"A STUDY TO COMPARE COMBINATION OF MODIFIED EARLY WARNING SCORE (MEWS) AND LACTATE WITH SEQUENTIAL ORGAN FAILURE ASSESSMENT (SOFA) SCORE AND ACUTE PHYSIOLOGY AND CHRONIC HEALTH EVALUATION (APACHE) II SCORE IN PREDICTING SEVERITY IN SEPSIS PATIENTS"

BY

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DISSERTATION SUBMITTED TO
SRI DEVARAJ URS ACADEMY OF HIGHER EDUCATION AND RESEARCH, KOLAR, KARNATAKA
In partial fulfilment of the requirements for the degree of
DOCTOR OF MEDICINE
IN
EMERGENCY MEDICINE

Under the Guidance of

DR. RAJESH K

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2025

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Abstract

Background:
Sepsis, an exceedingly lethal medical condition that can lead to organ dysfunction and a significant mortality rate, is defined by an aberrant response of the host to infectious agents. Early identification of patients at risk of poor outcomes is critical. While scoring systems like SOFA and APACHE II are widely used in ICUs, the utility of combining simpler tools such as the Modified Early Warning Score (MEWS) with serum lactate requires further evaluation.

Objectives:
To compare the predictive performance of MEWS + Lactate with that of SOFA and APACHE II scores in assessing severity and predicting mortality in patients with sepsis.

Methods:
This prospective observational study involved 201 adult patients who arrived at a tertiary care center's emergency room with sepsis. Serum lactate levels, APACHE II scores, SOFA, and MEWS were used to assess patients upon admission. The MEWS + Lactate score was computed by combining the two parameters. Outcomes were classified as discharged or deceased. Statistical analysis included Mann–Whitney U test, Spearman's correlation, ROC curve analysis, and Kruskal-Wallis test to evaluate predictive accuracy and score variations across infection sources.

Results:
The in-hospital mortality rate was 21.9%. All four scores were significantly higher in non-survivors ($p < 0.001$). APACHE II showed the strongest predictive value (mean 29.48 ± 6.97; AUC = 0.920; r = –0.601), followed by MEWS + Lactate (AUC = 0.853; r = –0.549) and SOFA (AUC = 0.872; r = –0.559). Lactate alone and MEWS also showed moderate correlation with outcome. SOFA and lactate scores showed significant variation across different sepsis sources ($p = 0.006$ and $p = 0.017$, respectively), with the highest scores seen in neurointection and unknown source groups.

Conclusion:
For patients with sepsis, APACHE II continues to be the most reliable indicator of in-hospital mortality. However, MEWS + Lactate offers a practical and rapid bedside alternative with strong predictive power, making it highly useful in emergency settings. SOFA continues to be a valuable tool for organ dysfunction assessment. Integrating clinical scores with lactate measurement can enhance early sepsis risk stratification and guide timely intervention.

Keywords: Sepsis, MEWS, SOFA, APACHE II, Lactate, Mortality Prediction, Clinical Scoring, Risk Stratification.

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Date: **DR. ARAVIND S R**

Place:

LIST OF ABBREVIATIONS

ACCP	American College of Chest Physicians
AKI	Acute Kidney Injury
APACHE II	Acute Physiology and Chronic Health Evaluation II
ARDS	Acute Respiratory Distress Syndrome
AUC	Area Under the Curve
AVPU	Alert, Verbal, Pain, Unresponsive
BLA	Blood Lactate
CAP	Community-Acquired Pneumonia
CAUTI	Catheter-Associated Urinary Tract Infections
CBC	Complete Blood Count
CLABSI	Central Line-Associated Bloodstream Infections
CRP	C-Reactive Protein
DAMP	Damage-Associated Molecular Patterns
DIC	Disseminated Intravascular Coagulation
DNR	Do Not Resuscitate
ED	Emergency Department
EGDT	Early Goal Directed Therapy
ESICM	European Society of Intensive Care Medicine
EWS	Early Warning Score
GCS	Glasgow Coma Scale
HAP	Hospital-Acquired Pneumonia
ICU	Intensive Care Unit
IL-10	Interleukin-10
IL-1β	Interleukin-1 Beta
IL-6	Interleukin-6
IL-8	Interleukin-8
LDH	Lactate Dehydrogenase
LODS	Logistic Organ Dysfunction Score
MAP	Mean Arterial Pressure
MEDS	Mortality in Emergency Department Sepsis

MEWS	Modified Early Warning Score
MODS	Multi-Organ Dysfunction Syndrome
MRSA	Methicillin-Resistant Staphylococcus Aureus
NLR	Nod-Like Receptors
NO	Nitric Oxide
PAMP	Pathogen-Associated Molecular Patterns
PCT	Procalcitonin
PIRO	Predisposition, Infection, Response and Organ Dysfunction Scores
PRR	Pattern Recognition Receptors
qSOFA	Quick Sequential Organ Failure Assessment
REMS	Rapid Emergency Medicine Score
ROC	Receiver Operating Characteristic
ROS	Reactive Oxygen Species
SAPS II	Simplified Acute Physiology Score II
SBP	Systolic Blood Pressure
SCCM	Society of Critical Care Medicine
SIRS	Systemic Inflammatory Response Syndrome
SOFA	Sequential Organ Failure Assessment
SSC	Surviving Sepsis Campaign
TGF-β	Transforming Growth Factor-Beta
TLR	Toll-Like Receptors
TNF-α	Tumor Necrosis Factor-Alpha
VAP	Ventilator-Associated Pneumonia
WBC	White Blood Cells

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ABSTRACT

Background:

Sepsis, an exceedingly lethal medical condition that can lead to organ dysfunction and a significant mortality rate, is defined by an aberrant response of the host to infectious agents. Early identification of patients at risk of poor outcomes is critical. While scoring systems like SOFA and APACHE II are widely used in ICUs, the utility of combining simpler tools such as the Modified Early Warning Score (MEWS) with serum lactate requires further evaluation.

Objectives:

To compare the predictive performance of MEWS + Lactate with that of SOFA and APACHE II scores in assessing severity and predicting mortality in patients with sepsis.

Methods:

This prospective observational study involved 201 adult patients who arrived at a tertiary care center's emergency room with sepsis. Serum lactate levels, APACHE II scores, SOFA, and MEWS were used to assess patients upon admission. The MEWS + Lactate score was computed by combining the two parameters. Outcomes were classified as discharged or deceased. Statistical analysis included the Mann–Whitney U test, Spearman's correlation, ROC curve analysis, and Kruskal-Wallis test to evaluate predictive accuracy and score variations across infection sources.

Results:

The in-hospital mortality rate was 21.9%. All four scores were significantly higher in non-survivors ($p < 0.001$). APACHE II showed the strongest predictive value (mean 29.48 ± 6.97; AUC = 0.920; r = –0.601), followed by MEWS + Lactate (AUC = 0.883; r = –0.549) and SOFA (AUC = 0.873; r = –0.539). Lactate alone and MEWS also showed moderate correlation with outcome. SOFA and lactate scores showed significant variation across different sepsis

sources ($p = 0.006$ and $p = 0.017$, respectively), with the highest scores seen in neuroinfection and unknown source groups.

Conclusion:

For patients with sepsis, APACHE II continues to be the most reliable indicator of in-hospital mortality. However, MEWS + Lactate offers a practical and rapid bedside alternative with strong predictive power, making it highly useful in emergency settings. SOFA continues to be a valuable tool for organ dysfunction assessment. Integrating clinical scores with lactate measurement can enhance early sepsis risk stratification and guide timely intervention.

Keywords: Sepsis, MEWS, SOFA, APACHE II, Lactate, Mortality Prediction, Clinical Scoring, Risk Stratification.

INTRODUCTION

INTRODUCTION

The hallmark of sepsis, a complicated clinical condition that can lead to potentially fatal organ failure, is a dysregulated host response to infection. [1] When the body's reaction to an infection damages its own tissues and organs, sepsis results. If left untreated, sepsis can result in septic shock and multi-organ failure. Globally, sepsis remains a major public health challenge, accounting for significant morbidity and mortality, and is currently recognized as one of the leading causes of death worldwide, ranking as the tenth most common cause.[2] Sepsis is more common in low- and middle-income nations because of a lack of funding and delayed access to medical care. Studies have demonstrated that delays in diagnosis and treatment greatly raise the risk of death for individuals with sepsis, making early detection and prompt intervention essential.

The pathophysiology of sepsis is intricate, involving a cascade of immune responses to an infectious trigger. When the body encounters a pathogen, an exaggerated inflammatory reaction ensues, marked by excessive release of pro-inflammatory cytokines—a phenomenon often referred to as a "cytokine storm."[3] This dysregulated immune response leads to widespread endothelial damage, microcirculatory dysfunction, and impaired oxygen delivery to tissues.[4] As a result, cellular metabolism becomes deranged, often reflected by elevated lactate levels due to anaerobic metabolism, which serves as an important marker of tissue hypoperfusion. If left unchecked, these pathophysiological processes drive the progression from sepsis to septic shock and, ultimately, multi-organ disfunction syndrome (MODS), contributing to poor clinical outcomes.[5]

Accurate and timely prognostication is essential due to sepsis's high mortality and quick progression. Various clinical scoring systems have been developed to assess the severity of illness and predict outcomes in critically ill patients. Among these, the Modified Early Warning Score (MEWS) is a straightforward bedside measure that predicts in-hospital mortality and ICU admissions based on physiological markers that are easily measurable.[6] Meanwhile, the SOFA[7]

and the APACHE II[8] scores are more comprehensive, incorporating laboratory data and detailed clinical information to quantify organ dysfunction and disease severity. While these tools are well-established in critical care settings, they may be resource-intensive and time-consuming, especially in settings where rapid decisions are needed.

However, there is growing interest in whether combining simple bedside assessments like MEWS with readily available biomarkers such as lactate can provide an effective alternative for early prognostication in sepsis. Lactate is frequently used to gauge the severity of the illness and direct resuscitation because it is a well-known indicator of tissue hypoperfusion and metabolic stress in sepsis. A combination of MEWS and lactate may offer a practical, cost-effective, and rapid method of assessing the severity of sepsis, particularly in resource-limited environments. Currently, there is a lack of studies directly comparing this combination with more established scores like SOFA and APACHE II in predicting outcomes in sepsis patients. Therefore, this study aims to fill that gap and evaluate whether the combination of MEWS and lactate can match or surpass traditional scoring systems in prognostic accuracy.

Improved methods of early risk stratification in sepsis are essential not only for guiding clinical decisions but also for optimizing resource allocation and improving patient outcomes. By comparing the combination of MEWS and lactate with SOFA and APACHE II, this study seeks to identify a simple, effective prognostic tool that could potentially enhance bedside assessment of sepsis severity, aid in timely interventions, and provide valuable insights for future research in the ongoing quest to reduce the global burden of sepsis.

AIM & OBJECTIVES

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AIM:

To prognosticate the severity and outcome of patients with sepsis based on Combination of Modified Early Warning score with lactate, in comparison with SOFA and APACHE II scores.

OBJECTIVES:

1) To assess the clinical outcome with Combination of MEWS and Lactate
2) To assess the clinical outcome with SOFA
3) To assess the clinical outcome with APACHE II
4) To compare the scores obtained by Combination of MEWS and Lactate with SOFA and APACHE II in predicting outcome

REVIEW OF LITERATURE

REVIEW OF LITERATURE

Definition and Evolution of Sepsis Criteria

A host response to infection that is dysregulated and can result in potentially fatal organ malfunction is the hallmark of sepsis, a complex clinical entity. The definition of sepsis has changed several times throughout the years as our knowledge of its aetiology and clinical manifestation has advanced. These changes aimed to improve diagnostic accuracy, facilitate early identification, and guide appropriate management. The evolution of sepsis definitions is closely linked to advances in critical care and the need for a standardized framework in both clinical practice and research.[9]

SIRS Criteria (1991)

The first major consensus definition of sepsis was established in 1991 by the ACCP and the SCCM.[10] This definition was grounded in the concept of the Systemic Inflammatory Response Syndrome (SIRS), which described a generalized inflammatory state triggered by infection, trauma, burns, or other insults. Under this model, sepsis was defined as the presence of infection along with two or more SIRS criteria. The SIRS criteria included:

- Body temperature >38°C or <36°C
- Heart rate >90 beats per minute
- Respiratory rate >20 breaths per minute or $PaCO_2$ <32 mm Hg
- White blood cell count >12,000/µL, <4,000/µL, or >10% immature (band) forms.

