

## Pediatric Sepsis: Evolving Diagnostic Criteria, Organ Dysfunction Complications, and Evidence-Based Management

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### ABSTRACT

**Background:** Sepsis remains a principal cause of pediatric morbidity and mortality globally, accounting for more than 3 million childhood deaths annually. The 2024 Phoenix Sepsis Criteria represent a paradigm shift from SIRS-based definitions toward organ dysfunction-centered assessment, improving diagnostic accuracy and prognostic stratification. **Objective:** This review synthesizes current evidence on the epidemiology, pathophysiology, complications, and management of pediatric sepsis, contrasting historical and contemporary approaches. **Methods:** A narrative review of publications from PubMed, EMBASE, and Cochrane Library (2000–2025) was conducted. **Results:** Evidence supports POCUS-guided, titrated fluid resuscitation; early broad-spectrum antibiotics within one hour; norepinephrine as first-line vasopressor; and structured post-discharge surveillance for post-intensive care syndrome in pediatrics (PICS-p). **Conclusion:** Integrating the Phoenix Score into clinical practice alongside personalized resuscitation protocols and long-term follow-up will meaningfully reduce sepsis-associated mortality and long-term functional impairment.

**Keywords:** *pediatric sepsis; Phoenix Sepsis Score; organ dysfunction; septic shock; fluid resuscitation; PICS-p; biomarkers*

### I. INTRODUCTION

Sepsis in children constitutes one of the most formidable challenges in global healthcare, carrying a disproportionate burden of morbidity and mortality compared to most other acute pediatric conditions. Worldwide estimates indicate that approximately 25.2 million children are affected by sepsis annually, resulting in at least 3.4 million fatalities, with more than half of all cases occurring in children under five years of age [1].

Historically, pediatric sepsis was defined by the 2005 International Pediatric Sepsis Consensus Conference (IPSCC) as a suspected or confirmed infection

accompanied by at least two of four systemic inflammatory response syndrome (SIRS) criteria — fever or hypothermia, tachycardia, tachypnea, and leukocytosis or leukopenia [2]. While operationally convenient, SIRS criteria demonstrated poor specificity, capturing a broad population that included children with uncomplicated infections, post-surgical states, and non-infectious inflammatory conditions [3]. The consequent diagnostic imprecision contributed to both under-recognition of true sepsis and over-treatment of non-septic children with broad-spectrum antibiotics [4].

In January 2024, a landmark redefinition was introduced through the publication of the Phoenix Sepsis Criteria in the *Journal of the American Medical Association*, developed by a multidisciplinary Society of Critical Care Medicine (SCCM) task force [5]. The Phoenix Sepsis Score (PSS) defines pediatric sepsis as a score of 2 or greater in a child with suspected infection, incorporating organ dysfunction across four domains: respiratory, cardiovascular, coagulation, and neurological systems [6]. Septic shock is now identified when cardiovascular dysfunction ( $PSS \geq 1$ ) is present in a child meeting sepsis criteria [7]. This reconceptualization aligns pediatric definitions more closely with the Sepsis-3 adult framework and improves identification of children at greatest risk of adverse outcomes [8].

Beyond diagnosis, the management of pediatric sepsis has evolved considerably. The 2020 Surviving Sepsis Campaign (SSC) pediatric guidelines emphasize early, goal-directed but individually titrated fluid resuscitation, prompt antibiotic administration, and vasopressor selection guided by hemodynamic phenotype [9]. Concurrently, emerging evidence highlights the long-term sequelae of pediatric sepsis — including post-intensive care syndrome in pediatrics (PICS-p), persistent functional decline, and recurrent infections — which underscore the imperative for structured post-discharge surveillance [10].

This review aims to provide a comprehensive synthesis of the current understanding of pediatric sepsis, encompassing its epidemiology, pathophysiology, diagnostic evolution, complications, and evidence-based management strategies, with reference to the most contemporary clinical guidelines and research literature.

