodium ion in the treatment of burn shock. Monafo decreased the fluid load by increasing the tonicity of the fluid and thus administered the same salt load in a greatly reduced total fluid volume. His early studies utilized a solution containing sodium 300 mmol/l and chloride 100 mmol/l, but subsequently the sodium concentration was reduced to 250 mmol/l and the chloride raised to 150 mmol/l whilst the remaining 100 mmol/l of anion was provided by lactate. From the studies of Fox, Monafo and others, it has become clear that the quantity of sodium administered and retained by the body is important. A fourth basic principle has thus been elucidated.

Fourth principle: The quantity of sodium ions required for effective resuscitation has an order of magnitude of 0.5 mmol/kg/% burn.

How much water?

At first sight it may seem that because a simple inverse relationship applies to the concentration of salt and the volume of solution, i.e. 2 ml/kg/% burn of hypertonic lactated saline (Na 250 mmol/l) and 4 ml/kg/% burn of Ringer's lactate (Na 130 mmol/l) would each deliver about 0.5 mmol Na/kg/% burn, any regime that results in a total fluid administration much above 2 ml/kg/% burn must be suspect. Certainly, it would seem logical to deliver the optimum quantity of sodium in the minimum acceptable fluid volume in order to avoid unnecessary oedema, particularly in the unburned tissues. Part of the rationale of hypertonic therapy is that, to some extent, the patient is resuscitated with his own intracellular water; extracellular hypertonicity will result in a tendency for water to move out of cells, cellular overhydration will thus be prevented and the burn oedema 'satisfied' with the minimum exogenous water load.

Consequently, the advocates of hypertonic fluid therapy criticize any regime that is not hypertonic. They argue that in any regime where the average sodium concentration of the total fluid therapy is of the order of 140 mmol/l (or less), the patient must be receiving an unnecessarily large water load. This argument, however, fails to take into account the fact that the 'average' sodium concentration of a fluid therapy regime is relevant only in the context of total body water balance. If sodium-free water losses from the body (e.g. evaporation) are small, a hypertonic resuscitation regime results in hypertonicity of total fluid therapy, since little fluid other than the resuscitation fluid need be given. In marked contrast, when sodium-free water losses are high, as by evaporation from a burn wound exposed in a warm and dry environment, failure to replace this sodium-free water results in a progressive plasma hyperosmolality. If there is any impairment of renal function that limits renal concentrating power, death from hyperosmolality is a likely outcome (Eklund 1970). It can be seen, therefore, that even if the resuscitation fluid is hypertonic, should the burn wound be exposed, the necessity

to administer large volumes of sodium-free water will result in a relatively low sodium concentration of the total fluid therapy.

The treatment of the burn wound during the shock phase is thus highly relevant to the choice of a resuscitation regime. Surrounding the burn wound with a markedly hypotonic fluid in the form of 0.5% silver nitrate solution, as practised by Monafo *et al.* (1973), will not only prevent sodium-free water loss from the burn wound but may also result in a water gain and a salt loss. If the burn wound is exposed or covered with dry dressings, the evaporative water loss is between one and two litres per 9% burn during the first 24 hours (Roe 1966). Topical creams applied to the wound and then covered with absorbent dressings would seem to limit this loss to the lower end of the range (Settle & Eve 1975).

Table 1 shows the theoretical fluid and sodium loads and the extrarenal losses calculated for a 70 kg patient with 40% burn during the first 24 hours post-burn, treated either by traditional colloid therapy and topical cream dressings to the burn (Settle 1974*a*, 1981), or hypertonic lactated saline intravenously and silver nitrate soaks to the burn (Monafo *et al.* 1973). Evaporative water loss is occurring in both regimes. In the first, the water comes from the patient and thus has to be administered to him if severe hyperosmolality is to be avoided. In the second, it comes from the wet dressings and is replenished from the silver nitrate solution poured on every few hours. When the evaporative water loss from the patient is subtracted from the total water load, it can be seen that the net sodium and water balances are not dissimilar, particularly when the possible differences in renal and burn wound sodium losses are remembered. These differences include the marked increase in renal excretion of sodium that follows the infusion of a hypertonic salt solution compared with an isotonic salt solution; and the possibility that more sodium is lost from the burn wound into the dressings soaked with silver nitrate solution than into those containing a topical cream, even when the cream is a silver compound such as silver sulphadiazine.

Since the renal mechanism for the excretion of sodium-free water is blocked by the persistent activity of antidiuretic hormone, if sodium-free water intake exceeds the loss by evaporation and respiration, hyponatraemia will develop and result in cellular overhydration. As this is clearly undesirable, the aim should be to maintain a moderate degree of extracellular hypernatraemia whichever fluid regime is chosen. This is possible either with 'hypernatraemic' fluid therapy plus near-total prevention of evaporative water loss from the wound (wet soaks or truly occlusive dressings), or an 'isonatraemic' or even 'hypo-

Table 1. Fluid and sodium loads and extrarenal losses calculated for a 70 kg patient with 40% burn

	Treatment method	
First 24 hours	Settle	Monafo
Replacement fluid	Plasma	HLS
Volume (ml)	7000	5488
Na^+ concentration (mmol/l)	153	250
Na^+ load (total mmol)	1071	1372
Na^+ free water (ml)	3000	Nil
Water load (total ml)	10000	5488
Evaporative water loss (ml)	4400(?)	Nil
Insensible water gain	Nil	+?
Extrarenal sodium loss (into dressings)	+?	++?

HLS = Hypertonic lactated saline

natraemic' fluid therapy so long as sufficient water evaporates from the wound to produce a moderate net hypernatraemia. It is not possible if 'isonatraemic' fluid therapy is combined with gross restriction of evaporative water loss from the wound.