Sepsis is understood as a gradient of severity, progressing from uncomplicated sepsis (identified by the coexistence of infection and SIRS) to severe sepsis (which encompasses sepsis accompanied by organ dysfunction) and ultimately culminating in septic shock (distinguished by

sepsis-induced hypotension that is unresponsive to sufficient fluid resuscitation). While the SIRS-based definition provided a useful clinical tool, it was criticized for its lack of specificity and sensitivity. Many hospitalized patients met SIRS criteria without infection, and conversely, some septic patients failed to meet the criteria, leading to misclassification.

Sepsis-2 (2001)

In 2001, an updated consensus conference was convened to refine the sepsis definitions, leading to what is retrospectively referred to as Sepsis-2. This revision acknowledged the limitations of SIRS and expanded the diagnostic criteria to include various signs of organ dysfunction, hypoperfusion, and hypotension. Key parameters for identifying sepsis included altered mental status, hypoxemia, elevated lactate levels, oliguria, and coagulopathy. However, the 2001 update did not discard the SIRS framework and continued to rely on it as the entry point for diagnosis. The emphasis remained on recognizing severe sepsis and septic shock as distinct entities within the continuum.

Despite the additions, the Sepsis-2 definition still faced considerable challenges. Clinical studies demonstrated that SIRS criteria were often met in a broad range of hospitalized patients without infection, diluting the specificity of the diagnosis. Moreover, early detection of sepsis remained problematic, and inconsistencies persisted in epidemiological reporting and clinical trial enrolment, necessitating further refinement.

Sepsis-3 (2016)

A major paradigm shift occurred with the introduction of Sepsis-3 definitions in 2016, jointly proposed by the ESICM and the SCCM.[11] This revision fundamentally reformulated sepsis as "life-threatening organ dysfunction caused by a dysregulated host response to infection." This updated definition discards the SIRS criteria from the diagnostic process, instead focusing on the presence of organ dysfunction quantified by the SOFA score.[7]

According to Sepsis-3, an increase of ≥2 points in SOFA score from baseline in the context of suspected or confirmed infection indicates sepsis. This change aimed to better identify patients at higher risk of mortality and guide timely management. Additionally, Sepsis-3 redefined septic shock as a subset of sepsis that is characterized by severe circulatory and metabolic abnormalities, including persistent hypotension that necessitates the use of vasopressors to maintain a mean arterial pressure (MAP) ≥65 mm Hg and serum lactate levels >2 mmol/L despite adequate fluid resuscitation.[12]

An important addition in Sepsis-3 was the introduction of the quick Sequential Organ Failure Assessment (qSOFA) score, designed as a simple bedside tool to rapidly identify patients at risk of poor outcomes outside the ICU. qSOFA is positive when two or more of the following criteria are met:[13]

- Respiratory rate ≥22 breaths per minute
- Altered mental status (Glasgow Coma Scale <15)
- Systolic blood pressure ≤100 mm Hg

While qSOFA is not intended to diagnose sepsis directly, it serves as a prompt for clinicians to investigate and manage possible sepsis in non-ICU settings. The implementation of SOFA and qSOFA as part of Sepsis-3 has shifted the focus from generalized inflammation (as in SIRS) to measurable organ dysfunction, which is more closely correlated with patient outcomes.

Flow and Transition:

This evolution in sepsis definitions reflects the ongoing efforts to balance simplicity and accuracy in sepsis identification. With the adoption of organ dysfunction scores like SOFA and tools like qSOFA, the clinical approach has become more structured, allowing earlier identification of severe

cases. Understanding these definitions is crucial when evaluating scoring systems like MEWS, SOFA, and APACHE II, as their application hinges on the pathophysiological concepts and diagnostic criteria that define sepsis today.

Etiology of Sepsis

An aberrant host response to infection leads to sepsis, with the underlying etiology intricately linked to the causative bacteria and the location of the primary infection. Understanding the etiology is essential, as it directly influences diagnostic approaches, antimicrobial selection, and overall management strategies. Despite variations across regions and patient populations, certain pathogens and infection sites are consistently implicated in the development of sepsis.[14]

Common Causative Organisms

Microbial pathogens responsible for sepsis are diverse, including bacteria, viruses, fungi, and occasionally parasites. However, bacterial infections remain the predominant cause globally, accounting for the majority of sepsis cases.[15]

Bacterial Pathogens

Gram-negative and Gram-positive bacteria are both common etiological agents, though the prevalence of each may vary based on geographical location, healthcare setting, and patient-specific factors.

- **Gram-negative bacteria** are frequently implicated, especially in healthcare-associated infections. Common Gram-negative organisms include:
    - *Escherichia coli* (particularly in urinary tract infections and intra-abdominal infections)
    - *Klebsiella pneumoniae*
    - *Pseudomonas aeruginosa*
    - *Acinetobacter baumannii*

- **Gram-positive bacteria** are also major contributors to sepsis, especially in community-acquired infections and skin/soft tissue infections. Key Gram-positive pathogens include:
    - *Staphylococcus aureus* (including methicillin-resistant *Staphylococcus aureus* [MRSA])
    - *Streptococcus pneumoniae*
    - *Enterococcus* species

Viral Pathogens

While bacterial infections dominate, viral infections can also lead to sepsis, particularly in immunocompromised patients, neonates, and during viral epidemics. Viruses implicated in sepsis include:

- Influenza virus
- SARS-CoV-2 (COVID-19)
- Herpes simplex virus (HSV)
- Human immunodeficiency virus (HIV)
- Cytomegalovirus (CMV)

Viral sepsis is often characterized by profound immune dysregulation, which can predispose patients to secondary bacterial or fungal infections, further complicating the clinical course.

Fungal Pathogens

Fungal sepsis, while less common than bacterial or viral sepsis, is increasingly recognized, particularly in critically ill or immunosuppressed populations (e.g., patients receiving chemotherapy, long-term corticosteroids, or post-transplant). The most frequently involved fungi include:[16]

- *Candida* species (notably *Candida albicans* and *Candida glabrata*)
- *Aspergillus* species in cases of invasive aspergillosis
- *Cryptococcus neoformans*, particularly in individuals with advanced HIV/AIDS

The increasing prevalence of invasive fungal infections is partly attributed to the widespread use of broad-spectrum antibiotics, central venous catheters, and prolonged ICU stays.

Sites of Infection Leading to Sepsis

The development of sepsis often begins with a localized infection that progresses to systemic involvement. Identifying the source of infection is crucial for appropriate management, including source control measures and targeted antimicrobial therapy.[17]

1. Lungs

The most frequent site of infection that results in sepsis is the respiratory tract, and pneumonia is the primary cause of sepsis globally. Both community-acquired pneumonia (CAP) and hospital-acquired pneumonia (HAP), including ventilator-associated pneumonia (VAP), can precipitate sepsis. Typical pathogens include:

- *Streptococcus pneumoniae*
- *Staphylococcus aureus*
- *Pseudomonas aeruginosa*
- Influenza and SARS-CoV-2 during viral epidemics

Pulmonary infections can rapidly lead to respiratory failure, necessitating mechanical ventilation and contributing to higher mortality in septic patients.

2. Abdomen

Intra-abdominal infections are another major source of sepsis, often arising from:

- Peritonitis
- Intra-abdominal abscesses
- Biliary tract infections (e.g., cholangitis, cholecystitis)
- Gastrointestinal perforations

Polymicrobial infections are common in abdominal sepsis, frequently involving a mix of Gram-negative bacilli, Gram-positive cocci, and anaerobes. Timely surgical intervention for the purpose of source control is frequently essential alongside antimicrobial treatment.

3. Urinary Tract

The urinary tract is a frequent source of sepsis, particularly in elderly populations, individuals with indwelling catheters, and patients with structural abnormalities of the urinary system. Urosepsis refers to sepsis originating from urinary tract infections (UTIs), which may involve:

- Pyelonephritis
- Prostatitis
- Catheter-associated urinary tract infections (CAUTIs)

Common pathogens include:

- *Escherichia coli*
- *Klebsiella* species
- *Proteus* species
- *Enterococcus* species

4. Bloodstream

Primary bloodstream infections, such as central line-associated bloodstream infections (CLABSIs), are notable causes of sepsis in hospitalized and ICU patients. These infections often involve:

- Coagulase-negative *Staphylococcus*
- *Staphylococcus aureus*
- *Candida* species (particularly in prolonged hospital stays with central venous access)

Secondary bloodstream infections can also occur from distant foci, including pneumonia, intra-abdominal infections, or skin and soft tissue infections.

Other Sites

Although less common, sepsis can also originate from:

- Skin and soft tissue infections (e.g., cellulitis, necrotizing fasciitis)
- Meningitis (central nervous system infections)
- Bone and joint infections (osteomyelitis, septic arthritis)

Regional and Demographic Variations

The etiological profile of sepsis varies according to geography, patient population, and healthcare setting. In low-resource environments, tropical infections (such as malaria, dengue, and leptospirosis) may present as sepsis-like syndromes. In contrast, in high-income countries, healthcare-associated infections are more prevalent due to invasive procedures, immunosuppressive therapies, and prolonged hospitalizations.

Pathogenesis of Sepsis

Sepsis is characterized by a complex interplay between the invading pathogen and the host's immune system. The pathogenesis involves a dysregulated immune response that leads to widespread inflammation, endothelial dysfunction, coagulation abnormalities, and ultimately, multiple organ dysfunction. Understanding the fundamental mechanisms behind sepsis pathogenesis is essential to guide therapeutic strategies and prognostic assessments.[18]

Host-Pathogen Interactions

When a pathogen—typically bacteria, viruses, fungi, or parasites—enters a normally sterile bodily compartment, sepsis is initiated. The immune cells of the host employ pattern recognition receptors (PRRs), including Toll-like receptors (TLRs) and NOD-like receptors (NLRs), to discern pathogen-associated molecular patterns (PAMPs), which are unique molecular structures present in pathogens.[19]

Upon recognition of PAMPs, innate immune cells—particularly macrophages, neutrophils, and dendritic cells—are activated. These cells rapidly release a cascade of signalling molecules that initiate the inflammatory response. In parallel, DAMPs, released from injured host cells, further amplify this immune activation, even in the absence of ongoing infection.

The interaction between pathogens and host defences aims to neutralize and eliminate the infectious threat. However, in sepsis, this response becomes dysregulated. The localized immune defence escalates into systemic inflammation, with widespread endothelial activation, capillary leakage, and disruption of vascular integrity. These changes facilitate further pathogen dissemination, perpetuating a vicious cycle of infection and inflammation.

Role of Pro-inflammatory and Anti-inflammatory Mediators

Sepsis is marked by a paradoxical and simultaneous release of pro-inflammatory and anti-inflammatory mediators, leading to a complex immune landscape that can be harmful when unbalanced.[20]

Pro-inflammatory Mediators

In the early phases of sepsis, the host mounts an aggressive pro-inflammatory response. Key mediators include:

- **Cytokines**: such as TNF-α, IL-1β, and IL-6, which trigger fever, recruit immune cells, and promote the acute phase response.
- **Chemokines**: like IL-8, which direct neutrophil migration to infection sites.
- **Reactive oxygen species (ROS)** and **nitric oxide (NO)**, which aid in microbial killing but also cause collateral tissue damage.

This uncontrolled inflammation results in endothelial dysfunction, increased vascular permeability, and microvascular thrombosis, contributing to tissue hypoperfusion and organ injury.

Anti-inflammatory Mediators

Almost concurrently, the body activates counter-regulatory mechanisms to dampen the inflammatory response and prevent excessive tissue damage. These include:

- **IL-10** and **TGF-β**, which suppress cytokine production and downregulate immune cell activity.

- Increased expression of inhibitory receptors on immune cells, leading to immune cell exhaustion.
- Induction of apoptosis in lymphocytes and dendritic cells, leading to immunosuppression.

This compensatory anti-inflammatory response syndrome (CARS) can progress to a state of immunoparalysis, rendering the host susceptible to secondary infections and impairing the resolution of the primary infetion.[21]

The simultaneous activation of pro-inflammatory and anti-inflammatory responses reflects the dynamic and biphasic nature of sepsis, where some patients may suffer from hyperinflammation, while others succumb to immunosuppression.

Hemodynamic Alterations and Shock States

Sepsis has profound effects on the cardiovascular system, leading to significant hemodynamic instability. The hallmark of severe sepsis and septic shock is circulatory failure due to a combination of vasodilation, impaired cardiac function, and abnormal distribution of blood flow.[22]

Vasodilation and Vascular Permeability

Inflammatory mediators such as NO cause profound systemic vasodilation, leading to reduced systemic vascular resistance. Concurrently, endothelial injury increases capillary permeability, resulting in fluid extravasation into the interstitial space. This combination leads to relative hypovolemia, despite adequate or even increased intravascular fluid volumes.

