Introduction

Acute kidney injury (AKI) is one of the most common serious complications in critically ill patients. Severe AKI occurs in more than one of every twenty patients requiring intensive care unit (ICU) care, and has been associated with mortality rates ranging from 50% to more than 70%. In burn patients, AKI is a growing health concern as it is associated with both short and long term adverse events. These frequently lead to extended intensive care unit stays and high mortality rates. Although kidney function returns to normal for most burn survivors, a minority require long-term dialysis. Despite decades of research on the etiopathogenesis of AKI in thermal injury, the treatment of this entity is still not well defined; therefore, prevention and early diagnosis are key to avoid the unfavorable prognosis of AKI. These entail a comprehensive understanding of the global physiologic changes underlying the condition of burn patients and a judicious interpretation of their continuous homeostatic alterations. The aim of this review is to present the salient features in burn patient physiology that contribute to AKI. Strategies for identifying early AKI are presented. Finally, the different treatment modalities are revisited.

Definition

Over the past few decades, definitions used to describe AKI have varied widely; however, the common underlying theme is an abrupt reduction in glomerular filtration rate (GFR) with failure of the kidneys to regulate volume and electrolyte homeostasis. In an effort to standardize the definition of renal insufficiency, the International Acute Dialysis Quality Initiative (ADQI) group developed the RIFLE criteria (Table I). This classification system relies on a reduction in GFR or urine output as a means to define increasing levels of renal insufficiency. There are three grades of increasing severity of renal insufficiency (Risk, Injury, Failure) based on changes in either serum creatinine or urine output, as well as two outcome categories (Loss and End-stage kidney disease). These high risk patients require a comprehensive understanding of the pathophysiology of renal dysfunction in burns. The aim of the present article is to review the salient features underlying the etiology and pathogenesis of AKI associated with thermal injury and to summarize the efficacy of the available diagnostic and treatment modalities.
setting since AKI is defined as an abrupt decrease in kidney function (<48 h). Currently there is no evidence to support the use of one system over the other; nevertheless, these two consensus definitions have facilitated comparisons between studies analyzing AKI in burn injuries.

### Etiology

Multiple conditions contribute to early AKI (first 24 h) in the burn patient: hypovolemia, cardiac dysfunction, release of inflammatory mediators and denatured proteins (from extensive tissue destruction), and nephrotoxic drugs. Late AKI usually falls within the multi-organ dysfunction syndrome (MODS) frequently associated with severe sepsis. Hypovolemia and under-resuscitation have classically been thought of as the primary causes of early AKI; however, recent studies suggest that AKI can develop despite adequate resuscitation.

Furthermore, recent studies in critically ill patients, including burn patients, have suggested that a positive fluid balance may have a negative influence on kidney function and mortality. All these observations suggest that AKI is more likely dependent on the degree of shock caused by the initial injury, and the subsequent release of injurious inflammatory mediators. Global indicators of tissue perfusion (i.e. lactate and base deficit) may be adequate predictors of AKI in this setting, as they have been shown to predict increased risk of systemic inflammatory response syndrome (SIRS), acute respiratory distress syndrome (ARDS), multi-organ dysfunction syndrome (MODS), and mortality. Base deficit may also be a better marker of poor perfusion than serum lactate because it represents the combined sum of lactate and all other metabolic acids released during tissue hypoxia.

### Table 1 - RIFLE & AKIN criteria: Increase: ↑; decrease: ↓

<table>
<thead>
<tr>
<th>Kidney dysfunction</th>
<th>RIFLE criteria</th>
<th>AKIN criteria</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Parameter</strong></td>
<td>Risk</td>
<td>Injury</td>
</tr>
<tr>
<td>Serum creatinine (Cr)</td>
<td>↑ in Cr ≥1.5X baseline Or ↓ in GFR ≥25%</td>
<td>↑ in Cr ≥2.0X baseline Or ↓ in GFR ≥50%</td>
</tr>
<tr>
<td>Urine output</td>
<td>&lt;0.5 mL/kg/h for ≥6 h</td>
<td>&lt;0.5 mL/kg/h for ≥12 h</td>
</tr>
</tbody>
</table>

