Editorial

sb&h

Scars, Burns & Healing
Volume 4: 1–7
DOI: 10.1177/2059513118790658
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Sepsis 3 and the burns patient: do we need Sepsis 3.1?

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Keywords
Burns, definition, sepsis, sepsis 3, SIRS, SOFA

Sepsis definitions and their relevance to the burns patient

Sepsis has been described as 'one of the oldest and most elusive syndromes in medicine'.[1] Since ancient times, the human mind has been able to conceptualise infection and the possibility of its spread from a localised area to the rest of the body.[2] The term first appeared at the time of the ancient Greek culture with a meaning close to that of putrefaction.[2] The construct has since evolved into blood and body *poisoning* from microbial species, with subsequent recognition of the fundamental role of the host response in the development of this complex clinical syndrome.[3,4]

In more recent times, and particularly over the last three decades, the definitions of sepsis and related syndromes (severe sepsis and septic shock) have evolved, with consequences on the diagnostic identification of these conditions and for epidemiological purposes.[5–7] Sepsis is a condition with high mortality risk.

Many factors, such as genetics, age, gender, ethnicity, co-morbid conditions, number of dysfunctional organs and temporal trends in markers of acute physiological derangement have been linked to survival outcome in severe sepsis.[8–12]

Sepsis is a significant cause of additional morbidity and mortality in the burns patient, although most studies conducted on sepsis have excluded burns sufferers.[13] Unsurprisingly, sepsis-related multiple organ failure is often associated with mortality in the burns patient.[14] Many *sepsis-like* clinical manifestations in the burns patient are often *normal or expected* for the burns population, despite the presence of physiological parameters derangement which would normally alert the clinician to the potential for sepsis in other patients' groups. The identification of sepsis in burns patients has required modifications of the standard definitions, due to the peculiarities of this population subset.[15]

To understand the relevance to the adult burns patients of the evolving definitions of sepsis, we review the successive versions of the consensus definitions and consider their applicability specifically to this population.

Sepsis 1, Sepsis 2 and the American Burn Association definitions

A first sepsis consensus definition (Sepsis 1) was developed at a multi-agency conference of 1991, held jointly by experts of the American College of Chest Physicians and the Society of Critical Care Medicine.[5] The aim of this conference was to provide a standardised framework for the sepsis syndromes and enhance their detection and timely treatment, as well as research protocols. Sepsis was defined as *confirmed* infection plus a Systemic Inflammatory Response Syndrome (SIRS), and severe sepsis as sepsis *associated with* organ dysfunction. SIRS was described as a

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physiological response to a triggering factor, infective or not, characterised by at least two of the following features: temperature > 38 °C or < 36 °C, heart rate > 90 beats/min, respiratory rate > 20 breaths/min or partial arterial pressure of carbon dioxide ($PaCO_2$) < 4.3 kPa (32 mmHg), white blood cell count (WCC) > 12,000/mm^3 or < 4000/mm^3 or presenting > 10% immature neutrophils.[5] Septic shock was recognised as 'a subset of severe sepsis and defined as sepsis-induced hypotension, persisting despite adequate fluid resuscitation', hence representing the most severe end within the spectrum of the infective syndromes.[5,16] At the time, the consensus conference had concluded that universally applicable criteria for the detection and quantification of individual organ dysfunction could not be recommended.[5] No guidance was provided on how to establish a causal relationship between sepsis and organ dysfunction, or how to evaluate baseline organ/system physiological status. Furthermore, no clarification was provided about what could be considered as *adequate* fluid resuscitation.[17,18] Consequently, where there was a requirement for definitions of dysfunctional organs (a fundamental element in the definition of severe sepsis), specific *ad hoc* criteria were presented in the relevant study protocols.[19]

Mortality in burn injury patients is related to factors such as age, burn size and presence of inhalation injury, with the leading cause of death being multiple organ failure, estimated to be as high as 27.5% in registry data.[20] In the burns patient, infection usually precedes multi-organ dysfunction and failure, and severe physiological derangement and septic shock have been associated with mortality following burn trauma.[21] The early identification of infection and sepsis, through reliable criteria, supported by an appropriate set of definitions, is therefore equally important in the burns patient as in any other septic patient. Importantly, the SIRS criteria, as described in the Sepsis 1 definitions, are commonly met by patients with extensive burns, even in absence of infection, making them less relevant to the burn population.[15]