Distributive Shock

Septic shock is classified as a form of distributive shock, where blood flow is maldistributed due to vasoplegia and microvascular dysfunction. This leads to inadequate tissue perfusion despite normal or elevated cardiac output in some cases.[23]

Myocardial Depression

Sepsis can also induce septic cardiomyopathy, characterized by reduced ejection fraction and impaired contractility. TNF-α, IL-1β, and other mediators exert direct negative effects on myocardial cells, compounding the hemodynamic compromise.

Microcirculatory Dysfunction

Despite systemic efforts to maintain perfusion, microcirculatory flow becomes heterogeneous, with some tissue beds experiencing severe hypoperfusion. This leads to localized tissue hypoxia and plays a role in the development of multiple organ dysfunction syndrome. (MODS).

Progression to Shock

When these hemodynamic derangements become severe and unresponsive to fluid resuscitation, septic shock ensues. Septic shock is clinically defined as persistent hypotension requiring vasopressor support to maintain a MAP of ≥65 mmHg and a serum lactate level >2 mmol/L, despite adequate fluid resuscitation. This state is associated with high mortality due to irreversible organ injury.

Clinical Manifestations

Sepsis presents as a clinical continuum, ranging from mild, non-specific symptoms to severe organ dysfunction and death. The manifestations evolve over time, and early recognition is essential to prevent progression to septic shock and multi-organ failure. Understanding the typical features at different stages and the patterns of organ dysfunction is crucial, as it aids in prompt identification, risk stratification, and timely intervention.

Early vs. Late Features of Sepsis

Early Features

The initial clinical signs of sepsis are often subtle and may mimic other less severe infections. These early manifestations result from the host's acute systemic inflammatory response to infection. Recognition of these early warning signs is vital to prevent deterioration.

Key early features include:

- **Fever or Hypothermia**: Fever (>38°C) is common due to cytokine-mediated hypothalamic regulation. Paradoxically, some patients, particularly the elderly and immunocompromised, may present with hypothermia (<36°C), which is associated with worse outcomes.
- **Tachycardia**: Resting heart rates exceeding 90 beats per minute are typical in early sepsis, reflecting compensatory mechanisms to maintain cardiac output.
- **Tachypnoea**: Respiratory rates often increase (>20 breaths per minute) due to metabolic acidosis, hypoxia, or the systemic inflammatory response.
- **Altered Mental Status**: Subtle changes such as confusion, agitation, anxiety, or reduced alertness may occur early, particularly in elderly patients, and often precede overt hypotension.
- **Leukocytosis or Leukopenia**: Elevated white blood cell counts (>12,000/mm^3) or reduced counts (<4,000/mm^3), often with a left shift, indicate systemic inflammation.
- **Elevated Serum Lactate**: Even in the absence of hypotension, rising lactate levels (>2 mmol/L) may reflect early tissue hypoperfusion and anaerobic metabolism.

- **Warm Shock Phase** (in some patients): In the initial phases of septic shock, peripheral vasodilation may lead to warm extremities, flushed skin, and bounding pulses, despite underlying circulatory failure.

These early signs are often non-specific, which poses a significant diagnostic challenge, especially in community and emergency settings. Delayed recognition of these features is a major contributor to the progression of sepsis.

Late Features

If sepsis is not promptly treated, it progresses to severe sepsis and septic shock, marked by widespread cellular injury, impaired tissue perfusion, and escalating organ dysfunction.

Late features include:

- **Hypotension**: Systolic blood pressure <90 mmHg or mean arterial pressure <65 mmHg, refractory to fluid resuscitation, indicates septic shock.
- **Cool, Mottled Skin**: As shock progresses, peripheral vasoconstriction develops, leading to cyanosis, mottling, and cold extremities, signalling profound circulatory failure.
- **Oliguria or Anuria**: Reduced urine output (<0.5 mL/kg/hour) reflects acute kidney injury secondary to hypoperfusion.
- **Marked Altered Mental Status**: Progression from confusion to stupor or coma due to cerebral hypoxia and metabolic disturbances.
- **Disseminated Intravascular Coagulation (DIC)**: Bleeding tendencies, petechiae, ecchymoses, and prolonged clotting times due to widespread microthrombi and consumption of clotting factors.

- **Severe Metabolic Acidosis**: Resulting from sustained anaerobic metabolism and organ failure.
- **Cytokine Storm**: In some patients, overwhelming release of inflammatory mediators can lead to refractory shock and irreversible damage.

At this stage, mortality risk significantly increases, highlighting the importance of identifying and intervening during the early phase.

Organ Dysfunction Patterns

Organ dysfunction in sepsis results from impaired perfusion, mitochondrial dysfunction, and microvascular injury. Multiple organs are often affected simultaneously, and the extent of dysfunction correlates with mortality.

1. Neurological Dysfunction

- **Sepsis-Associated Encephalopathy (SAE)** is common, presenting as delirium, confusion, decreased consciousness, and, in severe cases, coma.
- It arises from altered cerebral blood flow, blood-brain barrier dysfunction, and neuroinflammation.

2. Respiratory Dysfunction

- **ARDS** is a frequent complication, characterized by:
    - Hypoxemia refractory to oxygen therapy.
    - Bilateral pulmonary infiltrates on imaging.
    - Decreased lung compliance.

- Pulmonary dysfunction can also be exacerbated by fluid overload and infection-related lung injury.

3. Cardiovascular Dysfunction

- Persistent hypotension despite fluid resuscitation.
- Reduced myocardial contractility and ejection fraction (septic cardiomyopathy).
- Dysregulated vascular tone leading to distributive shock.

4. Renal Dysfunction

- **Acute Kidney Injury (AKI)** occurs due to hypoperfusion, nephrotoxic medications, and inflammatory damage.
- Oliguria and rising creatinine levels are key indicators.
- AKI is a strong predictor of mortality in septic patients.

5. Hepatic Dysfunction

- Cholestasis with elevated bilirubin.
- Coagulopathy due to impaired synthesis of clotting factors.
- Hepatocellular injury in advanced cases.

6. Haematological Dysfunction

- Thrombocytopenia (low platelet count).
- DIC with prolonged prothrombin time (PT), activated partial thromboplastin time (aPTT), and low fibrinogen.
- Anaemia and leukopenia may also be present.

7. Gastrointestinal Dysfunction

- Ileus, impaired motility, and gut barrier dysfunction, which may facilitate bacterial translocation and perpetuate sepsis.
- Gastrointestinal bleeding in severe cases.

8. Endocrine and Metabolic Dysfunction

- Stress hyperglycaemia due to insulin resistance.
- Adrenal insufficiency in severe cases, contributing to refractory shock.
- Lactic acidosis as a marker of ongoing hypoperfusion.

Laboratory Markers in Sepsis

Laboratory investigations play a pivotal role in the identification, assessment, and monitoring of sepsis. While clinical evaluation is essential, laboratory markers help confirm infection, assess the inflammatory response, and detect organ dysfunction.[24] They are also critical for prognostication and guiding therapeutic decisions. Among the various biomarkers, some are routinely used in clinical practice, while others are employed for risk stratification or research purposes. This section elaborates on key laboratory markers with established roles in sepsis, with an emphasis on their diagnostic and prognostic value.[25]

1. Complete Blood Count (CBC)

The CBC is one of the most basic and widely available tests, providing essential insights into the systemic response to infection. In sepsis, the CBC reveals several characteristic abnormalities:

- **Leukocytosis or Leukopenia**: An elevated total WBC count (>12,000 cells/mm^3) is frequently seen in early sepsis as part of the body's response to infection. However,

paradoxically, leukopenia (<4,000 cells/mm³) may also occur, particularly in severe cases, and is associated with poor outcomes due to immune system exhaustion.

- **Neutrophilia with Left Shift**: A predominance of neutrophils, especially immature forms like band cells, reflects acute bacterial infection. The "left shift" is indicative of heightened bone marrow activity attempting to meet the demand for neutrophils.
- **Thrombocytopenia**: Decreased platelet counts are common in sepsis and may signify developing DIC or bone marrow suppression. Persistent or worsening thrombocytopenia is associated with increased mortality.
- **Anaemia**: Normocytic, normochromic anaemia often develops as sepsis progresses, due to inflammatory cytokine-mediated suppression of erythropoiesis, blood loss, and haemolysis.

Overall, CBC provides a rapid, cost-effective snapshot of the host's hematologic response to infection and helps monitor disease progression and complications such as DIC.

2. C-Reactive Protein (CRP)

In reaction to interleukin-6 (IL-6) and other pro-inflammatory cytokines during infection and tissue damage, the liver produces C-Reactive Protein (CRP), an acute-phase reactant. After an infection begins, CRP levels rise quickly (6–12 hours), reaching their peak 48 hours later.

Role of CRP in Sepsis:

- **Diagnostic Aid**: Elevated CRP levels are commonly observed in bacterial infections, including sepsis. However, CRP is not specific to sepsis and can rise in any inflammatory condition, such as trauma, surgery, or autoimmune diseases.
- **Monitoring**: Serial CRP measurements are often more useful than a single value. A declining trend in CRP after initiation of antibiotics suggests a favourable response,

whereas persistently high or rising levels may indicate treatment failure or complications such as secondary infections.

- **Limitations**: CRP lacks specificity and may lag in early recognition. Its levels are influenced by various factors, including chronic inflammatory states and liver function.

Despite its limitations, CRP remains a valuable adjunct in the clinical assessment and monitoring of sepsis due to its wide availability and low cost.

3. Procalcitonin

In response to systemic bacterial infections, parenchymal tissues primarily produce procalcitonin (PCT), a precursor of the hormone calcitonin. Unlike CRP, procalcitonin is more specific to bacterial infections and is minimally elevated in viral infections or non-infectious inflammatory conditions.

Role of Procalcitonin in Sepsis:

- **Early Detection**: PCT levels elevate within 3–6 hours of bacterial infection onset and correlate with the severity of sepsis. Levels >2 ng/mL are suggestive of systemic bacterial infection, while levels <0.5 ng/mL may indicate a low likelihood.
- **Differentiating Infections**: Procalcitonin is particularly useful in distinguishing bacterial from viral or fungal infections, which can guide antibiotic stewardship and reduce unnecessary antibiotic use.
- **Prognostication**: High or persistently elevated PCT levels are associated with worse outcomes, including higher mortality and progression to septic shock.
- **Guiding Therapy**: Serial PCT measurements can inform decisions on the initiation or discontinuation of antibiotics. A decreasing PCT trend over time reflects therapeutic success.

Limitations:

- PCT levels can be elevated in non-infectious conditions like major surgery, severe trauma, and prolonged cardiogenic shock.
- Cost and availability may limit its routine use in resource-limited settings.

Overall, procalcitonin has emerged as a valuable biomarker in the diagnosis, prognosis, and antibiotic management of sepsis.

4. Serum Lactate – Focus on Hyperlactatemia and Its Prognostic Value

Serum lactate is a critical marker in sepsis, reflecting tissue hypoxia and impaired oxidative metabolism. Hyperlactatemia occurs when cellular oxygen delivery is insufficient, leading to anaerobic glycolysis and lactate accumulation.[26]

Mechanisms of Hyperlactatemia in Sepsis:

- **Tissue Hypoperfusion**: Due to vasodilation, capillary leakage, and microvascular thrombosis, leading to inadequate oxygen delivery.
- **Mitochondrial Dysfunction**: Even with adequate oxygen delivery, mitochondrial injury can impair oxidative phosphorylation, increasing lactate production.
- **Adrenergic Stimulation**: Increased catecholamines in sepsis enhance glycolysis, contributing to elevated lactate levels.

Prognostic Value:

- **Initial Lactate Levels**: A serum lactate >2 mmol/L is concerning, while levels >4 mmol/L are indicative of severe tissue hypoxia and are associated with higher mortality.

- **Lactate Clearance**: Beyond absolute lactate levels, the rate of lactate clearance over time (within the first 6 hours) is a powerful predictor of outcomes. Failure to clear lactate is associated with ongoing tissue hypoperfusion and poor prognosis.
- **Guiding Resuscitation**: Current sepsis management protocols emphasize lactate monitoring. Lactate-driven resuscitation aims to reduce lactate levels as a surrogate marker of improving perfusion.

