ACUTE BURN RESUSCITATION AND FLUID CREEP: IT IS TIME FOR COLLOID REHABILITATION

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SUMMARY. Fluid overloading has become a global phenomenon in acute burn care. The consensus Parkland formula that has excluded colloid use, the impact of goal-directed resuscitation, and the overzealous on the scene crystalloid resuscitation combined with subsequent inefficient titration of fluid administration and lack of timely reduction of infusion rates, have all contributed to this phenomenon of fluid overloading, known as fluid creep and recognized only recently, constituting a landmine in modern burn care. Solid evidence is supportive to the fact that excessive administration of crystalloid and the abandonment of colloid replenishment at some point of resuscitation are the major contributors to fluid creep. With available evidence from the literature, the present is a comprehensive review of literature about fluid creep, trying to determine the etiology behind it as well as to propose strategies to control its magnitude and complications, namely through colloid administration amongst other options.

Keywords: acute burns, fluid creep, post-burn resuscitation

Introduction

Acute resuscitation is a unique aspect of burn care and the ability to effectively resuscitate patients is critical to survival and overall outcome.1,2 The profound inflammatory response generated by a burn far surpasses that seen in trauma or sepsis, and the resultant fluid needs can be extreme.3 Before recognition of the magnitude of fluid shifts and the massive fluid requirements of severely burned patients, failed resuscitation was the leading cause of death.4,5 Delayed or inadequate fluid replacement results in hypovolemia, tissue hypoperfusion, shock and multiple organ failure.4,5 The primary goal of burn resuscitation is to maintain adequate tissue perfusion to end-organs and prevent ischemic injury at the lowest physiologic cost.6 With continued refinements in protocols, successful resuscitation of the majority of burn patients can now be achieved and acute renal failure has become rare.5

Baxter described the use of 4 cm$^3$/kg/% TBSA of an isotonic crystalloid solution, Lactated Ringer’s (LR), as a guideline for fluid resuscitation during the first 24 h post-burn.7,8 Half of the total fluids calculated are administered over the first 8 h, and the rest given over the remaining 16 h.9 As originally noted by Baxter, only 12% of patients would require resuscitation volumes greater than what he has proposed.7 These comprised patients with inhalation injuries and electrical burns as well as those with delayed resuscitation. He also noted that crystalloid alone was not sufficient to maintain the needed plasma volume for successful resuscitation.6

Baxter’s observations formed the basis of the original Parkland formula. Subsequently, an NIH-sponsored conference concluded that organ perfusion in burn patients should be maintained with as little fluid as possible, consisting of isotonic crystalloid at a volume between 2 and 4 ml/kg/%TBSA for the first 24 h and titrated to maintain urine output of 30 to 50 ml/h. The use of colloid in the second 24-h period was not included. This recommendation has defined the Parkland formula that has been since the accepted “consensus” for burn resuscitation.6

Despite the considerable variability in the recommended volume and salt loads among published fluid resuscitation recommendations, all concurred on the importance of sodium-containing fluids.10 To date, LR remains the most popular choice11 and is the cornerstone of initial burn management.12,13 Consensus guideline accepted by the American Burn Association (ABA) recommend initiation of fluid resuscitation with LR solution at a rate of 2 ml/kg/% to 4 ml/kg/% TBSA administered over the first 24 h post-burn, providing one-half of the estimated fluid over the first 8 h and the remainder over the remaining 16 h. In an effort to simplify the calculation of the initial fluid rate
and prevent fluid overload the rule of 10 was recently deve-
loped.\textsuperscript{10} Once this initial fluid rate is reached, optimal
resuscitation can only occur by carefully titrating fluids
based on patient’s response.\textsuperscript{8} Several reports, however,
have shown that fluids are being generously administered,
far beyond the original recommendations of the formula.\textsuperscript{12}