The first definition of sepsis was superseded by a subsequent one, subtly but importantly dissimilar, developed in 2001 by representatives of multiple international societies (Society of Critical Care Medicine, European Society of Intensive Care Medicine, American College of Chest Physicians, American Thoracic Society, Surgical Infection Society).[6] While retaining the initial conceptual structure established in 1991, the 2001 set of definitions further expanded the diagnostic criteria for sepsis, across various domains (increasing the SIRS criteria to include a total of 24 parameters classified as general, inflammatory, haemodynamic and tissue perfusion related).[6] One additional important difference is the modification of the requirement for infection from being *confirmed* to *documented or suspected*.[5,6] This modification allows for a wider case capture by the definition, as the clinician only needs to 'suspect' (as opposed to 'confirm') infection. The 2001 definitions also attempted to overcome one of the limitations of the first set, by making direct reference to two different classification systems to quantify the extent of organ dysfunction: the Multiple Organ Dysfunction Score (MODS), developed by Marshall et al.; and the Sepsis-related Organ Failure Assessment score (SOFA) described by Vincent et al.[6,22–24] Hence the emphasis in both the first and the second definitions was on the presence of a confirmed (or suspected) infection plus SIRS by the host, with similar reliance on the fundamental constructs of sepsis, severe sepsis and septic shock.[5,6]

Similar to the septic patient, the patient suffering with severe burn injury is subject to an inflammatory mediators' *storm* while recovering from the condition, until the skin has regained its barrier function, or due to superimposed infection in the same site or elsewhere.[13] A burns patient will display raised heart and respiratory rates, leukocytosis and other features of SIRS, as a consequence of their hyper-metabolic state, independently from presence or otherwise of infection. Furthermore, the burns patient is at increased risk of opportunistic and healthcare-associated infections.[25]

In 2007, the American Burn Association (ABA) convened a consensus conference to define sepsis and infection in burns. According to the resulting ABA definition, all patients with burns surface > 20% of their total body surface area (TBSA) have SIRS. Sepsis in burns patients is also defined relying on specific SIRS criteria (particularly with regards to different temperature, heart and respiratory rates cut-offs, thrombocytopaenia, glucose and feeding intolerance) and a *documented* infection (culture positivity, pathologic tissue source identified or response to antimicrobials). Of note, the ABA consensus conference definitions do not rely on the term *severe sepsis*.[15] When defining organ dysfunction in the context of sepsis in the burns patient, the ABA definition refers to the modified multiple organ

Table 1. Important distinguishing features of the general and ABA consensus definitions of Sepsis and related syndromes.[5–7,15]

	Sepsis 1[5]	Sepsis 2[6]	Sepsis 3[7]	Sepsis (ABA)[15]
Year	1991	2001	2016	2007
SIRS	Two or more of 4 original SIRS criteria	Two or more from the expanded list of SIRS criteria	Not used	Three or more of the modified SIRS criteria or burns surface > 20% TBSA
Sepsis	*Confirmed* infection plus SIRS	*Documented* or *suspected* infection plus expanded SIRS criteria	Infection plus organ dysfunction (life-threatening organ dysfunction caused by a dysregulated host response to infection)	*Documented* infection (culture positive infection or pathologic tissue source identified or clinical response to antimicrobials) plus burns-specific SIRS
Severe sepsis	Sepsis plus organ dysfunction, hypoperfusion, hypotension	Sepsis plus organ dysfunction	Not used	Not used
Sepsis-induced organ dysfunction	Not defined	Reference to MODS and SOFA	Increase by 2 or more SOFA points from baseline	As per Marshall's MODS[22] (modified by Cook[26])
Septic shock	Sepsis-induced hypotension despite adequate fluid resuscitation or need for vasopressors/ inotropes plus perfusion abnormalities	Sepsis-induced hypotension despite adequate fluid resuscitation	Sepsis-induced circulatory and cellular/metabolic abnormalities associated with increased mortality despite adequate fluid resuscitation	Sepsis plus shock-like haemodynamic parameters defined in the '*Sepsis Bundles*' of SSC 2004 guidelines[41]
Adequate fluid resuscitation	Not specifically defined	Not specifically defined	As per definition in SSC 2012[42]	Not specifically defined

ABA, American Burn Association; SIRS, Systemic Inflammatory Response Syndrome; SSC, Surviving Sepsis Campaign.

dysfunction syndrome (MODS) score as modified by Cook et al. Of note, the MODS, like the SOFA score, considers six specific organ dysfunctions (cardiovascular, respiratory, renal, central nervous system, hepatic and haematologic), but does not include, among others, the gastrointestinal system, nor—crucially for the burns patient—the skin.[26]