### Hypovolemia and Abdominal Compartment Syndrome

Following thermal injury plasma losses are in excess of 4 ml per kg of body weight per h in a burn exceeding 30% TBSA. This is sufficient to cause decreased renal perfusion. Kim et al. observed that burn size was an independent predictor of acute renal failure in the burn population. This depressed renal blood flow, which is compounded by local and systemic cytokine storms, and results in ischemia, cellular death, and subsequent release of oxygen free radicals. These mediators cause direct tubular damage and disrupt tight junctions, leading to obstructive nephropathy which further reduces GFR. The ischemia time is a critical determinant of AKI occurrence. Aggressive and early fluid replacement protects from renal failure. The Shriners Burn Institute for Children (Galveston) showed that the time to fluid resuscitation initiation was directly related to the incidence of renal dysfunction and overall mortality. They advocated early aggressive fluid resuscitation which might prevent renal injury and hence improve overall outcomes.

While older data clearly demonstrated an association between renal failure and a delay in resuscitation, more recent data suggest that aggressive fluid replacement does not eliminate the occurrence of AKI. AKI can develop in burn patients despite normal to average urine output (0.5-1.0 ml/kg/h), and even when fluid resuscitation exceeds the Parkland formula recommendations. In fact, it has become clear that during resuscitation of patients with thermal trauma, parameters such as urine output and mean arterial pressure may not independently accurately reflect perfusion of organs at the cellular level. A state of poor perfusion can exist despite acceptable urine output and blood pressure chartings, and the use of urine output...
alone as a primary measure of adequacy of resuscitation is not recommended by the authors. Klein et al. recently reviewed data from the Glue Grant and found that the majority of patients with AKI were adequately resuscitated; early AKI was therefore probably the result of the inflammatory mediators storm.57,58 Conversely, the risk of over-resuscitation and fluid creep exists and warrants close monitoring.7 Over-resuscitation increases the risk of pneumonia, pulmonary edema, ARDS, and compartment syndromes (orbital, abdominal, extremity) and ultimately contributes to an increase in mortality.59-61 In spite of all efforts to monitor endpoints of resuscitation, intercompartmental fluid shifts occur,62 and these can be especially hazardous if involving fascial bound compartments such as the peritoneal cavity. Intra-abdominal hypertension (IAH) then leads to splanchnic edema with subsequent increase in gut permeability and bacterial translocation. IAH, as defined by an intra-abdominal pressure (IAP) > 12 mmHg, has numerous adverse effects on visceral perfusion,63-65 and the occurrence of IAH in critically ill patients is an independent predictor of increased mortality.66 In the thermally injured patient, abdominal compartment syndrome (ACS) is defined as an IAP > 20 mmHg with at least one concomitant organ failure.67-69 It has been suggested that a crystalloid rate over 20 ml/kg/h during the initial 24 h of resuscitation should alert physicians to possible IAH/ACS.69 There should be close monitoring for the signs of ACS (decreased cardiac output, decreased lung compliance, decreased urine output) in all massive burn patients. In general, as resuscitation volumes approach 6 ml/kg/h, or as signs of IAH arise, the addition of colloid, in the form of albumin, has been shown to decrease fluid requirements and edema.60,61 A “permissive hypovolemia” approach to resuscitation after severe burns was advocated, and may be highly appropriate in certain cases since it was associated with significantly lower multiple-organ dysfunction score (MODS) than resuscitation following the Parkland formula.70 Considering the negative impact on kidney perfusion of under-resuscitation (ischemic injury) and over-resuscitation (fluid creep and ACS), burn surgeons need to personalize fluid therapy according to the evolution of the patient’s general physiologic condition.

Cardiac dysfunction

Burn injury produces substantial hemodynamic and cardiodynamic derangements;72 compromised cardiac function results in global hypoperfusion and reduced renal blood flow contributing to AKI.73 While it was previously suggested that depressed cardiovascular function in burn shock was primarily a sequel of vascular fluid loss,74 recent data point toward direct myocardial suppression, possibly caused by elevated plasma levels of catecholamines, vasopressin, angiotensin-II, neuropeptide-Y,75,76 and various cytokines (TNF-α, IL-1β, IL-2, IL-6 and IFN-gamma).77-83 This occurs despite adequate fluid resuscitation (assessed by normal central venous pressure, pulmonary capillary wedge pressure, and mean arterial pressure).84-85 In addition, large burn areas (>50% TBSA) may result in cardiac infections from the wounded skin, further compromising organ perfusion. The incidence of bacterial endocarditis in such patients is six times higher than in the general population, with mortality rates reaching up to 95%.86 Burn surgeons should be aware of all these physiologic responses when working towards correcting the preload and re-establishing renal blood flow.