It appeared at first glance that excessive fluid loads
did not seem to be harmful.\textsuperscript{5} Early reports indicated that
only a minority of patients would require larger than pre-
dicted crystalloid resuscitation volumes and the issue of
over-resuscitation did not initially raise much concern.\textsuperscript{5}
However, in recent years, a number of authors have re-
ported that the Parkland formula significantly underesti-
mated the actual fluids required and given to patients.\textsuperscript{7}
Larger fluid volumes, as much as 5 to 8 mL/% TBSA/kg,
are being required for successful burn resuscitation in an
increasing number of burn patients.\textsuperscript{5,13} A recent survey has
shown that 55% of burn clinicians gave more crystalloid
than was predicted.\textsuperscript{12}

At present, fluid overloading has become a global phe-
nomenon in acute burn care.\textsuperscript{5} Many reports have revealed
that very large resuscitation volumes are being given even
in otherwise “uncomplicated” patients, with none of the
recognized factors of increased fluid requirements such as
inhalation injury, delay in resuscitation, polytrauma, or
high-voltage electrical contact. The insidious trend toward
providing increasing amounts of crystalloid fluid is a well-
meaning effort to avoid the onset of early acute renal fail-
ure.\textsuperscript{5} Sadly enough, “more is not better”. In fact, increased
fluid load, a phenomenon called “fluid creep”, can lead to
excessive edema and organ failure.\textsuperscript{18}

**Burn edema**

Tissue edema in or directly surrounding the burned
tissues is a well-recognized characteristic of burn injury
that can even develop in non-burned tissues.\textsuperscript{1,5} Some re-
ports have indicated that as much as 50% of the extracel-
lar edema observed with large burns occurs in non-burned
tissues.\textsuperscript{13,14} Massive edema formation, however, is the most
significant effect of fluid over-resuscitation that can result
in serious and even fatal complications.\textsuperscript{4,13}

Patients with burns in excess of 20% TBSA are typ-
ically at risk for developing burn shock. Their physiolog-
ic response can be divided into two phases: the emergent
phase also known as the initial ebb phase and the flow
phase.\textsuperscript{7} 12 h into the post-burn period, the ebb phase is at
its maximum and usually lasts 72 h. It is characterized by
a combination of increased capillary permeability and cel-
ular changes causing massive fluid shifts in burned as well
as non-burned tissues.\textsuperscript{7} Cellular changes comprise decrease
in cell trans-membrane potential with influx of sodium
leading to increased cellular swelling.\textsuperscript{7} Altered capillary perme-
ability is not only caused by heat injury alone; it is re-
lated to burn toxin activation and increased levels of in-
flammatory mediators in the blood especially oxidants, his-
tamine, prostaglandins, and other vasoactive substances.\textsuperscript{13,14}
The maximal capillary permeability and edema formation
within the wound occurs almost immediately post-burn.
The duration and magnitude of this transient effect are pro-
portional to burn size.\textsuperscript{7} In burns involving more than 25%
body surface area, capillary permeability is increased not
only in the damaged tissue but also in non-burned areas.\textsuperscript{15}
In contrast to early edema formation, subsequent fluid se-
questration occurs prominently outside the wound.\textsuperscript{7} Nev-
evertheless, the emerging literature indicates that increased
capillary permeability may only partly explain the edema
process, especially in full-thickness burns where much of the
capillary bed is coagulated and occluded by the ther-
mal injury.\textsuperscript{13}

Transcapillary fluid flux is governed by a set of phys-
ical forces and properties summarized by Starling’s equa-
tion of both the capillary bed and the interstitium.\textsuperscript{6,13} A bal-
ance is normally present so that edema does not form. Cap-
ilary hydrostatic pressure forces fluid out; this is coun-
terbalanced by colloid osmotic pressure dependent on plas-
ma proteins concentration that holds fluid in the capillary
lumen. Interstitial fluid pressure and interstitial colloid os-
motic pressure are the other forces controlling fluid flux.\textsuperscript{6,13}
Thus the forces driving fluid out of the capillary bed are
capillary hydrostatic pressure and interstitial osmotic pres-
 sure; these are opposed by plasma colloid osmotic pres-
sure and interstitial hydrostatic pressure.\textsuperscript{7} Following burn
injury, an imbalance in favor of fluid flux from the cap-
illary bed develops. Since protein will not transfer back
from the interstitium across the capillary membrane, all
the proteins that cross the capillary wall, whether in nor-
amal or abnormal circumstances, can regain the intravas-
cular space accompanied by much of the fluid that has
seeped out only in the presence of a functioning lymphatic
system.\textsuperscript{13}