## II. METHODS

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This narrative review was conducted following a structured search of the PubMed, EMBASE, and Cochrane Library databases for publications spanning January 2000 to April 2025. Search terms employed included "pediatric sepsis," "septic shock children," "organ dysfunction pediatrics," "Phoenix Sepsis Score," "fluid resuscitation children," "PICU mortality," "PICS-p," and "pediatric sepsis biomarkers," combined using Boolean operators (AND, OR). Studies were included if they involved

children aged 0–17 years, reported on sepsis diagnosis, management, or outcomes, and were published in peer-reviewed English-language journals. Exclusion criteria comprised adult-only studies, non-sepsis inflammatory conditions, and non-peer-reviewed sources. In total, 50 publications were identified and reviewed, encompassing randomized controlled trials, prospective and retrospective cohort studies, systematic reviews, meta-analyses, consensus guidelines, and narrative reviews. Data were synthesized thematically under the IMRAD framework.

**Table 1: Comparison of Pediatric Sepsis Diagnostic Scoring Systems**

Criterion / Score	Basis	Organ Systems Assessed	Sensitivity (%)	Specificity (%)
SIRS (2005 IPSCC)	Fever, HR, RR, WBC	Systemic (non-specific)	~87	~24
pSOFA Score (2017)	Organ dysfunction	6 organ systems	~85	~67
PELOD-2 (2013)	Organ failure	5 organ systems	~83	~70
Phoenix Score (2024)	Organ dysfunction	Resp, CV, Coag, Neuro	~91	~75
qSOFA (adults, 2016)	AMS, RR, BP	3 quick indicators	~72	~78

*SIRS = Systemic Inflammatory Response Syndrome; HR = Heart Rate; RR = Respiratory Rate; WBC = White Blood Cell count; CV = Cardiovascular; Coag = Coagulation; AMS = Altered Mental Status; Resp = Respiratory. Sensitivity and specificity values are approximate ranges drawn from published validation cohorts.*

### III. RESULTS

#### 3.1 Epidemiology and Global Burden

Pediatric sepsis imposes a massive and unequally distributed global burden. A 2017 systematic analysis estimated 25.2 million annual sepsis episodes in children, with 3.4 million attributable deaths [11]. Mortality rates vary dramatically by region: high-income countries report in-hospital case fatality rates of 2–8%, whereas low- and middle-income countries bear rates exceeding 20–30% due to resource constraints, delayed presentation, and limited intensive care capacity [12]. Children under five years — particularly neonates and infants — constitute the highest-risk population, accounting for more than 60% of all pediatric sepsis deaths [13].

The most prevalent sources of infection leading to pediatric sepsis include respiratory tract infections (pneumonia representing 43.6% of cases in several PICU cohorts), bloodstream infections, urinary tract infections, and gastrointestinal sources

[14]. Gram-negative organisms predominate in community-acquired sepsis, while Gram-positive pathogens, particularly *Staphylococcus aureus* and *Streptococcus pyogenes*, account for a significant proportion of severe septic shock presentations [15]. Immunocompromised children — including those with malignancies, congenital heart disease, or chronic pulmonary conditions — face amplified risk and poorer baseline functional reserve, which substantially influences prognosis [16].

### 3.2 Pathophysiology and Organ Dysfunction

The pathophysiology of pediatric sepsis reflects both universal host-pathogen dynamics and age-specific immunological characteristics. The pediatric immune system differs fundamentally from that of adults, exhibiting a combination of immunological resistance, disease tolerance, and enhanced resilience that modulates both the clinical presentation and the severity of the inflammatory cascade [17]. Activation of pattern recognition receptors triggers an initial pro-inflammatory surge, characterized by cytokine release (TNF- $\alpha$ , IL-1 $\beta$ , IL-6, IL-8), complement activation, and coagulation pathway dysregulation. If not controlled, this dysregulated host response leads to endothelial dysfunction, microvascular injury, and progressive multi-organ dysfunction [18].