Denatured proteins

Muscle breakdown and the release of denatured proteins are implicated in the development of AKI. Rhabdomyolysis in particular contributes to AKI following severe burns,87 caused by direct thermal injury, compartment syndrome, or severe electrical injury. The release of myoglobin and free hemoglobin results in the blockage of renal tubules, constriction of afferent arterioles, and the generation of oxygen free radicals.88 Myoglobinuria occurs when serum myoglobin is greater than 1,500-3,000 ng/ml and is typically associated with elevated levels of creatine kinase (CK). AKI has been shown to be associated with CK levels in excess of 5,000 U/L. Fortunately the incidence of denatured proteins causing AKI is low, and the overall prognosis is favorable when appropriate therapy, in the form of hydration with isotonic crystalloids, is initiated in a timely fashion.77

Sepsis

Sepsis and septic shock are the most common causes of death in the ICU and are observed in up to 87% of burn patients suffering AKI.77,78 The extent of sepsis is directly related to the incidence of AKI.79-81 The etiology of AKI in sepsis is multifactorial. It can be thought to occur as a result of three pathological processes that result from alterations in the homeostatic balance, between production and inactivation, of inflammatory mediators: direct endothelial damage, vasoparalysis, and a procoagulant state. Vasoparalysis results in a state of severe hypotension and decreased tissue perfusion. This is followed by activation of the neurohumoral axis and an increase in plasma levels of catecholamines, vasopressin, and angiotensin-II in an effort to increase in cardiac output and restore normal tissue perfusion.82 However, this also results in direct renal arteriolar vasoconstriction, which may cause a pre-renal state compromising renal perfusion. This is further compounded by the release of vasoconstrictive agents (TNF, endothelin).83-85 With respect to the pro-coagulant state, sepsis is known to upregulate the expression of complement and to enhance the fibrinolytic cascade.86-88 This may lead to disseminated intravascular coagulation and subsequent direct injury to glomeruli by microthrombi.89 Acute tubular necrosis ensues
Diagnosis

Diagnosing AKI in burn patients requires a comprehensive understanding of the changes underlying the physiology of burn patients throughout their course of treatment. Renal injury may occur despite normal renal parameters (urine output, biochemical markers, etc.) and burn surgeons must therefore constantly monitor their patients’ global physiologic picture in an effort to anticipate any sign pointing to early renal injury. A stepwise approach to the diagnosis and treatment of AKI should be implemented.

Urine output

A decrease in urine output is probably the first and most obvious sign of renal dysfunction. This parameter has a high specificity but a low sensitivity. Furthermore, several non-GFR determinants of creatinine (e.g., muscle mass and diet) bias GFR estimates and result in misclassification. The illness itself, due to the resulting non steady physiologic state and muscle wasting, seems to considerably bias the use of serum creatinine as a marker for GFR since plasma creatinine concentration depends on its clearance by the kidney but also varies according to its total production and volume of distribution within the body. In critically ill patients, changes in serum creatinine typically lag behind the timing of renal injury and recovery, and it is therefore more appropriate to consider variations in serum creatinine over change in

secondary to the induced ischemic injury.

Urine microscopy and chemistry

Although a decrease in urine output may be indicative of an underlying renal dysfunction, it does not specify etiology. This can be established by microscopic and biochemical analysis of the urine, which can be used to support the diagnosis of AKI and guide treatment options. A combination of normal urinary sediment and hyaline casts and an oliguric/anuric state suggests a pre-renal cause. The presence of epithelial casts with tubular epithelial cells is pathognomonic for acute tubular necrosis. Pigmented casts on microscopy point to myoglobinuria (most likely due to rhabdomyolysis). It is worth noting that urinary electrolytes also carry a diagnostic potential and should be obtained when microscopy is not diagnostic. Urine electrolytes are also beneficial in differentiating between the pre-renal and renal forms of AKI. For this purpose, the fractional excretion of sodium (FENa) is calculated and interpreted.

The underlying concept is that a pre-renal state in the presence of a normally functioning nephron causes increased absorption of sodium. FENa = (urine sodium X plasma creatinine)/(plasma sodium X urinary creatinine). A value < 1% is associated with pre-renal injury while a value > 1% suggests intrinsic renal injury. There are, however, certain instances when renal absorption of sodium is impaired, and hence FENa would not be a reliable discriminating test between pre-renal azotemia and renal injury.