Following large burn injury and in presence of the
burn “vascular leak syndrome”, the fluid required to main-
tain blood volume and blood supply to vital organs feeds
the fluid flux process and contributes to more edema for-
mation. In many instances fluid resuscitation is delayed
and the corresponding period of relative ischemia causes
further release of inflammatory cytokines that aggravate
edema following restoration of the circulating volume.\textsuperscript{14}

Actual peak edema develops in the first 24 h post-
burn, but increased permeability persists for 48 hours at
least and can have a direct impact on tissue loss. As mar-
ginal tissues become necrotic, the deepening and exten-
sion of the burn injury, which has been extensively re-
ported, takes place and is seen all too frequently.\textsuperscript{4,14} More-
over, persistent edema within the tissues exacerbates tis-
sue scarring and limits function.\textsuperscript{14}

Over-resuscitation of the severely and multi-injured
patient often results in fluid overload and “resuscitation morbidity” characterized by anasarca, orbital compartment syndrome, extremity compartment syndrome, and abdominal compartment syndrome (ACS) as well as pulmonary edema requiring a prolonged and potentially complicated hospital stay. If not treated promptly and effectively, many of these complications will increase morbidity and mortality.2,3,5

Soft-tissue edema in the extremities necessitates more frequent escharotomies and even fasciotomies.6 Abdominal compartment syndrome remains however the most dramatic and clinically challenging complication directly related to fluid resuscitation volumes.3,6 Several studies have now determined that intra-abdominal hypertension will develop once a threshold of cumulative crystalloid fluid reaches 250 to 350 mL/kg during the acute resuscitation phase.6 More precisely, a resuscitation volume greater than 237 cc/kg over the course of 12 h (16 l during a 12-h period in a 70-kg man) appears to be the threshold for the development of ACS.7 When the burn size exceeds 60% TBSA, resuscitation-related ACS is associated with a mortality of 97%.6

Increased fluid volumes independently increase the risk of pneumonia, bloodstream infections, acute respiratory distress syndrome, multi-organ failure, and death.8 A large percentage of resuscitated patient exceed the Ivy Index, defined as 24 h volumes exceeding 250 mL/kg, a well acknowledged independent predictor of mortality based on multivariate logistic regression.7

Possible etiologies of fluid creep

Recognized nearly a decade ago, fluid creep is a landmine in modern burn care.2,6 In contrast to earlier reports documenting increased fluid requirements for exceptional patients, recent publications have reported much higher crystalloid fluid requirements for resuscitation of a majority of routine patients with major burn injuries.8 The reasons for this phenomenon are still unclear; they are almost certainly multiple.3,6

Prompt institution of fluid resuscitation is certainly an important contributor to improved survival after extensive burn injury. Unfortunately, first responders and inexperienced physicians often greatly overestimate burn size and sometimes run intravenous infusions wide open. Patients often arrive at burn centers after receiving significantly large amounts of crystalloid and much of their first 8-hour Parkland requirements in just an hour or two because of inaccurate estimations of burn size or overzealous or inattentive resuscitation.4,13 Excessive initial resuscitation is a likely contributor to fluid creep that may not be apparent until much later.4 As described recently, “fluid begets more fluid”; the higher the starting point in terms of initial fluid rate, the higher the final 24-h resuscitation volume.10,27

Compared to smaller injuries, large burn injuries require disproportionately more fluid for resuscitation. A few decades ago, it was noticed that the Parkland formula was not accurate in predicting resuscitation fluid requirements for such injuries. Failed resuscitation was the norm and patients with the largest injuries were most likely to die. At present, current mortality from extensive injuries is at an all-time low. Many severely burned patients do in fact survive following aggressive resuscitation well beyond the estimates of the Parkland formula. This may have encouraged practitioners to apply the same overzealous resuscitation to patients with less severe injuries favoring fluid over-loading.5