Organ dysfunction in pediatric sepsis most frequently involves the cardiovascular, respiratory, and neurological systems. In large multicenter PICU cohorts, cardiovascular dysfunction was documented in approximately 70% of non-survivors, followed by respiratory failure (68%) and coagulopathy (45%) [19]. Multiple organ dysfunction syndrome (MODS) — defined as sequential or concurrent failure of two or more organ systems — is the primary driver of PICU mortality, with non-survivors displaying significantly higher pSOFA scores at 24 and 48 hours post-admission compared to survivors [20]. Elevated serum lactate at presentation serves as an independent predictor of MODS and mortality, with lactate clearance at 24 hours correlating inversely with adverse outcomes [21].

In validated PICU cohorts, neurological and respiratory dysfunction were consistently the most predictive organ system failures for 30-day mortality, regardless of the scoring system employed (IPSCC, pSOFA, PELOD-2, or Phoenix Score) [22]. The Phoenix Score demonstrated superior discrimination in the 2024 validation cohorts, with an area under the receiver operating characteristic (AUROC) exceeding 0.91 for predicting hospital mortality in children with infection [23].

### 3.3 Complications of Pediatric Sepsis

Pediatric sepsis generates both acute and long-term complications that extend far beyond the index hospitalization. Acute complications include acute respiratory

distress syndrome (ARDS), acute kidney injury (AKI), disseminated intravascular coagulation (DIC), septic encephalopathy, and myocardial dysfunction. AKI occurs in 20–40% of PICU sepsis admissions and is independently associated with prolonged ventilation, longer ICU stay, and elevated mortality [24].

Long-term outcomes are increasingly recognized as equally critical. The Life After Pediatric Sepsis Evaluation (LAPSE) study, a landmark prospective multicenter cohort of 389 children hospitalized for septic shock, demonstrated that 35% of survivors experienced significant decline in health-related quality of life (HRQL) persisting for at least one year post-discharge [25]. Approximately one in four children failed to recover to their pre-sepsis functional baseline, with immune compromise, malignancy, and complex chronic disease identified as independent risk factors for persistent HRQL deficit [26]. Post-intensive care syndrome in pediatrics (PICS-p) encompasses cognitive impairment, emotional disturbances, and physical disability affecting survivors and their caregivers, representing an underappreciated public health burden [27].

A 2026 systematic review and meta-analysis of 72,065 records reported that long-term mortality (beyond hospital discharge) in pediatric sepsis remains substantially elevated, particularly within the first year after discharge, highlighting the need for structured post-discharge surveillance programs targeting HRQL, neurodevelopmental status, and recurrent infection risk [28].

### **3.4 Management: Evidence-Based Strategies**

Contemporary management of pediatric sepsis rests on three cornerstones: early antimicrobial therapy, goal-directed but individualized fluid resuscitation, and hemodynamic support with vasoactive agents, all underpinned by continuous clinical reassessment [29].

**Antibiotic Therapy:** Empiric broad-spectrum antibiotic administration within one hour of sepsis recognition is a strongly endorsed recommendation in the SSC 2020 pediatric guidelines [30]. Cultures should be obtained prior to antibiotic initiation whenever feasible without delaying therapy. De-escalation to targeted agents at 48–72 hours, guided by microbial sensitivity data, supports antimicrobial stewardship and reduces selection pressure [31].

**Fluid Resuscitation:** The SSC 2020 guidelines recommend fluid boluses of 10–20 mL/kg in aliquots over 5–20 minutes, titrated to clinical markers of cardiac output including heart rate, capillary refill time, blood pressure, lactate, and central venous oxygen saturation [32]. Fluid bolus therapy should be suspended if signs of fluid overload (hepatomegaly, pulmonary crackles, worsening oxygenation) emerge. Point-

of-care ultrasound (POCUS) is recommended to guide resuscitation where local expertise allows, as it enables dynamic assessment of volume responsiveness and cardiac function [33]. The Resuscitation, Equilibrium, and De-escalation (RED) strategy proposes a phased, personalized hemodynamic approach — acknowledging that resuscitation needs evolve from initial volume replacement to optimization to active fluid removal in the recovery phase — and represents a promising framework for precision management [34].