Numerous reports of low FENa (< 1%) have appeared in various clinical settings of oliguric and nonoliguric states such as acute tubular necrosis, urinary tract obstruction, acute glomerulonephritis, hepatorenal syndrome, renal allograft rejection, sepsis, and drug-related alterations in renal hemodynamics. In this situation, one particular urinary index cannot be used to differentiate between pre-renal azotemia and acute renal failure. An additional modality, such as urea excretion, may be appropriate. Recently, it was suggested that fractional excretion of urea (FEUrea < 35) may have more specificity and sensitivity than FENa in discriminating between pre-renal and renal azotemia.

Plasma creatinine

A true biomarker for AKI is needed; a signature molecule (found in blood or urine) that signals the presence of early renal injury, detects the nephron segment mostly affected, identifies the best drug or fluids therapy, measures the progress of renal function, and can be rapidly and easily measured. At present, however, such an ideal biomarker does not exist and creatinine-based estimated GFR (eGFRcreatinine) is considered the key measure of kidney function in clinical practice. Creatinine is freely filtered across the kidney and is neither reabsorbed nor metabolized. The Chronic Kidney Disease Collaboration (CKD-EPI) creatinine equation was used to estimate eGFRcreatinine, as follows:

\[ eGFR_{creatinine} = 141 \times \left( \frac{\text{minimum of standardized serum creatinine mg/dl} / \kappa \text{ or } 1}{1} \right) \times \left( \frac{\text{maximum of standardized serum creatinine mg/dl} / \kappa \text{ or } 1}{1.018 \text{ if female } \times (1.159 \text{ if black}) \times 0.993^{0.01} \times (1.018 \text{ if female }) \times (1.159 \text{ if black})} \right) \]

where \( \kappa \) is 0.7 if female and 0.9 if male and \( \alpha \) is −0.329 if female and −0.411 if male.

It should be noted that creatinine clearance often overestimates the true GFR by 10-20%; this is because the renal tubules secrete a small quantity of creatinine in the urine which adds to the amount filtered by the glomeruli. Furthermore, several non-GFR determinants of creatinine (e.g., muscle mass and diet) bias GFR estimates and result in misclassification. The illness itself, due to the resulting non steady physiologic state and muscle wasting, seems to considerably bias the use of serum creatinine as a marker for GFR since plasma creatinine concentration depends on its clearance by the kidney but also varies according to its total production and volume of distribution within the body. In critically ill patients, changes in serum creatinine typically lag behind the timing of renal injury and recovery, and it is therefore more appropriate to consider variations in serum creatinine over change in
time. Moran and Myers demonstrated this concept in a computerized model of creatinine kinetics in patients with post-ischemic acute renal failure. They showed that the relationship between creatinine clearance and plasma creatinine concentration was not always inversely proportional and that it dissociated in conditions of acute renal failure and recovery. In other words, a rising serum creatinine is not always a reliable indicator of deteriorating kidney function; conversely, a decreasing serum creatinine does not always reflect an improving GFR. What appeared to be of prognostic value was the number of days plasma creatinine continued to rise following the initial ischemic injury. Day 4 post-injury seems to be a point of no return; that is, a continued increase in serum creatinine beyond day 4 indicates that renal recovery has not begun and a severe protracted course of renal failure is likely to follow. Although this study was not conducted in the burn population, it parallels key points in the ischemic pathophysiology of renal failure, in particular early renal failure.

Physicians caring for burn patients should understand the limitations of any biologic molecule estimating GFR during renal injury. Serum creatinine is currently regarded as the “gold standard”, but this is not an “ideal marker”. GFR estimations based on creatinine clearance should be calculated over short time intervals with the serum creatinine value reflecting the central tendency of the values obtained at the beginning and end of the collection interval. The search for the ideal biomarker or combination of markers to predict kidney function is an area of substantial interest. Cystatin C for example, an alternate marker of kidney function, was shown to estimate GFR as accurately as serum creatinine in a large sample of chronic kidney disease patients. Additional data also suggested that eGFR by Cystatin C (eGFR<sub>creatinine</sub>) had a stronger association with mortality and cardiovascular disease than eGFR<sub>creatinine</sub><sup>94-101</sup>

### Treatment of acute renal failure

As outlined above, several studies have shown that AKI is associated with an increase in morbidity and mortality in critically ill and hospitalized patients. Few therapeutic interventions, however, have been successful in treating or preventing AKI, possibly owing to delayed diagnosis. Patients at risk for AKI, or those with AKI, require careful attention to hemodynamic and general physiologic status. The key to adequate AKI treatment is early diagnosis and rapid termination of the underlying insult while preserving renal function and preventing iatrogenic injury. When conservative measures fail, RRT is required. This section will re-examine measures for the prevention of AKI, and will outline current evidence regarding the use of RRT initiation.