Clinical Relevance:

- Elevated lactate serves as a trigger for aggressive management. The presence of hyperlactatemia, even in the absence of hypotension (termed "cryptic shock"), is indicative of occult hypoperfusion and necessitates prompt intervention.
- Serial lactate measurements are used to assess the response to fluid resuscitation and other supportive measures.

Hyperlactatemia remains one of the most critical laboratory markers in sepsis, with established roles in both diagnosis and prognostication. Its integration into the Sepsis-3 definition further underscores its clinical importance.

Lactate in Sepsis

Lactate has emerged as a cornerstone biomarker in the identification, management, and prognostication of sepsis. Elevated serum lactate levels reflect underlying disturbances in cellular metabolism and oxygen utilization, making it a critical tool in sepsis care. Understanding lactate physiology and the mechanisms contributing to hyperlactatemia in sepsis is essential for its optimal clinical application.

a. Physiology of Lactate

Normal Lactate Metabolism

A naturally occurring byproduct of the metabolism of glucose is lactate. Under normal circumstances, skeletal muscles, erythrocytes, the brain, and the gastrointestinal system are among the tissues that continually produce lactate. Normally, arterial lactate concentrations are maintained between 0.5 and 2.0 mmol/L. The major part of lactate produced is cleared by the liver (accounting for approximately 60%–70% of clearance) through gluconeogenesis and oxidation. The kidneys and skeletal muscles also contribute to lactate clearance, particularly during stress conditions.

In healthy individuals, lactate production and clearance remain in equilibrium, ensuring stable plasma levels. This balance is tightly regulated by oxygen delivery, mitochondrial function, and hormonal influences, such as catecholamines.

Anaerobic vs. Aerobic Lactate Production

Traditionally, lactate has been considered a marker of anaerobic metabolism, produced when oxygen delivery to tissues becomes insufficient to support oxidative phosphorylation. In this state, pyruvate, the end product of glycolysis, is converted into lactate via the enzyme LDH, allowing continued ATP production through glycolysis.

However, lactate production is not limited to anaerobic conditions. Under aerobic metabolism, lactate is also produced as a byproduct of increased glycolytic activity, especially during times of heightened metabolic demand (e.g., sepsis, exercise, or catecholamine surges). This phenomenon, known as “stress-induced hyperlactatemia,” occurs despite adequate oxygen availability and reflects a hypermetabolic state rather than purely hypoxia-induced anaerobiosis.

Therefore, lactate in sepsis can arise from both oxygen-independent and oxygen-dependent mechanisms, making its interpretation complex and multifactorial.

b. Causes of Elevated Lactate in Sepsis

Tissue Hypoperfusion

One of the primary causes of elevated lactate in sepsis is tissue hypoperfusion. Sepsis triggers widespread vasodilation, increased vascular permeability, and microcirculatory dysfunction, all of which impair oxygen delivery to tissues. Inadequate perfusion leads to anaerobic metabolism, driving up lactate production as cells switch from oxidative phosphorylation to glycolysis for energy. Hypoperfusion is often most pronounced in vital organs such as the kidneys, liver, and GIT, contributing to systemic lactate accumulation.[27]

Mitochondrial Dysfunction

Sepsis is increasingly recognized as a disease of bioenergetic failure, where despite adequate oxygen delivery, cells are unable to utilize oxygen effectively due to mitochondrial dysfunction. Sepsis-induced mitochondrial injury disrupts the electron transport chain, impairing ATP generation and forcing cells to rely on glycolysis, even in the presence of sufficient oxygen—a phenomenon referred to as cytopathic hypoxia. This leads to increased lactate production independent of global tissue hypoxia.

Impaired Clearance

Lactate clearance is primarily dependent on hepatic metabolism. In sepsis, hepatic dysfunction is common due to reduced perfusion (shock liver) and the direct effects of inflammatory cytokines on hepatocytes. Additionally, renal dysfunction in sepsis may contribute to decreased lactate

elimination, particularly when AKI develops. These factors combine to prolong the half-life of circulating lactate and exacerbate hyperlactatemia, even if lactate production stabilizes.

Thus, in sepsis, elevated lactate reflects a combination of overproduction (due to hypoxia and stress responses) and reduced clearance (due to organ dysfunction), making it a robust marker of disease severity.

c. Clinical Use of Lactate

As a Marker of Tissue Hypoxia

Lactate has long been utilized as a surrogate marker for global tissue hypoxia and hypoperfusion in critically ill patients. Elevated lactate levels, particularly in the context of hypotension or organ dysfunction, suggest inadequate oxygen delivery or utilization. Hyperlactatemia often precedes other signs of shock, making it a valuable early indicator of impending circulatory failure. As such, lactate measurement is included in sepsis guidelines for the early identification of high-risk patients and is a core component of the Surviving Sepsis Campaign (SSC) recommendations.

Role in Early Goal-Directed Therapy (EGDT)

The concept of **EGDT**, popularized by **Rivers et al**. in 2001, emphasized early recognition and aggressive management of septic shock, with lactate levels serving as a key marker for guiding resuscitation. In EGDT protocols, elevated lactate (>4 mmol/L) triggered fluid resuscitation, vasopressor support, and oxygenation strategies to optimize tissue perfusion and oxygen delivery.

Subsequent large-scale trials (ProCESS, ARISE, ProMISe) questioned the superiority of strict EGDT protocols over standard care. However, lactate monitoring remained integral to both strategies, highlighting its value in identifying patients who require intensive resuscitative efforts.

Lactate Clearance as an Indicator of Treatment Response

Lactate clearance refers to the reduction in serum lactate levels over time following therapeutic interventions. A lactate clearance of ≥10% within the first 6 hours of resuscitation is associated with improved survival in septic patients. Rapid lactate normalization indicates effective restoration of perfusion and metabolic recovery, while persistently elevated levels suggest ongoing hypoperfusion, treatment failure, or evolving organ dysfunction.[28]

Incorporating lactate clearance into sepsis management protocols provides dynamic feedback on the adequacy of interventions, helping guide ongoing resuscitation efforts and predict outcomes.

d. Lactate Levels and Mortality Correlation

There is a strong correlation between higher lactate levels and higher sepsis mortality, according to numerous research. The relationship between lactate concentration and mortality is dose-dependent, with higher lactate levels portending worse outcomes. For example, patients with lactate levels:

- **<2 mmol/L** typically have a favourable prognosis.
- **2–4 mmol/L** are at intermediate risk and warrant close monitoring.
- **>4 mmol/L** face significantly increased mortality rates, often exceeding 40%–50% in the presence of septic shock.

Importantly, it is not just the initial lactate value but also the trajectory over time that matters. Failure to achieve lactate clearance within the early hours of treatment is independently associated with higher mortality. Persistent hyperlactatemia indicates ongoing circulatory dysfunction, mitochondrial injury, or inadequate therapy.

Incorporating lactate into risk stratification models enhances the ability to predict outcomes and allocate resources effectively. Given its strong prognostic power, the quantification of lactate is an advised element within the Sepsis-3 criteria, which includes hyperlactatemia as a criterion for identifying septic shock.

Scoring Systems in Sepsis

Modified Early Warning Score (MEWS)

A popular bedside tool for assisting in the early detection of clinical deterioration in hospitalized patients is the MEWS. It is a simple, physiology-based scoring system that helps predict patient outcomes and trigger timely medical interventions, particularly in acute settings such as sepsis. Understanding the historical evolution, the physiological parameters involved, and the utility of MEWS in sepsis is essential for its application in clinical practice.[6]

Historical Background and Development

The development of early warning scores originated from the increasing recognition that many in-hospital adverse events, such as cardiac arrest, unplanned ICU admissions, and unexpected deaths, are often preceded by measurable physiological deterioration. In the late 1990s, studies highlighted that abnormal vital signs could predict these adverse outcomes if identified early. In response, the Early Warning Score (EWS) was first introduced in the United Kingdom as a standardized method to detect patient deterioration based on vital sign abnormalities.

The MEWS evolved as an adaptation of the original EWS, aiming to enhance the tool's sensitivity and specificity by refining the scoring of physiological parameters and incorporating the AVPU scale for assessing consciousness. MEWS was designed to be easily applied at the bedside, enabling nurses and clinicians to monitor patients effectively and trigger rapid response

interventions. Over time, MEWS has become a fundamental component of many hospital observation protocols and has demonstrated particular relevance in the context of sepsis management, where early detection of clinical decline is paramount.

Physiological Parameters Used

MEWS is calculated based on five routinely measured physiological parameters, each assigned a weighted score according to the degree of deviation from normal values. These parameters are chosen due to their strong association with critical illness and impending deterioration.

Heart Rate

An elevated heart rate (tachycardia) is one of the earliest signs of systemic infection and sepsis. In MEWS, heart rate thresholds are stratified to assign higher scores as the pulse deviates from normal ranges, reflecting the body's compensatory response to infection, hypovolemia, and metabolic stress. Persistent tachycardia in septic patients may suggest ongoing shock or inadequate resuscitation.

Respiratory Rate

Often the first physiological alteration in sepsis, respiratory rate is one of the most sensitive indicators of clinical decline. Increased respiratory rate (tachypnoea) reflects metabolic acidosis, hypoxia, or systemic inflammation. MEWS assigns significant weight to respiratory rate abnormalities, acknowledging its predictive value for acute deterioration, especially in septic patients progressing to respiratory failure.

Systolic Blood Pressure

Systolic blood pressure (SBP) in MEWS is a vital marker of circulatory status. Hypotension in sepsis, typically defined as SBP <90 mmHg, is indicative of septic shock and inadequate perfusion. MEWS captures both hypotensive and hypertensive extremes, with low SBP scoring

higher due to its correlation with severe outcomes. Monitoring SBP trends is crucial for recognizing early circulatory compromise in sepsis.

Temperature

Fever is a hallmark of infection, but temperature abnormalities in sepsis can range from hyperthermia (>38°C) to hypothermia (<36°C). Both extremes are associated with increased mortality, with hypothermia indicating a failure of the host immune response. MEWS incorporates temperature to reflect the systemic inflammatory response and guide clinical suspicion of infection.

Level of Consciousness (AVPU Scale)

The AVPU scale (Alert, responds to Voice, responds to Pain, Unresponsive) is integrated into MEWS to assess neurological status. Altered mental status in sepsis can result from cerebral hypoperfusion, metabolic encephalopathy, or sepsis-associated delirium. A deterioration from full alertness to decreased responsiveness is heavily weighted in MEWS, signaling significant clinical decline and the need for urgent intervention.

Interpretation of MEWS Values

MEWS scores typically range from 0 to 14, with higher scores indicating greater physiological derangement and a higher risk of adverse outcomes. Thresholds for action are institution-specific but often follow this framework:[29]

- **MEWS 0–2:** Low risk; routine monitoring.
- **MEWS 3–4:** Moderate risk; increased observation frequency.
- **MEWS ≥5:** High risk; triggers rapid response team (RRT) or critical care evaluation.

In the context of sepsis, a MEWS of **≥5** is frequently associated with impending septic shock or multi-organ failure and warrants immediate escalation of care. Serial MEWS calculations help track patient trajectory, with rising scores indicating progressive deterioration.

Role of MEWS in Early Identification of Clinical Deterioration

The primary purpose of MEWS is to facilitate the early detection of clinical deterioration before irreversible organ dysfunction occurs. In sepsis, early identification and intervention are vital, as delayed recognition is closely linked to increased mortality. MEWS provides an objective framework for frontline healthcare providers to identify patients at risk, initiate sepsis protocols, and prompt rapid resuscitation efforts.[30]

Studies have demonstrated that incorporating MEWS into hospital workflows reduces in-hospital cardiac arrests, ICU transfers, and mortality rates. In septic patients, MEWS has been shown to corelate with disease severity, predict ICU admission, and guide decisions regarding escalation of care. By emphasizing continuous monitoring of physiological parameters, MEWS ensures timely recognition of sepsis progression from early infection to severe sepsis and septic shock.

Strengths and Limitations in Sepsis Patients

Strengths

MEWS offers several advantages in sepsis management:

- **Simplicity and feasibility:** MEWS uses routinely collected vital signs without the need for complex laboratory tests.
- **Early detection:** Captures subtle physiological changes before overt clinical deterioration occurs.

- **Standardization:** Provides a uniform language for communication between healthcare providers and facilitates timely interventions.
- **Prognostic value:** Higher MEWS scores are associated with worse outcomes, helping identify high-risk septic patients.