**Vasopressor Therapy:** For children with fluid-refractory septic shock, norepinephrine is preferred over dopamine as the first-line vasopressor based on its more favorable hemodynamic and adverse event profile [35]. Epinephrine is recommended for refractory shock, while vasopressin and terlipressin serve as adjuncts in vasodilatory shock phenotypes. Initiating vasoactive agents before completing 40 mL/kg of fluid may be appropriate in severe hypotension to prevent prolonged hypoperfusion, though evidence remains limited [36].

**Corticosteroids:** Stress-dose hydrocortisone (2–4 mg/kg/day) is recommended for children with documented or suspected adrenal insufficiency, purpura fulminans, or persistent refractory shock not responding to vasopressors [37]. Routine corticosteroid use in septic shock without these indications lacks robust evidence of benefit and is not recommended by current guidelines [38].

**Immunomodulation:** Intravenous immunoglobulin (IVIG) is indicated for confirmed or suspected streptococcal or staphylococcal toxic shock syndrome. Emerging data suggest potential roles for corticosteroid phenotype-guided therapy and novel biologic agents targeting specific inflammatory pathways, though these remain under investigation [39].

**Table 2: Comparison of Conventional vs. Contemporary (2024) Management Approaches in Pediatric Sepsis**

Management Domain	Conventional Approach	Contemporary (2024) Approach	Evidence Level
<b>Fluid Resuscitation</b>	20 mL/kg bolus, 3 rounds; aggressive early loading	10-20 mL/kg aliquots titrated to clinical markers; POCUS-guided; limit overload	Moderate (SSC 2020)
<b>First-line Vasopressor</b>	Dopamine (inotropic + vasopressor)	Norepinephrine preferred; epinephrine for fluid-refractory shock	Moderate (SSC 2020)

Management Domain	Conventional Approach	Contemporary (2024) Approach	Evidence Level
<b>Antibiotics</b>	Broad-spectrum empiric; delayed de-escalation	Within 1 hour; cultures first; de-escalate at 48-72 h per sensitivity	Strong (SSC 2020)
<b>Corticosteroids</b>	Routine in septic shock	Reserved for refractory shock or adrenal insufficiency; hydrocortisone 2-4 mg/kg/day	Low (conditional)
<b>Monitoring</b>	Vital signs, CVP, lactate	POCUS, lactate clearance, pSOFA serial scoring, Phoenix Score	Low-Moderate
<b>Immunomodulation</b>	Not routine; IVIG only in toxic shock	IVIG for toxic shock; investigational: corticosteroid phenotyping, biologics	Low (emerging)
<b>Post-discharge Care</b>	Limited follow-up; no structured protocol	Structured surveillance; assessment; rehabilitation	Emerging consensus

SSC = Surviving Sepsis Campaign; POCUS = Point-of-Care Ultrasound; HRQL = Health-Related Quality of Life; PICS-p = Post-Intensive Care Syndrome in Pediatrics; CV = Cardiovascular. Evidence levels reflect SSC 2020 grading (Strong, Moderate, Low, Conditional).

### 3.5 Biomarkers and Monitoring

Accurate biomarker integration into sepsis recognition and monitoring remains an active research frontier. Procalcitonin (PCT) is the most widely validated biomarker for distinguishing bacterial infection from non-infectious causes and for guiding antibiotic de-escalation [40]. C-reactive protein (CRP), while more commonly available, offers lower specificity. Serum lactate, in addition to its diagnostic value, serves as a critical monitoring tool: lactate clearance of less than 10% at 24 hours is independently associated with increased mortality and MODS in PICU cohorts [41].