It is clear that burn care practitioners have mastered the indications for increasing the rate of fluid administration in the face of inadequate (<30 ml/h) urine output (UOP).3,6,11 However, clinicians and burn unit staff members are less likely to reduce fluid infusions in timely manner whenever UOP exceeds 50ml/h. It seems that they are permitting resuscitation to escape their control through lack of attention or carelessness.5,4 In fact, burn clinicians frequently allow UOP to drift above what should be a tightly controlled range of 0.5 to 1 ml/kg/h.16 In one study, the LR infusion rate was appropriately reduced in 35% of the time only.16

Opiates are the mainstay of present pain control in burn patients. With better pain management over the past decade, it appears that opioid dosage correlates with fluid requirements and that fluid creep is a consequence of the increasing use of narcotics during initial burn care, a phenomenon referred to as “opioid creep”.3,6,8 Administration of morphine to critically ill patients certainly partially antagonizes the adrenergically mediated cardiovascular response to stress, but it is unlikely that the use of opiates alone can account for the dramatic magnitude of fluid creep observed in recent years.8

Since the introduction of the Parkland formula, UOP has been the “gold standard” for determining the adequacy of fluid resuscitation. A decline in UOP is an almost universal indication to increase intravenous fluids.7 The value of UOP as an accurate and sole indicator of appropriate fluid resuscitation has however been disputed over the past two decades. In an attempt to tailor resuscitation to achieve both the normalization of base deficit (BD) and lactic acid (LA) levels and, at the same time, above normal levels of cardiac index (CI) and oxygen delivery (DO2) and/or consumption (VO2), practitioners have shifted to a “goal-directed” therapy, even when vital signs and urine output are adequate. This in turn dictated increased volumes of fluid and blood, up to 56% of what may be predicted by the Parkland formula. Certain reports have even noted that the administration of larger quantities of fluids - as much as four times Parkland predictions, was necessary to normalize CI and/or VO2.2,8
Without being superior to resuscitation based on standard clinical parameters, goal-directed resuscitation is associated with a higher incidence of ACS.\textsuperscript{6,16} However, although urine flow is a useful clinical variable to follow, excessive and exclusive tracking of it to titrate the rate of fluid infusion can be a deadly pitfall.\textsuperscript{11} In overhydrated severely burned patients, a decreased UOP may reflect over-resuscitation and the onset of abdominal compartment syndrome.\textsuperscript{4} It is obvious that unnecessary goal-directed resuscitation may significantly contribute to fluid creep in many situations, but fails to explain the tendency for fluid creep to persist despite attempts to reduce fluid infusions.\textsuperscript{6}

Solid evidence supports the fact that excessive administration of crystalloid and the abandonment of colloid replenishment at some point of resuscitation are major contributors to fluid creep.\textsuperscript{4} In fact, fluid creep may be considered an iatrogenic phenomenon resulting from misuse of the originally described approaches to crystalloid resuscitation by Baxter, who anticipated colloid infusion in the fourth 8-h period post-burn.\textsuperscript{16} Departure from the original Baxter formula may help explain the occurrence of fluid creep.\textsuperscript{4}

As summarized by Saffle,\textsuperscript{3} with the onset of increased capillary permeability immediately following burn injury, the initial leakage of proteins largely eliminates the oncotic pressure gradient, favoring fluid flux from the intravascular compartment. This is paralleled by a disruption of the “safety valve” against edema formation of the densely configured collagen-hyaluronate interstitial matrix, which increases interstitial compliance and generates osmotically active fragments as well as negative “sucking” interstitial pressure, facilitating rapid fluid sequestration. Despite the neutralization of the gradient within a few hours, as interstitial gel is hydrated, compliance continues to increase, allowing ongoing accumulation of fluid with little change in hydrostatic pressure.\textsuperscript{6,19} Any excessive fluid given in the early post-burn period would thus increase capillary hydrostatic pressure and further reduce oncotic pressure, both contributing to a cycle of accelerated capillary leakage requiring ever-greater amounts of crystalloid infusion to satisfy. This is probably why fluid requirements escalate to volumes far in excess of Parkland calculations, seemingly without limit, as documented in some recent reports. It seems that fluid creep becomes self-perpetuating and creates its own physiology of edema formation.\textsuperscript{4} This mechanism could explain why fluid creep is prominently manifested by edema in unburned tissues such as the abdomen. It also explains why paradoxically fluid requirements are usually fairly close to Parkland predictions for the first 8 h post-injury when capillary leakage should be greatest, only to become increasingly problematic after this period.\textsuperscript{6}

Control of crystalloid fluid infusion

As it is obvious that large volumes of crystalloid determine fluid creep occurrence, adequate titration of fluids used for acute burn resuscitation is probably the first step necessary to prevent this complication.