Limitations

Despite its utility, MEWS has several limitations, particularly in sepsis:

- **Limited specificity:** Elevated MEWS scores are not exclusive to sepsis and may be triggered by other conditions like trauma, haemorrhage, or cardiac events.
- **Over-reliance on static thresholds:** Single-time-point measurements may miss dynamic clinical changes, potentially delaying interventions.
- **Underperformance in elderly and immunocompromised patients:** These populations may not mount typical physiological responses (e.g., fever or tachycardia), resulting in falsely low MEWS.
- **Neurological assessment variability:** Subjectivity in applying the AVPU scale can lead to inconsistent scoring, particularly in patients with pre-existing cognitive impairment.

In summary, while MEWS is a valuable tool for recognizing early clinical deterioration in sepsis, it should be integrated with clinical judgment, laboratory markers (like lactate), and other scoring systems (such as SOFA or qSOFA) for comprehensive patient assessment.

Sequential Organ Failure Assessment (SOFA) Score

To gauge the extent of organ failure in critically ill patients, particularly those with sepsis, a validated, objective scoring system known as the SOFA Score was developed. By assessing multiple organ systems, the SOFA score provides a standardized method for evaluating the

severity of illness and predicting morbidity and mortality in intensive care settings. Its use has been integral in sepsis management, as it facilitates the identification of organ failure, guides therapeutic interventions, and allows continuous monitoring of patient status over time.[31]

Development and Purpose

The SOFA score was originally introduced in 1996 by the ESICM as part of an effort to create a simple, reproducible tool for assessing the degree of organ dysfunction in ICU patients, particularly in the setting of sepsis and multiple organ failure. The main objective of SOFA was not to directly predict mortality rates, but rather to articulate the degree of organ impairment or failure over a time period, thereby offering healthcare professionals a dynamic representation of a patient's clinical progression.[9]

In 2016, with the publication of the Sepsis-3 definitions, the SOFA score gained prominence as a critical component in defining sepsis. Sepsis is defined in these updated guidelines as a potentially lethal organ failure caused by a dysregulated host response to infection. A SOFA score increase of ≥2 points from baseline in the presence of infection indicates significant organ dysfunction and is associated with increased mortality risk. This shift placed SOFA at the centre of modern sepsis diagnosis and management.

Components of SOFA

SOFA evaluates six key organ systems; each scored from 0 (normal function) to 4 (severe dysfunction) based on specific clinical and laboratory parameters. The cumulative score ranges from 0 to 24, with higher scores reflecting greater degrees of organ failure.[32]

Respiratory Function (PaO_2/FiO_2 Ratio)

The assessment of respiratory function in SOFA is based on the arterial oxygen partial pressure (PaO_2) divided by the fraction of inspired oxygen (FiO_2) ratio. This metric evaluates the efficiency

of pulmonary gas exchange. A normal PaO_2/FiO_2 ratio is greater than 400 mmHg. Ratios below this threshold indicate varying degrees of hypoxemia, with scores escalating as oxygenation deteriorates. In septic patients, acute respiratory failure due to ARDS or sepsis-induced pulmonary capillary leakage is common, making this parameter crucial for early identification of respiratory compromise.

Coagulation (Platelet Count)

The coagulation component of SOFA is measured by platelet count, reflecting the function of the haematological system. Thrombocytopenia is frequently observed in sepsis due to consumption coagulopathy, disseminated intravascular coagulation (DIC), and bone marrow suppression. Platelet counts below 150,000/µL warrant increasing SOFA scores, with severe thrombocytopenia (<20,000/µL) indicating profound coagulation dysfunction and a higher risk of bleeding complications.

Liver Function (Bilirubin Levels)

Liver dysfunction in SOFA is evaluated by serum bilirubin levels. Hyperbilirubinemia in sepsis can result from hepatic hypoperfusion, cholestasis, or direct hepatocellular injury caused by inflammatory mediators. Bilirubin levels above 1.2 mg/dL contribute to the score, with progressively higher scores for levels exceeding 12 mg/dL. Liver dysfunction serves as a marker of systemic inflammation and multi-organ involvement in sepsis.

Cardiovascular System (Hypotension and Vasopressor Requirement)

Cardiovascular assessment in SOFA is based on mean arterial pressure (MAP) and the need for vasopressor support. Hypotension, defined as MAP <70 mmHg, reflects inadequate perfusion. Septic shock often necessitates vasopressors such as norepinephrine to maintain target perfusion pressures. SOFA scores increase with escalating vasopressor requirements, distinguishing between

mild hypotension and refractory shock states needing multiple vasopressors to sustain hemodynamic.

Central Nervous System (Glasgow Coma Scale)

Neurological function is measured using the Glasgow Coma Scale (GCS), which evaluates consciousness based on eye-opening, verbal, and motor responses. Septic encephalopathy can lead to altered mental status due to systemic inflammation, cerebral hypoperfusion, or metabolic derangements. A fully alert patient with a GCS of 15 scores zero points, while lower GCS scores indicate worsening neurological dysfunction, with a GCS ≤6 scoring the maximum points for this parameter.

Renal Function (Creatinine and Urine Output)

Renal function is assessed by serum creatinine levels and urine output. Acute kidney injury (AKI) in sepsis arises from hypoperfusion, nephrotoxic medications, and inflammatory injury. A creatinine level above 1.2 mg/dL or urine output below 500 mL/day signals renal impairment. Severe renal dysfunction (creatinine >5.0 mg/dL or anuria) maximizes the SOFA score for this organ system and is strongly associated with poor outcomes in sepsis.

Scoring Method and Interpretation

Each organ system is scored from 0 to 4 points based on the degree of dysfunction, and the total SOFA score is the sum of these individual scores. The higher the SOFA score, the greater the extent of organ failure. While no absolute cutoff exists, certain thresholds provide clinical guidance:

- **Baseline SOFA of 0–1**: Normal or near-normal organ function.
- **An increase of ≥2 points**: Suggestive of sepsis in the presence of infection.

- **SOFA ≥11**: Associated with extremely high mortality (>90% in some studies).

The SOFA score is not static and is intended to be calculated daily or more frequently in unstable patients. Continuous monitoring allows clinicians to assess the progression or resolution of organ dysfunction, guide treatment adjustments, and evaluate prognosis.

SOFA Score as a Dynamic Marker (Daily Trend Monitoring)

A key feature of the SOFA score is its use as a dynamic marker. Rather than relying on a single time-point measurement, serial SOFA scores allow tracking of the patient's trajectory in response to therapy. In septic patients, a decreasing SOFA score over the first 48–72 hours of ICU admission is associated with improved survival, while persistent or rising scores indicate ongoing or worsening organ failure and higher mortality risk.

Dynamic assessment also aids in clinical decision-making, such as escalation of care, need for additional organ support (like renal replacement therapy), and evaluating the effectiveness of interventions, including antibiotics, fluid resuscitation, and vasopressor use.

Strengths and Limitations

Strengths

The SOFA score's strengths lie in its ability to:

- Provide a quantifiable, objective indicator of organ dysfunction.
- Forecast the prognosis of critically ill patients, especially those with sepsis.
- Facilitate communication between healthcare providers through standardized scoring.
- Serve as a monitoring tool to track patient progress over time.
- Support sepsis definitions and clinical research protocols.

Limitations

Despite its utility, SOFA has certain limitations:

- Requires laboratory measurements (e.g., PaO_2, bilirubin, creatinine), which may delay scoring in resource-limited settings.
- May underestimate organ dysfunction in early sepsis before laboratory abnormalities manifest.
- Neurological scoring using GCS can be confounded by sedation, intubation, or pre-existing neurological deficits.
- Variability in baseline organ function (e.g., chronic kidney disease) may affect scoring accuracy.
- Its application is primarily validated in ICU settings and may be less practical for use in emergency departments or general wards.

Acute Physiology and Chronic Health Evaluation (APACHE II)

The APACHE II score is one of the most widely used and extensively validated severity-of-illness scoring systems in critical care medicine. It was designed to objectively assess the severity of disease, predict patient outcomes, and stratify mortality risk among critically ill patients. APACHE II has been foundational in both clinical practice and research, particularly for benchmarking ICU performance, guiding resource allocation, and facilitating prognostic discussions.[33]

Historical Context and Evolution

The original APACHE scoring system was first introduced in 1981 by **Knaus et al.**, marking a significant advance in critical care by quantifying illness severity based on physiological derangements. This early model underwent refinement, leading to the development of APACHE II

in 1985, which offered improved usability, fewer variables, and enhanced predictive capabilities. Unlike its predecessor, APACHE II reduced the number of physiological variables while incorporating adjustments for age and chronic health conditions, making it a more practical tool for routine clinical application.

Since its introduction, APACHE II has become one of the most cited scoring systems in intensive care research. Although newer iterations such as APACHE III (1991) and APACHE IV (2006) have been developed with more complex algorithms and improved calibration, APACHE II remains prevalent globally due to its simplicity, wide validation, and accessibility, particularly in resource-limited settings.

Variables Assessed

Three primary factors are used to generate the APACHE II score: age points, chronic health points, and acute physiological data. Based on the severity of the illness during ICU admission, the combined score estimates the risk of death.

Physiological Measurements (12 Parameters)

The core of the APACHE II scoring system consists of 12 routinely available physiological variables; each assigned a point value from 0 (normal) to 4 (most severely deranged). These variables reflect critical body systems and serve as indicators of homeostatic stability. The 12 parameters include:

1. **Temperature (°C)** – Hypothermia and hyperthermia reflect systemic inflammatory responses and metabolic disturbances.
2. **Mean Arterial Pressure (MAP)** – Critical for assessing perfusion and cardiovascular function.

3. **Heart Rate (beats per minute)** – Tachycardia and bradycardia may reflect shock states or autonomic dysfunction.
4. **Respiratory Rate (breaths per minute)** – Reflects respiratory compensation or distress.
5. **Arterial pH** – Indicates acid-base balance and respiratory/metabolic derangements.
6. **Serum Sodium (mmol/L)** – Hyponatremia and hypernatremia signify fluid and electrolyte imbalances.
7. **Serum Potassium (mmol/L)** – Abnormalities can cause life-threatening cardiac arrhythmias.
8. **Serum Creatinine (mg/dL)** – Reflects renal function and perfusion.
9. **Haematocrit (%)** – Provides information on blood viscosity and oxygen-carrying capacity.
10. **White Blood Cell Count (cells/mm^3)** – High or low values may indicate infection, bone marrow suppression, or systemic inflammation.
11. **Glasgow Coma Scale (GCS)** – Evaluates neurological function and consciousness.
12. **Arterial Oxygenation (PaO_2 or A–a gradient)** – Reflects pulmonary gas exchange efficiency, with adjustments for ventilator status.

These physiological variables are typically measured within the first 24 hours of ICU admission, and the most extreme values during this period are used for scoring.

Age Points

Age serves as an autonomous prognostic factor for mortality in patients experiencing critical illness. APACHE II assigns additional points based on patient age:

- **0 points** for ≤ 44 years,
- **2 points** for 45 – 54 years,
- **3 points** for 55 – 64 years,
- **5 points** for 65 – 74 years,
- **6 points** for ≥ 75 years.

The incremental increase acknowledges the higher vulnerability and reduced physiological reserve with advancing age.

Chronic Health Points

Chronic health status is also factored into APACHE II, recognizing that pre-existing comorbidities impact mortality risk. If the patient is immunocompromised or has severe organ system insufficiency, chronic health points are added, specifically if:

- **Non-operative patients** or **emergency postoperative patients** with severe chronic disease receive 5 points.
- **Elective postoperative patients** with similar chronic conditions receive 2 points.

Conditions qualifying for chronic health points include severe chronic heart, lung, liver, or renal disease and immunosuppression due to therapy, malignancy, or immune disorders.

Scoring and Interpretation

The APACHE II score ranges from 0 to 71, with higher scores reflecting greater disease severity and higher predicted mortality. The total score is calculated by summing:

- The worst acute physiology points,
- The age points,
- The chronic health points.

Mortality risk increases exponentially with higher scores. For example:

- Scores ≤10 are typically associated with low mortality (<15%),
- Scores around 20 correlates with approximately 40% mortality,
- Scores ≥30 are linked with >75% mortality.

While the APACHE II score provides a numerical prediction, it is most accurate when applied to populations rather than individual patients due to interindividual variability and the influence of unmeasured factors.

APACHE II as a Mortality Predictor

APACHE II was designed as a probabilistic model to forecast the likelihood of hospital mortality in patients experiencing critical illness, independent of their specific diagnosis. It serves as a benchmark for ICU performance comparison, clinical research stratification, and as an adjunct to prognosis counselling.

Numerous studies have validated APACHE II's accuracy in predicting outcomes across diverse patient populations, including those with sepsis, trauma, postoperative complications, and respiratory failure. However, its predictive validity diminishes when used in isolation for individual decision-making due to its reliance on early physiological data, which may evolve during ICU care.