Sepsis 3 and the burns patient

In 2016, the Sepsis 3 definition was agreed and published, with the specific aim of offering 'greater consistency for epidemiological studies and clinical trials'.[7,27] With Sepsis 3 it was proposed that 'sepsis should be defined as life-threatening organ dysfunction caused by a dysregulated host response to infection. For clinical operationalisation, organ dysfunction can be represented by an increase in the Sequential [Sepsis-related] Organ Failure Assessment (SOFA) score of 2 points or more, which is associated with in-hospital mortality greater than 10%.' The SOFA score was devised by Vincent et al., on behalf of the European Society of Intensive Care Medicine in 1994, with the aim of setting criteria for indicating the level of organ dysfunction, over time, for the various organs/systems identified.[23] The score was developed with the major aims of: first, aiding the comprehension of the natural evolution of organ dysfunction, by quantifying the various organs' improving or deteriorating function with a numerical descriptor; second, describing the relationship between the various failing organs; and, third, evaluating the effects of

Table 2. SIRS criteria as per the general (Sepsis 1 and 2) and ABA consensus definitions.[5,6,15]

Sepsis 1[5]	Sepsis 2[6]	Sepsis (ABA)[15]
Two or more of 4 original SIRS criteria	Some of the SIRS criteria (expanded)	Three or more of the modified SIRS criteria or burns surface > 20% TBSA
• Temp > 38 °C or < 36 °C • HR > 90 bpm • RR > 20 breaths/min or • PaCO2 < 4.3 kPa • WCC > 12,000/mm^3 or < 4000/mm^3 or > 10% immature neutrophils	• Temp > 38.3 °C or < 36 °C • HR > 90 bpm • Tachypnoea or > 2 SD above normal for age • Altered mental status • Significant oedema or positive fluid balance (> 20 mL/kg over 24 h) • Glucose > 120 mg/dL (7.7 mmol/L) no pre-existing DM • WCC > 12,000/mm^3 or < 4000/mm^3 or > 10% immature neutrophils • CRP > 2 SD above normal value • PCT > 2 SD above normal value • SBP < 90 mmHg or MAP < 70 mmHg or SBP decrease > 40 mmHg • SvO2 > 70% • Cardiac index > 3.5 L/min/m^2 • PaO2/FiO2 < 300 • Urine output < 0.5 mL/kg/h • Creatinine increase > 0.5 mg/dL • INR > 1.5 or aPTT > 60 s • Ileus • PLTs < 100,000/μL • Bilirubin > 4 mg/dL (70 mmol/L) • Lacate > 1 mmol/L • Decreased capillary refill or mottling	• Temp > 39 °C or < 36.5 °C • HR > 110 bpm • RR > 25 • Breaths/min (non-ventilated) or MV > 12 L/min (ventilated) • PLTs < 100,000/μL (applies from 3rd day after resuscitation) • Glucose > 200 mg/dL (11.1 mmol/L) or insulin resistance (no pre-existing DM) • Intolerance to enteral feeding for 24 h due to: • abdominal distension; gastric residual volume twice volume of hourly feeding rate; uncontrollable diarrhoea (> 2.5 L/day)

The Sepsis 3 definition did not include SIRS.

Temp, temperature; HR, heart rate; bpm, beats per minute; $PaCO_2$, partial arterial pressure of carbon dioxide (CO_2); WCC, white blood cell count; MV, minute volume; PLTs, platelets; DM, diabetes mellitus; CRP, plasma C-reactive protein; PCT, plasma procalcitonin; SD, standard deviation; SBP, systolic blood pressure; SvO_2, mixed venous oxygen saturation; PaO_2/FiO_2, ratio of arterial oxygen partial pressure to fractional inspired oxygen; Bilirubin, total plasma bilirubin.

therapeutic interventions, by assessing the impact on the relevant organs' function. The score assigns a value in the range of 0–4 for each of the six organ systems assessed (respiratory, coagulation, liver, cardiovascular, central nervous system and renal).[23] The new definitions require that 'septic shock should be defined as a subset of sepsis in which particularly profound circulatory, cellular, and metabolic abnormalities are associated with a greater risk of mortality than with sepsis alone'.[7,27] The document describes how septic shock patients can be identified by detecting a serum lactate level > 2 mmol/L, and the need to use vasopressors to maintain a mean arterial pressure ⩾ 65 mmHg, in the absence of hypovolemia. The new set of definitions regards the term *severe sepsis* as redundant and does not rely on the use of the SIRS criteria, while placing significant importance on organ dysfunction and its assessment, as detected by changes of the SOFA score from baseline.[7] The important distinguishing features concerning the three general sepsis (identified as Sepsis 1, 2 and 3, respectively) and the ABA consensus definitions and the relevant SIRS criteria are presented in Tables 1 and 2, respectively;[5–7,15] the SOFA score is reported in Table 3.[28]