Fifth principle: The total water requirement (and hence the average sodium concentration of the fluid therapy) varies depending upon the treatment of the burn wound. The quantity of sodium-free water administered should not exceed that required to prevent marked hypernatraemia.

How much is enough?

The fluid loss in burns that without treatment leads to hypovolaemia, is different from almost all other mechanisms of fluid loss by virtue of its predictability. Unlike the patient with severe multiple injuries whose requirement for blood transfusion is quantified only as transfusion proceeds, the likely quantity of fluid required by the extensively burned patient can be estimated at the outset. Thus, the fluid regime is essentially a means of preventing gross shock rather than a series of actions each designed to correct the hypovolaemia present at the time of assessment. Assuming that appropriate fluid therapy is started within an hour or so of the burn and continued as part of a proven regime of treatment, the gross signs of hypovolaemic shock should rarely be seen. This is not to say that the use of a proven formula absolves the clinician from the need to monitor the effectiveness of the treatment. The formula simply indicates the order of magnitude of the fluid requirement so that monitoring can concentrate on the precise requirements of the individual patient.

It should be a general rule not to place total reliance on any one physiological variable. Rather

it should be the aim to correlate the information provided by the measurement of those variables that are relevant, reasonable and practicable. An investigation is relevant only if it is likely to be a fairly sensitive index of the effectiveness of resuscitation, bearing in mind the nature of the fluid being used. The progressive rise in haematocrit (Htc) that characterizes untreated hypovolaemia resulting from burns can be halted and reversed by the infusion of whole plasma. Therapy with plasma protein fraction that otherwise appears to be satisfactory is associated with higher Htc values than would be expected with whole plasma, whereas effective resuscitation with a crystalloid fluid will be accompanied by very high Htc values. Hence the serial measurement of Htc is more relevant to a colloid regime than one consisting solely of crystalloid. Similarly, because of the different properties of colloids and crystalloids as plasma expanders, the measurement of central venous pressure (CVP) has been found to be a more sensitive index of the state of the capacitance circuit when colloids are being used than when they are not. Indeed, reliance upon CVP measurement as the main index of effective crystalloid therapy can have disastrous consequences, because the readings may remain low until the whole of the extracellular space has been filled. Thereafter, the continued infusion of fluid may be associated with a rapid increase in venous pressure followed by pulmonary oedema almost before the clinician has had time to respond to the new information.

If it is believed that effective resuscitation means the restoration of effective tissue perfusion, then indices of tissue perfusion should be useful in any regime. Comparison of shell and core temperature is a valuable index of peripheral perfusion, whilst measurement of the volume and concentration of hourly urine output is informative about renal perfusion. If urine volume and osmolality are measured on a short time scale, it is quite possible to detect impairment of renal function within a few hours of it occurring (Settle 1974*b*). Not only does such information permit the early diagnosis and treatment of renal impairment or failure, it also alerts us to the fact that urine flow in that patient is no longer a relevant index of resuscitation.

'Is it reasonable?' means 'Is it cost effective?' in the clinical as well as the financial sense. Does the value of the information obtained justify the risk and inconvenience to the patient and the financial cost incurred? In the average patient with a moderate uncomplicated burn where resuscitation is proceeding uneventfully, there is little point in measuring the arterial blood pressure since this can be maintained at a normal value in the face of significant hypovolaemia. Monitoring the blood pressure by sphygmomanometer would be of little value but would probably be harmless. Monitoring the blood pressure in such a patient by an intra-arterial line, however, would be unreasonable. 'Is it practicable?' means 'Are there sufficient resources and skills to make the investigation worthwhile?' Invasive or complex procedures must be done well to be of value, and if this is not possible it would be better to concentrate on something simpler.

Practitioners who work in specialized units and are able to practise relatively high technology medicine are guilty of a great disservice to their fellows (particularly in less well developed countries) if it is suggested or implied that, until they can achieve the same technological standards, no advice can be given to them about the care of burns patients.

Sixth principle: Methods of monitoring the effectiveness of resuscitation should be chosen taking account of the behaviour in the body of the fluid regime in use. Furthermore, they must be reasonable and practicable.

The common ground of fluid therapy

From these considerations of fluid therapy, the common ground for safe and effective resuscitation can be identified. The resuscitation of a patient with an extensive burn requires the administration, during the first 48 hours, of fluid containing salt and water. The optimum sodium load is of the order of 0.5 mmol/kg/% burn and the total volume of fluid required (excluding the replacement of excessive evaporative losses) has a magnitude of between 2 and 4 ml/kg/% burn. The actual volume required can be minimized by the inclusion of colloid or by using a hypertonic salt solution. The fluid regime should aim to produce a moderate extracellular hypernatraemia. If there is little extrarenal water loss (because evaporation from the burn wound has been curtailed) the fluid therapy should be hypertonic with respect to sodium. If extrarenal water losses are great (large evaporative and/or respiratory losses) the fluid therapy cannot, on average, be hypertonic with respect to sodium, or fatal hypernatraemia may result. However, the quantity of sodium-free water administered to balance extrarenal losses should only be sufficient to prevent marked hypernatraemia.

The effectiveness of the fluid therapy in correcting existing deficits and preventing hypovolaemic shock should be reviewed at frequent intervals, bearing in mind that the rate of infusion required should decrease with time. Usually, at least half of the total fluid requirement will have been given in the first 12 hours and periodic

adjustments to the rate of infusion should be made in response to information gained by monitoring the patient's condition.

It is unwise to rely upon any one physiological variable as an index of effective resuscitation. The methods of monitoring chosen should be relevant to the particular fluid regime in use and should be commensurate with the skills and facilities available. All the components of the resuscitation regime should be compatible with one another and with all other aspects of the management of the burn injury.

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