**Early AKI**

Early AKI may be caused by hypovolemia, leading to a decrease in renal perfusion and resultant renal injury. Several studies have advocated early fluid administration to prevent or minimize the effects of AKI. In severe sepsis and septic shock, the administration of intravenous fluids and vasopressors in the first hours of an acute critical illness have been considered to be among the most important interventions toward improved outcomes. Several formulas have been suggested for optimizing resuscitation - which one to follow is not crucial. The key is to recognize that these formulas estimate the fluid requirements over a period of time. The true immediate amount depends directly on the patient’s general physiologic status and the extent of injury. As with any type of traumatic human insult, indicators of regional and global body perfusion should be continuously monitored and used to assess the adequacy of resuscitation. When the assessment of renal perfusion becomes challenging, invasive modalities for monitoring central pressures and global related volume variables (such as global end diastolic volume, extra vascular lung water volume, intrathoracic blood volume) may be of use.<sup>104</sup> Interestingly, Schiller et al. compared the resuscitation of burn patients to the use of invasive hemodynamic monitoring and a hyperdynamic resuscitation protocol to that of a control group for which resuscitation was guided by traditional end points such as blood pressure, heart rate, and urine output. Patients treated with hyperdynamic resuscitation showed improved microcirculatory flow, tissue perfusion and tissue oxygenation and appeared to have less renal and hepatic dysfunction with a significant reduction in the mortality rate. A statistically significant difference in early hemodynamic response was noted between survivors and non-survivors.<sup>106</sup> The association between improved survival and an early self-generated hyperdynamic response has been previously demonstrated. Whether the use of invasive monitors can actually improve outcome has not been proven conclusively and the use of invasive monitoring carries inherent risks, especially in an immunocompromised host, such as a burn patient.<sup>48,105</sup> Lastly, it is worth reiterating that, while striving to restore an effective circulatory volume, physicians should keep in mind the possibility of early myocardial dysfunction as a cause of decreased renal perfusion and early AKI.

**Late AKI**

Late AKI is viewed as being multifactorial and is often associated with sepsis and multi-organ dysfunction (MODS). The most effective therapy is prevention and early recognition of the septic state.<sup>107</sup> Early sepsis can be suspected on the basis of feeding intolerance, insulin resistance, elevation of acute phase reactants, and many other markers. Source control, identification of the offending organism or organisms, and early directed-goal therapy (EGDT) should
be initiated.\textsuperscript{107} The role of infectious surveillance in the burn patient for a targeted control of offending microbes and prevention of systemic dissemination cannot be overstated.

**RRT overview**

As a result of major advances in burn resuscitation and the management of sepsis,\textsuperscript{108} renal failure requiring RRT is becoming a relatively rare event.\textsuperscript{109} The reported incidence varies between 1 and 3%; however, the overall mortality associated with renal failure requiring RRT can be as high as 80\%\textsuperscript{,17,73,106,111}. 

**Optimal timing**

The issue of when to initiate RRT in patients with AKI has been debated nearly as long as hemodialysis has been part of the armamentarium of clinical medicine.\textsuperscript{12} Although numerous studies over more than a half century have attempted to resolve the issue of optimal timing, the level of evidence guiding current practice remains weak, derived primarily from retrospective and observational cohort studies and small underpowered prospective trials. Suffice it to say that the standard indications for dialysis (volume overload; severe hyperkalemia and metabolic acidosis; overt uremic symptoms) are less relevant in the therapeutically injured patient. A recent consensus advocated the initiation of RRT in critically ill patients with AKI before the development of extreme metabolic derangements and life-threatening events.\textsuperscript{13} Additionally, preliminary evidence suggests that a more aggressive approach to RRT in burn patients may be beneficial.\textsuperscript{112,113}