Regulation of resuscitation fluids as soon as possible after injury is essential.\textsuperscript{6} Fluid requirements may fall below Parkland predictions for the first few hours after injury and infusions can often be reduced during this phase.\textsuperscript{4} Close communication with first responders and referring physicians, if possible including telemedicine among other modalities, is essential and helps in the pacing of resuscitation as soon as possible after injury.\textsuperscript{6} Using widely accessible programs for calculating burn size and fluid requirements may help also to reduce over-resuscitation.\textsuperscript{6}

Attempts to titrate fluid infusion based on only a few hours of urinary output information, however, may not be altogether straightforward.\textsuperscript{6} The fine balance between too little or too much fluid to achieve adequate urine output without polyuria while limiting hypoperfusion or fluid creep in burn patients is hard to maintain and requires clinicians with extensive burn experience.\textsuperscript{11} With increasing urine flow and indices of clinical stability, a decrease in the rate of fluid administration is mandatory. With adequate UOP for two consecutive hours, decreasing the intravenous fluid rate by 10% is usually recommended. Decreasing the rate of fluid administration at an arbitrary time interval, as is described with many formulas, had better be avoided if a slow drift back of the patient into shock is to be prevented.\textsuperscript{11}

In order to reduce the dependence on clinical decision-making, there is a definite need to establish clinical guidelines for the timely reduction in the rate of resuscitation fluid infusion.\textsuperscript{6} Established algorithms, such as with nurse-driven resuscitation protocols, may have better reinforcement of downward titration of fluid volumes when urine output is high, and may even allow for reductions in infusion rates when urine output is adequate.\textsuperscript{3}

In order to avoid the element of human error, computerized decision support is proving to be a promising approach.\textsuperscript{4,6,10,19} The computer decision support system for burn resuscitation is based on an algorithm that defines a response (fluid administration) to a data input (urine production) and is the basis for a new closed loop concept for burn resuscitation.\textsuperscript{4} It proved to be more accurate than technician-run resuscitation in an experimental model algorithm and was recently adopted at the US Army Institute of Surgical Research Burn Center.\textsuperscript{4,6,10,19} The system improved fluid management as well as patient care of severely burned patients. All measures of crystalloid fluid volume could be reduced while maintaining patients within urinary output targets most of the time.\textsuperscript{19} Research on
future systems capable of dynamically adapting to changing situations is ongoing.4

**Colloid rescue and primary colloid resuscitation**

Controversy regarding the practicality and accuracy of the Parkland burn formula has persisted since its introduction more than three decades ago, and debate about the appropriate acute burn resuscitation, including the amount and type of fluid to be administered, still continues.20 At present, mean fluid resuscitation of 6 ml/kg/% burn is the norm and only 13% of patients undergoing crystalloid resuscitation are within the Parkland formula.20 Historically, prevailing opinion warned against the use of colloid in the first 24 h of resuscitation, on the assumption that colloid would pass through the “leaky” capillaries in burn shock and exert an osmotic pull, drawing even more fluid into the interstitial space and worsening burn edema.3

As mentioned earlier, Baxter recommended that 20% to 60% of circulating plasma volume needed to be replenished with colloid during the second 24 h post-burn.1,14 This is an underemphasized component in the consensus Parkland formula subsequently adopted. Colloid has been totally omitted from standard resuscitation.22 Colloid, whether given as plasma, albumin, or hetastarch, is significantly more expensive than crystalloid. Debate about its cost-effectiveness for fluid resuscitation is still not resolved.3 Moreover, a longstanding belief supported by early studies that colloid administration is associated with increased mortality among burn patients still persists. Current prejudice against the use of colloid is probably not justified.22 Reported higher mortality rates among patients who receive colloid, though based on studies regarded as highly authoritative, could possibly be attributed to the fact that patients included in these studies were often more critically ill or injured and required more intensive fluid management. Their mortality, therefore, cannot only be attributed to the use of colloid but rather to a combination of multiple factors and co-morbidities. These reports have been criticized for being based largely on unblinded and heterogeneous studies.23