In the context of sepsis, APACHE II is particularly useful in establishing baseline severity and facilitating early risk stratification, enabling targeted monitoring and resource prioritization.

Complexity and Practical Challenges in Emergency Settings

Despite its strengths, APACHE II presents several practical challenges, especially in acute or resource-limited environments:

- **Data Collection Requirements**: APACHE II depends on a comprehensive set of laboratory and physiological data. Delays in obtaining arterial blood gases, biochemistry panels, or GCS assessments can postpone scoring and diminish real-time applicability.
- **24-Hour Window**: The requirement to record the most abnormal values during the first 24 hours of ICU admission limits its utility in emergency departments (ED), where rapid decision-making is necessary. In emergent settings, earlier scoring systems such as MEWS or qSOFA may be more practical.
- **Complexity of Calculation**: APACHE II scoring involves multiple steps and variables, which can introduce calculation errors, particularly without electronic aids or scoring tools.
- **Static Assessment**: Unlike dynamic scores (such as SOFA), APACHE II does not account for changes in patient condition after the initial 24-hour window. This makes it less suitable for ongoing monitoring of treatment response.
- **Resource Constraints**: In low-resource settings, limited access to necessary laboratory tests and monitoring equipment can prevent full scoring, reducing its clinical utility.

Despite these limitations, APACHE II remains a cornerstone in ICU prognostication and continues to be extensively applied due to its balance of comprehensiveness and clinical relevance.

Combination of MEWS and Lactate in Sepsis Prognostication

Sepsis remains a leading cause of morbidity and mortality worldwide, particularly in critical care and emergency settings. Timely identification and risk stratification of septic patients are essential to improving outcomes. While numerous scoring systems and biomarkers are available, combining a rapid bedside physiological scoring tool like the MEWS with a critical metabolic biomarker such as serum lactate has been proposed as a synergistic approach to enhance early prognostication in sepsis. This combination aims to integrate real-time clinical deterioration signs with underlying cellular and metabolic dysfunction to better predict adverse outcomes and guide interventions.

The Theoretical Basis of Combining a Physiological Score with a Metabolic Marker

The pathophysiology of sepsis involves a complex interaction of host immune responses, circulatory abnormalities, and organ dysfunction, all of which can present variably across patients. No single parameter reliably predicts deterioration or mortality; hence, multi-dimensional assessment tools are advocated.

Physiological scores, such as MEWS, capture deviations in vital signs that reflect systemic instability. However, these may be late manifestations of profound underlying disturbances. **Metabolic markers**, particularly lactate, reflect cellular-level dysfunction, notably impaired oxygen utilization and anaerobic metabolism, which often precede overt clinical signs.

By combining these distinct but complementary domains, clinicians may detect high-risk patients more accurately. The rationale lies in:

- **Physiology (MEWS)**: Detecting observable systemic changes through vital signs.
- **Biochemistry (Lactate)**: Identifying hidden metabolic crises indicative of hypoperfusion.

This dual-layered approach aligns with the multifactorial nature of sepsis and supports earlier and more aggressive intervention in patients at high risk.

How MEWS Provides Real-Time Bedside Assessment

The MEWS, which measures irregularities in fundamental physiological indicators such heart rate, respiration rate, blood pressure, temperature, and level of consciousness, is intended to detect patients who are at risk of acute clinical deterioration. These variables are routinely measured and require no specialized equipment, allowing for:

- Continuous, non-invasive monitoring at the bedside.
- Immediate flagging of abnormal trends through cumulative scoring.
- Early detection of systemic derangement before the onset of organ failure.

In sepsis, the progression from systemic inflammatory response to severe sepsis and septic shock is often heralded by subtle changes in vital signs. MEWS enables early recognition of this trajectory by providing a structured framework for the nursing and medical staff to monitor and respond to deterioration, triggering timely escalation of care and intervention.

However, MEWS may fail to detect patients whose vital signs are within acceptable ranges despite underlying circulatory compromise, such as occult hypoperfusion, which underscores the need for adjunctive markers like lactate.

How Lactate Captures Underlying Perfusion Abnormalities

Serum **lactate** is a well-established biomarker of tissue hypoxia and impaired perfusion, commonly elevated in septic patients due to:[34]

- **Anaerobic metabolism** from inadequate oxygen delivery.
- **Mitochondrial dysfunction** impairing oxidative phosphorylation.

- **Microcirculatory failure**, where perfusion deficits persist despite normal macrocirculatory parameters.

Lactate elevation, particularly levels exceeding 2 mmol/L, has been independently associated with increased mortality in sepsis. It serves as a critical indicator of:

- Global tissue hypoperfusion, even before hypotension develops.
- Severity of metabolic stress in critically ill patients.
- Response to resuscitation efforts when serially measured.

Importantly, lactate elevation may occur without overt changes in vital signs, making it a sensitive early marker of occult shock and impending organ failure. Therefore, lactate measurement complements MEWS by adding metabolic insight into the patient's condition.

Synergistic Potential in Predicting Severity and Outcome

The combination of MEWS and lactate addresses both clinical instability and cellular distress, providing a broader and more accurate risk assessment framework. Several studies have demonstrated that:

- Patients with high MEWS and elevated lactate levels have significantly higher mortality rates than those with abnormalities in only one of these parameters.
- The predictive value of combining MEWS and lactate surpasses either metric alone, particularly in detecting patients at risk of rapid deterioration.
- Integrating lactate into early warning systems enhances prognostication and identifies patients who may benefit from aggressive intervention or ICU transfer.

The synergistic model works on the principle that:

- **MEWS ≥5** (or institution-defined thresholds) flags current physiological derangement.

- **Lactate ≥2 mmol/L** suggests underlying hypoperfusion, even if MEWS is low.
- When both are abnormal, the likelihood of progression to septic shock, organ failure, and death substantially increases.

Thus, combining MEWS and lactate supports a tiered risk stratification approach, where patients are escalated in care intensity based on combined physiological and biochemical evidence of severe disease.

Benefits in Low-Resource and High-Acuity Settings

In settings with limited resources or high patient volumes, such as busy emergency departments or under-resourced healthcare facilities, the combination of MEWS and lactate offers several distinct advantages:

- **Rapid and accessible**: MEWS uses basic vital signs, which are universally available. Point-of-care lactate testing is increasingly affordable and portable.
- **Early identification of high-risk patients**, enabling prioritized resource allocation.
- **Reduces reliance on complex scoring systems** that require laboratory parameters unavailable in real-time.
- **Supports protocolized sepsis pathways**, where MEWS triggers initial assessment and lactate refines the urgency and aggressiveness of interventions.
- **Minimizes missed cases of occult sepsis**, where vital signs alone may not reflect critical illness.

Especially in environments where intensive care beds are limited or delayed transfers are common, the ability to rapidly identify patients with both elevated MEWS and lactate levels can help focus intensive therapies on those with the greatest need and potential for benefit.

Importance of Prognostic Scores in Clinical Decision-Making

In the complex and dynamic management of sepsis, the early identification of patients at risk for deterioration is critical. Prognostic scoring systems, such as MEWS, SOFA, and APACHE II, provide objective frameworks for clinicians to assess disease severity, predict outcomes, and guide therapeutic decisions. These scores integrate physiological, biochemical, and clinical parameters to offer a quantifiable estimate of patient risk. Their role in improving sepsis outcomes is increasingly recognized, particularly in high-acuity settings where rapid decision-making is essential. Below are key domains where prognostic scores contribute to optimized care delivery.

Risk Stratification and ICU Admission Decisions

One of the primary utilities of prognostic scores in sepsis is the stratification of patients based on their risk of clinical deterioration or death. Accurate risk assessment helps to:

- Identify patients who require intensive monitoring, early organ support, and invasive interventions.
- Differentiate between patients who can be safely managed in general wards versus those needing escalation to high-dependency or intensive care units (ICU).
- Guide clinicians in prioritizing limited ICU beds, especially during periods of resource strain.

For example:

- A high SOFA score indicates multiple organ dysfunction and is associated with increased ICU mortality.

- Persistently elevated lactate levels signal ongoing tissue hypoperfusion, even if the patient is hemodynamically stable, advocating for critical care admission.
- A rising MEWS may prompt immediate evaluation and transfer to a higher level of care to pre-empt irreversible deterioration.

Through this objective stratification, prognostic scores reduce subjective variability in admission decisions and ensure timely escalation for those most at risk.

Triage and Resource Allocation

In environments where healthcare resources are limited—such as during sepsis surges, pandemics, or in under-resourced facilities—prognostic scores are indispensable for triage and judicious resource allocation. These tools help prioritize care by:

- Distinguishing patients who are likely to benefit from aggressive interventions from those with limited prognostic potential.
- Aiding in the fair and evidence-based distribution of ICU beds, mechanical ventilators, and vasopressors.
- Preventing under- or over-utilization of critical care services by guiding staff in identifying patients whose condition warrants high-intensity support.

For instance, in resource-limited settings, combining a physiological score (like MEWS) with a metabolic marker (such as lactate) enables rapid bedside triage that supports early goal-directed therapy while focusing resources where they are most impactful.

Furthermore, standardized prognostic scores promote transparency and consistency in triage decisions, which is essential for ethical care delivery during crises.

Early Aggressive Management Planning

Sepsis is a time-sensitive condition, where delays in identification and treatment increase mortality. Prognostic scores support the early identification of patients who require immediate, aggressive interventions such as

- Prompt initiation of broad-spectrum antibiotics.
- Aggressive fluid resuscitation.
- Early vasopressor support for refractory hypotension.
- Close monitoring of organ functions with serial assessments.

By quantifying illness severity at presentation and during ongoing management, these scores enable:

- The tailoring of treatment intensity based on dynamic risk.
- Objective monitoring of patient response to therapy.
- Early detection of treatment failure, prompting escalation or changes in therapeutic strategy.

For example, lactate-guided resuscitation protocols have demonstrated improved outcomes when lactate clearance targets are met, and SOFA trend monitoring helps track organ function progression to inform treatment adaptation.

Overall, integrating prognostic scores into early management algorithms helps move care from a reactive to a proactive approach, improving survival rates.

Communication with Patient Families About Prognosis

Prognostic scores provide a scientifically grounded framework for discussing disease severity and expected outcomes with patients and their families. Effective communication regarding prognosis is vital in sepsis care to:

- Set realistic expectations about the likely course of illness.
- Facilitate shared decision-making regarding treatment goals, including the appropriateness of escalation or palliative approaches.
- Provide objective evidence to support discussions about the probability of recovery or risk of mortality.

For example:

- A high APACHE II score correlates with increased mortality risk and can help guide conversations about prognosis and potential outcomes.
- Persistently elevated lactate levels despite resuscitation are associated with a poor prognosis, signalling the need for honest and empathetic communication about potential limitations of care.

Furthermore, prognostic scores assist in advance care planning, where discussions regarding do-not-resuscitate (DNR) orders, goals of care, and long-term outcomes can be anchored in validated clinical data rather than subjective impressions alone.

COMPARITIVE STUDIES:

Khwannimit B et al. (2019) conducted a retrospective study in predicting outcomes among sepsis patients admitted to Intensive Care Unit comparing SOFA, qSOFA with Search out Severity score, Modified version of Early warning score and National Early Warning score. Among the 1,589 patients analysed, the SOFA score exhibited the most significant predictive precision for mortality within the hospital setting, as indicated by an AUC of 0.880, closely followed by the SOS score (0.878), indicating nearly equivalent performance. The study concluded that the SOS score can serve as an effective alternative to the SOFA score for mortality prediction and risk stratification in ICU sepsis patients, highlighting the potential utility of early warning scores in critical care settings.[35]

In a prospective cohort study done by **Chen Y et al**. (2014) , prognostic value of lactate was assessed in sepsis patients presenting to ED along with its potential to improve conventional scoring systems like SOFA, APACHE II and MEDS (Mortality in Emergency Department Sepsis) and the results has shown that the lactate levels and all the scores were higher in non-survivors in comparison to survivors ($p < 0.001$) and also found that each one of them predicted 28-day mortality. The AUC for lactate (0.79) surpassed the individual scores of MEDS (0.74), APACHE II (0.74), and SOFA (0.75). Furthermore, combining lactate with these scoring systems improved their predictive accuracy (AUCs of 0.81–0.82), outperforming the scores alone. The study concluded that lactate has more prognostic significance as a marker in septic patients in the ED and can significantly enhance the predictive power of established severity scores for mortality risk.[36]

A similar kind of study was done by **Yoo J et al**. (2015) by combining blood lactate (BLA) and MEWS to forecast transfer to ICU and mortality among hospitalized patient with sever sepsis/septic shock within 28 days. In this study Out of 100 patients 38 required ICU admission.