The introduction of the Sepsis 3 definitions has encountered mixed responses and generated a vigorous debate in the scientific community about the appropriateness of the proposed criteria. The major criticisms surround the perceived decreased sensitivity of the new criteria, which abandon the use of SIRS, leading to the potential under-detection of the condition. In particular, the criticism refers to the fact that the

Table 3. SOFA score.[28]

SOFA score	PaO_2/FiO_2 (mmHg)	Glasgow Coma Scale	Mean arterial pressure (MAP) or vasopressors required	Bilirubin (mg/dL) [µmol/L]	Platelets (10^3/µL)	Creatinine (mg/dL) [µmol/L] (or urine output)
4	⩾ 400	15	MAP ⩾ 70 mm/Hg	< 1.2 [< 20]	⩾ 150	< 1.2 [< 110]
3	< 400	13–14	MAP < 70 mm/Hg	1.2–1.9 [20-32]	< 150	1.2–1.9 [110–170]
2	< 300	10–12	Dopamine ⩽ 5 µg/kg/min or dobutamine (any dose)	2.0–5.9 [33–101]	< 100	2.0–3.4 [171–299]
1	< 200 mechanically ventilated	6–9	Dopamine > 5 µg/kg/min or epinephrine ⩽ 0.1 µg/kg/min or norepinephrine ⩽ 0.1 µg/kg/min	6.0–11.9 [102–204]	< 50	3.5–4.9 [300–440] (or < 500 mL/d)
0	< 100 mechanically ventilated	< 6	Dopamine > 15 µg/kg/min or epinephrine > 0.1 µg/kg/min or norepinephrine > 0.1 µg/kg/min	> 12.0 [> 204]	< 20	> 5.0 [> 440] (or < 200 mL/d)

requirement for organ failure in the context of infection to define sepsis may lead to under-recognition of the less severe cases of sepsis which may not have yet caused organ dysfunction. Such perceived potential for under-recognition is an obviously undesirable feature for a screening tool, aimed at the identification of a possibly lethal condition, and crucially, critics argue, likely to be detrimental to patient care.[29] Others have argued that neither definition is perfect and that the lethality of sepsis requires a highly sensitive screening tool, without necessarily compromising on specificity. Furthermore, it has been highlighted that both definitions fail to adequately cater for those patients with an infection and high risk of mortality who do not meet SIRS criteria nor display features of organ failure at presentation.[30]

If the Sepsis 3 criteria were to be utilised, without any modification, for diagnosing sepsis in the burns patient, the burns patient, like any other, would be considered septic only in presence of organ dysfunction accompanied by an increase of ⩾ 2 SOFA score points from baseline. Consequently, the issues related to the overreliance on the SIRS syndrome, characterising the first two sets of consensus definitions, would be overcome, as SIRS is not relied upon in Sepsis 3. Conversely, the SOFA score fails to consider specific systems, such as the gastrointestinal tract and the skin, due to lack of agreement over their grading and definitions.[31–33] As the organ source of sepsis is often one of the dysfunctional ones, and as burns patients' mortality is strongly correlated to the extent of body surface area affected, the failure of the SOFA score to consider skin dysfunction may represent a limitation for the tool in the specific burns patient population.[34,35]

Conclusions

The consensus definitions of sepsis and related syndromes in the general population have evolved over the last four decades, with emphasis shifting in Sepsis 3, from reliance on the SIRS criteria to organ dysfunction. The first two consensus definitions required modification by the ABA with regards to SIRS, to adapt them to the burns population. In Sepsis 3, the key element for diagnosis of sepsis is the development of organ failure in the context of infection. As mortality from sepsis increases with worsening severity and increasing number of organ dysfunctions, capturing the number of organs involved, the degree of sepsis induced dysfunction and the response to treatment, for all relevant systems, becomes vital in early detection of sepsis and assessment of outcome.[24,36–40] To that effect, it could be argued that an adaptation of the SOFA score, to include a severity grading of skin dysfunction, may be desirable for the burns population.

Declaration of conflicting interests

The authors declare that there is no conflict of interest.

Funding

This research received no specific grant from any funding agency in the public, commercial, or not-for-profit sectors.

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How to cite this article

Tridente A. Sepsis 3 and the burns patient: do we need Sepsis 3.1? *Scars, Burns & Healing*, Volume 4, 2018. DOI: 10.1177/2059513118790658