The odds ratio for mortality with albumin usage for resuscitation in a variety of situations, including burn patients, has been calculated from several meta-analyses to be as high as 240 with 95% confidence.23 Valid studies with a high level of evidence involving burn patients are in fact very rare. Only one small trial conducted some two decades ago was identified by a Cochrane review. Even though it showed that colloid-resuscitated patients required less fluid than those who received crystalloid alone (298 vs 381 ml/kg/% TBSA), it also demonstrated progressive increase in lung edema up to 7 days post-burn associated with higher mortality.22 Though patients in that particular study died of causes not obviously related to fluid resuscitation, it has influenced thinking about burn resuscitation for more than two decades and has contributed, perhaps excessively, to the perpetuation of the prejudice against colloid burn resuscitation. A recent multicenter trial in almost 7,000 intensive care unit patients about routine albumin use for resuscitation did not demonstrate any increased risk of death or other adverse outcomes. A more recent randomized trial involving burn patients yielded the same conclusion and revealed no difference in rates of multiple organ failure.2,26 Although these reports have refuted the assumption that colloid administration for burn resuscitation is harmful, they have failed to provide clear data that colloid improves survival rates.2,26 Lack of definitive data on these issues has revived the debate over the real value of colloid-based resuscitation.3

It is obvious now that restoring cardiac preload during the period of burn shock by pure crystalloid resuscitation is not possible - a finding reached by Baxter and others 25 years earlier but left unrecognized.3 Regardless of the resuscitation formula used, restoration of preload and cardiac function and resolution of acidosis appear to require 24 to 48 h to occur. “Pushing” these parameters to specific “supraphysiologic” endpoints with increased preload or inotropes greatly increases fluid requirements without obvious improvements in outcome.4

Depletion of plasma proteins alone can mimic burn edema, and infusions of albumin or dextran can almost completely prevent edema in unburned tissues.5 At present the role of colloids in the resuscitation of burn patients is being re-examined.17 Several recent studies report reduced fluid requirements when colloids are used.6,7 Albumin administration rapidly reduces hourly fluid requirements, restores normal input/output (I/O) ratios, and ameliorates fluid creep in pediatric as well as adult burn patients.14,17 Albumin was not only safe but was shown to actually confer a mortality benefit.7

Control of fluid overload with the inclusion of colloid in the burn resuscitation strategy has been consistently demonstrated even though no outcome benefit has been proven so far.15 By increasing capillary osmotic pressure, fluid is retained in the intravascular space, limiting fluid flux to the interstitium.7 It must nevertheless be recognized that the use of colloids will prove to be of benefit only if normal capillary permeability is restored, either spontaneously or potentially with anti-histamine, anti-prostaglandins, and other anti-vasoactive mediators; otherwise the infused colloid will not remain in the intravascular space and will not increase intravascular osmotic pressure.13

One potential approach to controlling fluid creep, therefore, would be to adhere to the original Parkland formula and administer a colloid bolus 24 h post-burn. Alternatively, colloids may be administered to patients who develop increasing fluid requirements during resuscitation, as
a "rescue" from fluid creep. Some have advocated resuscitation with albumin at 12 h post-burn when fluid requirements exceed 120% of normal or when the projected 24-h resuscitation exceeds 6 ml/kg/h near the 12-hour mark.\textsuperscript{4,6,17,18} Patients requiring more fluid volumes than predicted by the formula may also get a third of their hourly fluid volumes as 5% albumin, with the other two-thirds given as LR solution.\textsuperscript{3}

Fresh frozen plasma is probably the best colloid solution available for acute burn resuscitation, particularly whenever there is a serious coagulopathy risk. Plasma, however, carries a biological risk for disease transmission as recommended by Baxter, the impact of goal-directed resuscitation, and the overzealous on the scene crystalloid resuscitation combined with subsequent inefficient titration of fluid administration and lack of timely reduction of infusion rates, have all contributed to the phenomenon of fluid creep that has been recognized only recently.\textsuperscript{4}