Patients transferred to ICU has higher MEWS (7.37 vs 4.85) and lactate levels (5 mmol/L vs 2.19 mmol/L) to those admitted in general wards. Combining Lactate with MEWS has shown superior predictive accuracy for ICU transfer (C-statistics: 0.898) compared to MEWS alone (0.816, p = 0.019). The 28 - day mortality rate was 19%, with MEWS identified as the only significant predictor of mortality (odds ratio 1.462; 95% CI: 1.122–1.905; p = 0.005). The study concluded that combining MEWS with lactate levels can enhance the prediction of ICU transfer in patients with severe sepsis/septic shock, although MEWS alone was more closely associated with short-term mortality.[37]

To anticipate the likelihood of mortality in hospitals for individuals diagnosed with severe sepsis, **Luo X et al**. (2009) examined the predictive capabilities of the Multiple Organ Dysfunction Score (MODS), SOFA, and Logistic Organ Dysfunction Score (LODS). MODS, LODS, SOFA and APACHE II scores were recorded upon admission and at their peak during hospitalization for 403 ICU patients. Results showed that LODS had the highest predictive accuracy at admission (AUC 0.811), followed by SOFA (0.787), APACHE II (0.770), and MODS (0.725). Maximum scores during hospitalization were significantly more accurate than admission scores ($P < 0.01$). Both LODS and SOFA outperformed MODS ($P < 0.01$), though there was no significant difference between LODS and SOFA ($P > 0.05$). LODS also performed better than APACHE II ($P < 0.01$), while no significant differences were observed between APACHE II, SOFA, and MODS ($P > 0.05$). The study concluded that LODS, SOFA, and MODS are useful predictors of mortality in severe sepsis, with maximum LODS providing the highest discriminatory power.[38]

In a study by **Jaiswal P et al. (2022)**, to find out how well serial serum lactate measurements predict mortality in critically ill sepsis patients, their prognostic performance was compared with two well-known ICU scoring systems: the SOFA and the APACHE IV. From August 2019 to September 2021, a total of 280 adult individuals diagnosed with sepsis and subsequently admitted to the intensive care unit were incorporated into this cross-sectional investigation. The

investigation indicated an ICU fatality rate of 43.21%. On the 3rd day, serum lactate levels (AUC 0.909; 95% CI: 0.867 to 0.941) were found to be equivalent to the APACHE IV score on that day (AUC 0.931; 95% CI: 0.893 to 0.960), and they even surpassed the SOFA score that was measured on day three (AUC 0.936; 95% CI: 0.898 to 0.963), based on the Receiver Operating Characteristic (ROC) curve analysis. The APACHE IV score on day three emerged as the most precise predictor of mortality, establishing a threshold of >132, with a 91.0% probability of accurately forecasting mortality outcomes. To predict mortality rates among individuals afflicted with sepsis, the research identified that repeated serum lactate assessments are dependable and analogous to SOFA and APACHE IV scoring systems. Moreover, they may function as a significant surrogate indicator, especially in resource-constrained rural environments.[39]

Su X et al. (2023) conducted a retrospective investigation aimed at evaluating the prognostic significance of APACHE II, SOFA, qSOFA, and MEWS scoring systems in relation to mortality rates among critically ill patients admitted to an ICU of a secondary hospital. Among 126 adult patients analysed from October 2022 to April 2023, 45 died and 81 improved. Univariate analysis showed significant differences in several clinical parameters, including procalcitonin, serum creatinine, blood urea nitrogen, albumin, coagulation markers, lactate levels, and the four scores (all $P < 0.05$). Multivarriate logistic regression identified APACHE II, SOFA, MEWS, and APTT as independent risk factors for mortality. ROC analysis revealed that all four scores effectively predicted mortality, with SOFA having the highest accuracy (AUC = 0.808). MEWS was the quickest to calculate [(1.03 ± 0.39) minutes], while APACHE II took the longest [(2.81 ± 1.04) minutes] ($P < 0.001$). The study concluded that SOFA is the most accurate predictor of mortality, while MEWS is the most time-efficient, supporting their use in secondary hospitals for early identification and management of critically ill patients.[40]

Morkar D et al. (2022) conducted a one-year prospective study to compare the effectiveness of SOFA, APACHE II, and SAPS II scores in predicting mortality among 100 sepsis patients

admitted in ICU by calculating score at 24 hours and 48 hours of admission and to evaluate the benefit of combining these scores. The overall mortality rate was 51%, with higher mortality among females (68.63%) and the elderly (39.22% in those aged 60–79 years). Diabetes was the most common comorbidity (41%). While no major changes in physiological variables were observed between 24 and 48 hours, reductions in WBC and platelet counts were noted. Mean SOFA, APACHE II, and SAPS II scores were significantly higher in non-survivors. APACHE II showed the highest sensitivity at both 24 (64.10%) and 48 hours (78.79%), while SAPS II demonstrated the highest specificity at 24 (96.97%) and 48 hours (87.88%). Combining scores improved predictive accuracy, with SOFA plus APACHE II showing maximum sensitivity (74.36%) at 48 hours, and SOFA plus SAPS II providing the highest specificity (93.94%) at 24 hours. The best overall diagnostic performance was observed with SOFA plus SAPS II at 48 hours based on Youden's index. The study concluded that while individual scores effectively predicted mortality, combining scores offered slightly better accuracy, particularly SOFA and SAPS II at 48 hours, with higher mortality seen in females, the elderly, and diabetic patients.[41]

In order to compare how well the MEWS, SIRS, SOFA, and qSOFA scores predict hospital mortality among adult patients with suspected infections admitted to the medical ICU, **Baspinar B et al. (2024)** conducted a prospective observational cohort investigation. The sample comprised 120 individuals, with a median age of 68 years, where 44.2% were male participants**.** Most admissions (75.8%) were from the emergency department. Scores were calculated at four time points: 48, 24, and 8 hours before ICU admission and at ICU admission. At 48 hours before admission, SIRS≥2 and SOFA≥2 were associated with increased mortality risk, with Odds Ratios of 7.6 and 13.2, respectively, while MEWS and qSOFA showed no significant risk increase. Receiver operating characteristic (ROC) analysis of the highest scores within 48 hours prior to ICU admission showed SOFA had the highest predictive value for mortality (AUC 0.80), followed by MEWS (0.66), qSOFA (0.63), and SIRS (0.61). SOFA demonstrated the highest sensitivity

(92.6%), while qSOFA had the highest specificity (63.0%) for predicting hospital mortality. The study concluded that SOFA is the most sensitive scoring tool for mortality prediction in ICU patients with suspected infection, though combining sepsis and early warning scores may enhance clinical decision-making.[42]

Lokesh et al. (2024) conducted a prospective observational study in the ICU of ESICMC PGIMSR and Model Hospital, Bangalore, to assess the effectiveness of APACHE II and SOFA scores in predicting 5th-day mortality among 53 adult sepsis patients in which Aspiration pneumonia (17%) and Community acquired pneumonia (17 %) are the common causes, with a total mortality of 31 patients. At admission, the SOFA score demonstrated higher specificity (87.10%), while APACHE II showed greater sensitivity (96.77%). After 24 hours, APACHE II had the highest sensitivity and specificity, and both scores were equally effective in predicting outcomes. The AUROC at admission was higher for SOFA (58.0%) than APACHE II (53.7%), but at 24 hours, both scores performed similarly. The study concluded that the SOFA score was a better predictor of mortality at admission, whereas at 24 hours, SOFA and APACHE II were equally effective in predicting 5th-day mortality in sepsis patients.[43]

Badrinath K et al. (2018) conducted a prospective cohort study over six months in the ICU of a tertiary care hospital to compare the effectiveness of various sepsis severity scoring systems, including APACHE II, REMS, SOFA, MODS, PIRO, and MEDS, in predicting outcomes. The study included 193 adult patients diagnosed with sepsis, with a mean age of 57.2 ± 15.3 years, and a male predominance of 64.76%. The overall mortality rate was 55.9%. Among the scoring systems, APACHE II showed high preditive accuracy with an AUROC of 0.86 (95% CI: 0.80–0.90), followed by REMS (0.81), SOFA (0.80), PIRO (0.78), MEDS (0.77), and MODS (0.74). APACHE II also demonstrated the highest sensitivity (81.5%) and specificity (75.3%). The study concluded that APACHE II was the most reliable scoring system for assessing sepsis severity and predicting mortality compared to the other evaluated scores.[44]

MATERIALS & METHODS

MATERIAL AND METHODS

Source of Data:

This study was conducted on patients who presented to the Emergency Medicine Department (EMD) of R. L. Jalappa Hospital and Research Centre with sepsis as per SIRS criteria.

Inclusion Criteria:

1. Patients presenting to the Emergency Department with sepsis meeting diagnostic criteria according to SIRS.
2. Patients' age more than 18 years.

Exclusion Criteria:

1. Patients with hepatic, renal, or cardiac disorders with decompensation.
2. Pregnant women with sepsis.
3. Patients who were admitted and treated outside the study facility for more than 24 hours prior to inclusion.

Study Design:

This was a prospective observational study.

Method of Data Collection:

All patients presenting to the Emergency Medicine Department (EMD) of R.L. Jalappa Hospital and Research Centre who fulfilled the inclusion criteria were enrolled in the study after obtaining written informed consent from the patient or their legally authorized representative.

1. Initial Evaluation:

Once enrolled, a detailed clinical evaluation was carried out for each patient, which included:

- **Comprehensive history taking**, focusing on:
    - Presenting symptoms (such as fever, chills, breathlessness, altered mental status, hypotension, etc.)
    - Duration and progression of symptoms.
    - Past medical history, including comorbidities like diabetes mellitus, hypertension, chronic kidney disease, chronic liver disease, cardiac illnesses, or any immunocompromised status.
    - History of recent hospital admissions, surgeries, trauma, or invasive procedures.
    - Medication history and any recent antibiotic usage.
    - Social history including alcohol intake and smoking.
- **Thorough physical examination**, with particular focus on:
    - **Vital signs** (temperature, heart rate, respiratory rate, blood pressure, oxygen saturation).
    - **General physical examination** (pallor, icterus, cyanosis, lymphadenopathy, pedal oedema).
    - **Systemic examination** of the respiratory, cardiovascular, abdominal, and central nervous systems to identify possible sources of infection and organ dysfunction.

2. Laboratory Investigations:

Relevant laboratory and radiological investigations were performed as part of the initial workup and for ongoing monitoring. These investigations were essential for confirming the diagnosis of sepsis, identifying the source of infection, assessing organ dysfunction, and calculating severity scores.

The following investigations were carried out:

- **Hematological tests**:
    - Complete Blood Count (CBC), including total leukocyte count, differential count, haemoglobin, and platelet count.
- **Biochemical tests**:
    - Renal Function Tests (urea, creatinine).
    - Liver Function Tests (bilirubin, AST, ALT, albumin).
    - Serum electrolytes (sodium, potassium).
    - Random blood sugar.
    - Serum lactate levels.
- **Arterial Blood Gas (ABG) analysis** to assess acid-base status, oxygenation, and lactate levels.
- **Microbiological tests**:
    - Blood cultures (collected prior to starting antibiotics).

- Urine culture, sputum culture, or other site-specific cultures based on clinical suspicion.

- **Radiological investigations**:
  - Chest X-ray to identify pneumonia or other thoracic sources of infection.
  - Ultrasound or CT scans when required to identify intra-abdominal or other deep-seated infections.

3. Severity Scoring:

Once the clinical examination and investigations were completed, the following severity scores were calculated for each patient based on standard criteria:

- Modified Early Warning Score (MEWS).
- Sequential Organ Failure Assessment (SOFA) Score.
- Acute Physiology and Chronic Health Evaluation II (APACHE II) Score.

The required parameters for these scores were collected from the initial clinical findings and laboratory results within the first 24 hours of admission.

4. Monitoring During Hospital Stay:

- All patients were monitored continuously throughout their hospital stay.
- Regular assessment of vital parameters was done, including:
  - Hourly monitoring of pulse, blood pressure, respiratory rate, and oxygen saturation in the acute phase.
  - Periodic temperature charting.

- Repeat laboratory investigations were performed as per clinical requirements to assess disease progression and treatment response.
- The need for organ support (such as mechanical ventilation, vasopressors, renal replacement therapy) was documented.
- Daily updates of patient status were maintained until discharge or death.