Combining multiple biomarkers with clinical early warning scores has demonstrated additive value in risk stratification, though resource constraints in many settings limit broad applicability [42]. Machine learning-based early warning systems, such as the TREWS algorithm, have shown promise in adult populations for early sepsis detection and outcome prediction, with translation to pediatric applications currently underway [43]. Serial pSOFA scoring and Phoenix Score monitoring at 24- and 48-hour intervals significantly improve prognostication and inform real-time management decisions in PICU settings [44].

#### IV. DISCUSSION

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The evolution of pediatric sepsis definitions from SIRS-based criteria to the organ dysfunction-focused Phoenix Score represents a clinically meaningful advance in both diagnostic precision and prognostic stratification. By incorporating the four most mortality-predictive organ systems — respiratory, cardiovascular, coagulation, and neurological — the Phoenix Score addresses a fundamental limitation of prior frameworks, namely the high sensitivity but critically low specificity of SIRS criteria that frequently led to overdiagnosis and antibiotic overuse [45, 46].

The clinical implications of this reclassification extend beyond semantics. Studies comparing the 2005 IPSCC and 2024 Phoenix criteria in prospective PICU cohorts have demonstrated that the Phoenix Score identifies a more severely ill subpopulation — with higher rates of organ dysfunction, greater vasopressor requirements, and higher 30-day mortality — compared to the broader SIRS-defined cohort [47]. This concordance between definition and severity enhances the clinical validity of future sepsis research and trial enrollment.

Fluid management remains among the most debated domains in pediatric sepsis care. The FEAST trial — which demonstrated increased 48-hour mortality with fluid bolus administration in African children with febrile illness — challenged the universality of aggressive early fluid resuscitation and catalyzed a global reassessment of resuscitation protocols [48]. Subsequent guidelines have refined recommendations toward individualized, titrated fluid delivery with POCUS-guided assessment, reflecting a movement from protocol-driven to precision-medicine approaches. The RED strategy embodies this evolution, proposing hemodynamic phenotyping to guide the transition from resuscitation to equilibrium to de-escalation phases [49].

Long-term outcomes represent a dimension of pediatric sepsis care that has historically received insufficient clinical and research attention. The recognition that 35% of septic shock survivors exhibit persistent HRQL decline, and that nearly one in four fail to recapture baseline functional status, imposes an obligation on clinical systems to develop and implement structured post-discharge surveillance frameworks [50]. PICS-p encompasses physical, cognitive, emotional, and social domains, and its management requires multidisciplinary rehabilitation spanning pediatrics, psychology, physiotherapy, and social support services.

The intersection of internal medicine and pediatrics — particularly in the domains of immunology, infectious disease, and critical care — underscores the value of combined training and interdisciplinary collaboration. Children with chronic conditions, including diabetes, malignancy, congenital heart disease, and chronic lung

disease, present unique sepsis phenotypes requiring individualized management that draws equally on principles of internal medicine and pediatric subspecialty care.

## V. CONCLUSION

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Pediatric sepsis stands at a transformative threshold. The introduction of the Phoenix Sepsis Score in 2024 signals the most significant redefinition of this condition in nearly two decades, offering clinicians a more precise, organ dysfunction-centered framework for identifying the children at greatest risk and guiding timely, targeted intervention. When paired with individualized, POCUS-guided fluid resuscitation, early broad-spectrum antimicrobial therapy, phenotype-appropriate vasopressor support, and vigilant monitoring of lactate and pSOFA trajectories, modern sepsis management holds tangible promise for reducing both in-hospital mortality and long-term morbidity.

Yet the story of survival cannot end at the PICU door. The pervasive burden of PICS-p, persistent functional decline, and long-term mortality demands that clinicians, health systems, and policymakers embrace structured post-discharge surveillance as an integral — not optional — component of sepsis care. In weaving together the disciplinary threads of internal medicine, pediatrics, critical care, infectious disease, and rehabilitation medicine, the medical community has both the tools and the obligation to transform pediatric sepsis from a frequently lethal emergency into a survivable condition with an achievable path to full recovery. The imperative now is to act on that knowledge — swiftly, systematically, and with unwavering attention to every dimension of a child's health.

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