Fluid resuscitation is evidently a clinical management problem. Since the amount and type of fluid needed may be influenced by a myriad of confounding variables, developing one formula applicable to all situations is virtually impossible.\textsuperscript{1} It is, however, essential to determine the appropriate combination of fluids needed and, most importantly, to establish clinical guidelines for the timely increase or reduction in the rate at which these fluids are being given. Reduction of the rate of administered fluid without delay, as soon as the patient becomes clinically stable and particularly when urine flow increases cannot be overemphasized.\textsuperscript{11}

Although the exact causes of fluid creep remain undetermined, controlling its magnitude and complications certainly requires several strategies, which may include restriction of early fluid resuscitation, tighter titration of fluid administration, colloid administration, and possibly the use of adjunctive pharmacologic agents as well as markers of resuscitation other than urinary output.\textsuperscript{6,15} The results of a recently conducted systematic review of burn edema were really surprising. A paucity was noted of good quality studies providing high level evidence: the authors were in fact unable to identify any study reporting definitive outcomes that was not likely to have been biased in some fashion because of lack of reporting or methodological flaws.\textsuperscript{16} Lack of definitive evidence to support consensus in fluid regimen or outcome measurement continues to be a topic of debate.\textsuperscript{11,24,25}

It may be useful at this point to reconsider the principles of burn resuscitation and to reassess current practice protocols.\textsuperscript{6} Crystalloid solutions alone may be successfully used to acutely resuscitate most uncomplicated patients with burns up to 20 to 25\% TBSA despite the fact that a degree of hypoproteinemia does occur in these patients. Conversely, with more extensive and deeper burns, and whenever there is crystalloid alone becomes increasingly difficult.\textsuperscript{11} In such patients, there is at present a clear trend towards increased use of colloids in the early phases to diminish the effect of hypoproteinemia, with albumin as the main colloid receiving attention.\textsuperscript{11,15}

\textbf{Conclusion}

It is clear now that successful resuscitation can be accomplished with lower initial fluid volumes.\textsuperscript{11} Moreover, four decades later, we are rediscovering what Baxter has initially described. Colloids seem to be an essential component of the acute resuscitation protocol of severely burned patients. Despite some reservation with the use of albumin in the early phases of burn resuscitation, recent work by Cochran et al.\textsuperscript{26} demonstrated a decreased likelihood of death. Encouraging results have also been reported with the use of albumin as early as 12 h post-burn in those who are predicted to have a higher resuscitation volume\textsuperscript{17,27} as well as in the elderly burn patients.\textsuperscript{28} However, certain aspects still need clarification: the indications for using this method, the exact time of initiation, the duration and volume required, plus the question whether colloid solution should be used primarily or reserved as a rescue strategy.

\textbf{RESUME.} La surcharge liquidienne est devenue un phénomène d’importance mondiale dans le secteur des soins aux patients brûlés. La formule concordée de Parkland qui a éliminé l’utilisation des colloïdes, l’impact de la réanimation axée sur l’objectif et l’excès de zèle pour la réanimation sur place avec l’utilisation des cristalloïdes, unis à la successive titration inefficace de l’administration des fluides et à l’absence d’une réduction rapide du taux de perfusion, ont tous contribué à ce phénomène de surcharge liquidienne (en anglais, « fluid creep »), reconnu seulement récemment, qui aujourd’hui est comme une mine antipersonnel dans le monde moderne des soins aux patients brûlés. Il y a des preuves bien solides dans la littérature que l’administration excessive de colloïdes et l’abandon de la reconstitution colloïdale à un certain moment au cours de la réanimation sont les causes principales de la surcharge liquidienne. Sur la base des preuves disponibles, nous présentons un examen exhaustif de la littérature qui s’occupe de la surcharge liquidienne dans le but de déterminer l’étiologie sous-jacente et de proposer des stratégies pour contrôler son ampleur et les complications qui en dérivent, notamment - parmi les autres options - en utilisant les colloïdes.

\textbf{Mots-clés:} brûlures aiguës, fluid creep, réanimation post-brûlure
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