**Modality of RRT**

Peritoneal dialysis (PD) has long been used as a form of RRT in AKI.\textsuperscript{114} In a burn management setting, this form is limited by clearance rates and the need for catheter insertion through the abdominal wall. In the past two decades, other RRT modalities were examined in the ICU setting, namely intermittent hemodialysis (IHD), continuous and prolonged intermittent RRT (CRRT and PIRRT), and sustained low-efficiency dialysis.\textsuperscript{115} There is no evidence that any single modality of RRT is associated with improved survival or recovery of kidney function, although slower modalities (e.g., CRRT, PIRRT) may be better tolerated in hemodynamically unstable patients and permit achievement of a more negative fluid balance.\textsuperscript{116} An additional potential benefit of continuous hemofiltration is the clearance of pro-inflammatory mediators and therefore the theoretical reduction in the development of multi-organ dysfunction.\textsuperscript{117} Conversely, studies have shown that the cytokine clearances attainable with even high-volume continuous filtration are trivial in comparison to endogenous production, and cytokine removal is nonselective and results in removal of both pro-inflammatory and anti-inflammatory mediators.\textsuperscript{118} In any case, although preliminary (non-prospective) studies with CRRT in the burn patient demonstrated improved survival,\textsuperscript{112,113} future prospective randomized studies are required.

**Intensity of RRT**

It has been suggested that prevention of severe metabolic derangements by earlier initiation of RRT in the critically burned patient may be beneficial. Likewise, prevention or correction of severe metabolic derangements with more intensive RRT has also been suggested. There is no solid evidence, however, that augmented doses of RRT in critically ill patients with AKI are associated with improved outcomes, and most studies examining the impact of more intensive RRT quantified the intensity of therapy based on the clearance of urea (a low molecular-weight solute), ignoring the clearance of higher-molecular-weight solutes and, even more importantly, the management of extracellular volume.\textsuperscript{112,118-121}

**Conclusion**

Significant advances in the management of burn patients have been made over the past four decades. Increasing evidence points to the need for early aggressive fluid repletion (probably with isotonic crystalloids rather than colloids) for expansion of intravascular volume. Urine output is still considered a good indicator for adequate resuscitation. Persistent low urine output invariably indicates inadequate resuscitation but an adequate urine output does not necessarily mean that the burn patient is not developing acute non-oliguric renal failure either due to insufficient resuscitation or to massive release of inflammatory mediators. Moreover, relying on urine output alone could be misleading, resulting in over-resuscitation and fluid creep. Consideration of urine output together with other resuscitation parameters should thus be the standard of care.

In instances where it is necessary to decrease overall fluid requirements, the addition of albumin appears justified. This is best coupled with early burn wound excision (first 72 h) and burn wound closure. While this approach appears to reduce the incidence of early AKI, this disorder may still occur at a later stage following prolonged or delayed initial resuscitation and following sepsis. At this stage, once AKI occurs and hemodynamics are stabilized, the relevance of a restrictive fluid balance and the use of diuretics or RRT to prevent or treat fluid overload and improve outcomes in this population, without worsening kidney function, need to be confirmed with RCTs. Nevertheless, physicians should monitor the complex physiologic state of the burn patient constantly in order to anticipate any condition that may injure the kidneys. This will ensure prompt initiation of appropriate treatment and possibly prevent the development of acute kidney injury. In an era where no pharmacologic agent exists to treat this condition, prevention remains the best (and only) remedy.
RÉSUMÉ. Les lésions rénales aigües (LRA) sont rares, mais elles constituent une complication majeure des brûlures qui mènent souvent à la mortalité. Ces lésions sont provoquées par une interaction complexe de divers changements cellulaires et neuro-humoraux qui affectent les patients brûlés. Les directives pour le traitement de ces patients ne sont pas encore bien définies et, par conséquent, la prévention et le diagnostic précoce sont essentiels pour éviter le pronostic défavorable des LRA. Cela nécessite une compréhension complète des changements physiologiques présents dans ces patients et une interprétation judicieuse de leurs continuelles altérations homéostatiques. Les Auteurs se sont proposé de présenter les principales caractéristiques de la physiologie du patient brûlé qui contribuent à ce type de lésion. Aprés avoir discuté les stratégies pour identifier ces lésions en phase précoce, ils concluent avec une description des différentes modalités de traitement.

Mots-clés: brûlures et insuffisance rénale, brûlures et dysfonction rénale, brûlures et dialyse, classification des lésions rénales, traitement de l’insuffisance rénale

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