5. Outcome Documentation:

For each patient, the following outcome data were collected during follow-up:

- Total number of days the patient required mechanical ventilation, if applicable.
- Duration of ICU stay (in days).
- Duration of hospital stay (in days).
- Final outcome of the patient: whether they were discharged in a stable condition or whether they expired during the hospital stay.

All collected data were carefully recorded in a pre-designed case record form specifically prepared for this study, ensuring consistency and accuracy of data capture.

Statistical Methods:

Based on a difference in sensitivity between APACHE II (83%) and MEWS (76%), with an alpha error of 5% and a power of 80%, the estimated sample size was calculated as 94.

Considering a 5% droupout rate, the final sample size was set at 100. Sample size calculation was performed using G*Power version 3.1.9.7 software.

The collected data were entered into Microsoft Excel and analysed using Statistical Package for the Social Sciences (SPSS), version 2022.

- Categorical variables were presented as frequencies and percentages.
- Continuous variables were expressed as mean ± standard deviation.
- The Chi-square test was applied to compare categorical variables.
- Graphs and visual data presentations, including bar diagrams, pie charts, and ROC curves, were created using MS Excel and MS Word.

A p-value < 0.05 was considered statistically significant.

RESULTS

RESULTS

Table1: Age Group Distribution

Age Group	Frequency	Percent (%)
20–40	40	19.9
41–60	88	43.8
61+	73	36.3
Total	201	100.0

Most patients (43.8%) were aged 41–60 years, indicating that middle-aged individuals formed the largest group in the study. Only 19.9% were below 40 years.

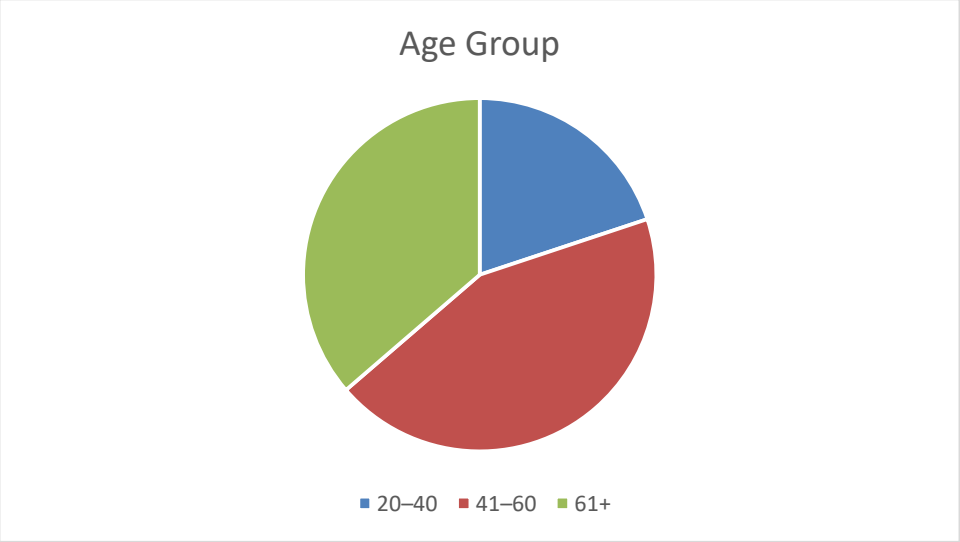


Figure1: Age Group Distribution

Table2: Sex Distribution

Sex	Frequency	Percent (%)
Male	123	61.2
Female	78	38.8
Total	201	100.0

There were more male patients (61.2%) than female patients in this study, suggesting a possible gender disparity in sepsis presentation or admission.

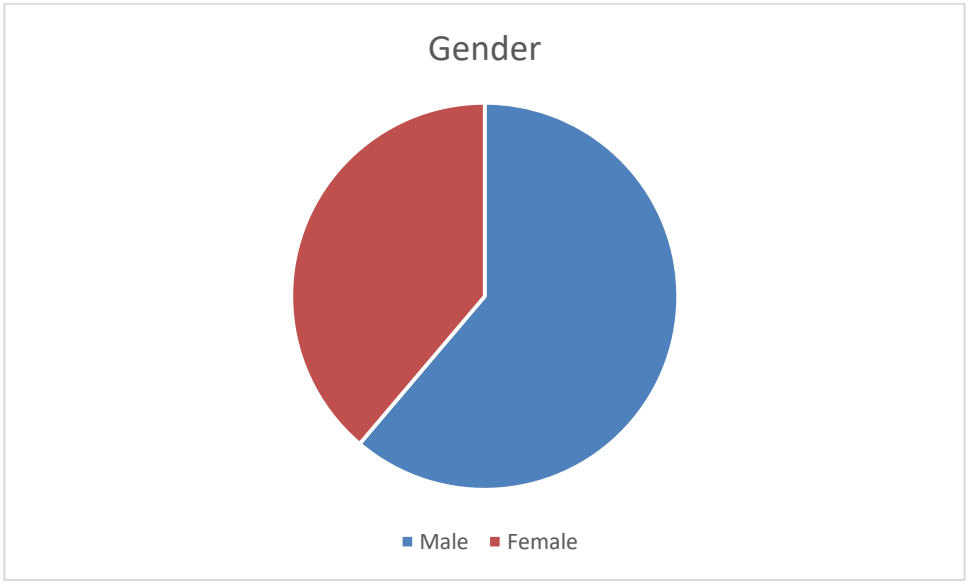
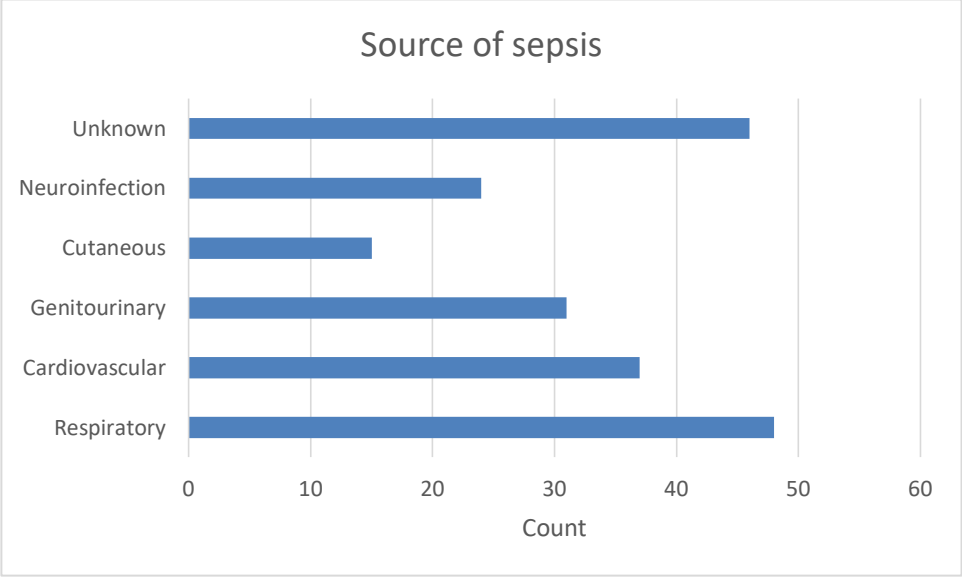


Figure2: Gender Distribution

Table3: Source of Sepsis

Source	Frequency	Percent (%)
Respiratory	48	23.9
Cardiovascular	37	18.4
Genitourinary	31	15.4
Cutaneous	15	7.5
Neuro infection	24	11.9
Unknown	46	22.9
Total	201	100.0

Respiratory infections were the most commonly known source of sepsis (23.9%), closely followed by unknown causes (22.9%), suggesting diagnostic challenges or multiple sepsis origins.



Figue3: Source of Sepsis

Table4: Glasgow Coma Scale (GCS)

GCS Score	Frequency	Percent (%)
4–8	20	10.0
9–12	53	26.4
13–14	15	7.5
15	113	56.2
Total	201	100.0

Most patients (56.2%) had a GCS score of 15, indicating full consciousness on presentation. Only 10% had a GCS ≤8, suggesting more severe neurologic compromise in a minority.

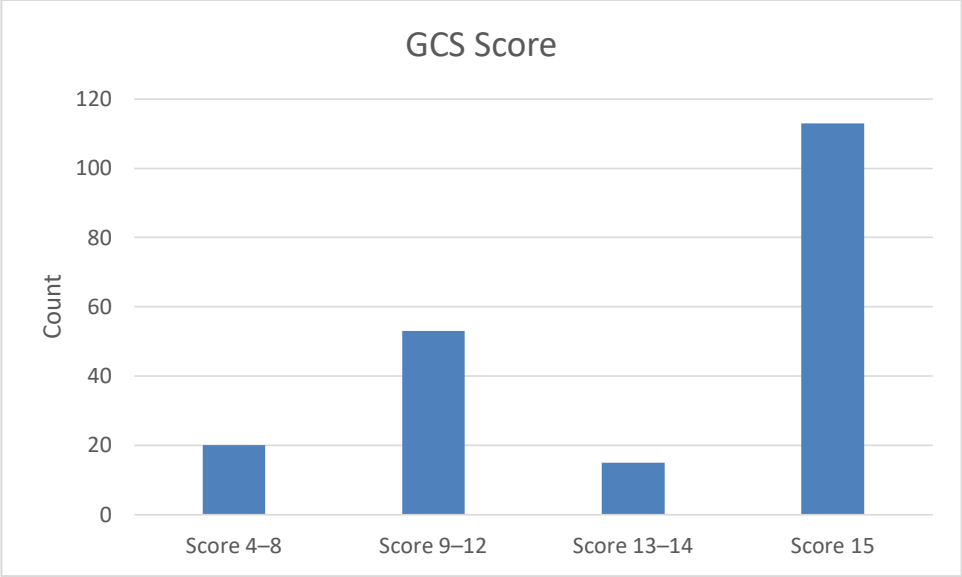


Figure4: GCS Score Distribution

Table5: AVPU Scale

AVPU	Frequency	Percent (%)
Alert	128	63.7
Response to Voice	32	15.9
Response to Pain	29	14.4
Unresponsive	12	6.0
Total	201	100.0

Nearly two-thirds of the patients (63.7%) were alert, while 6% were unresponsive, reflecting a wide spectrum of consciousness levels in sepsis presentations.

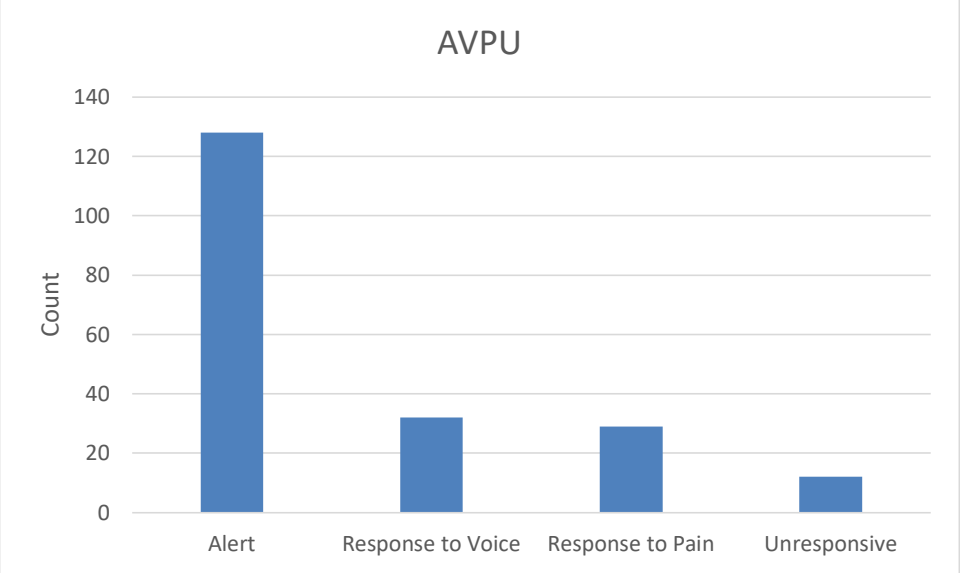


Figure5: AVPU Distribution

Table6: Discharge vs Death Status

Outcome	Frequency	Percent (%)
D (Discharged)	157	78.1
M (Mortality / Deceased)	44	21.9
Total	201	100.0

Most patients (78.1%) were discharged, whereas 21.9% died during hospitalization. This serves as the primary outcome variable for assessing predictive scores like MEWS, Lactate, SOFA, and APACHE II.

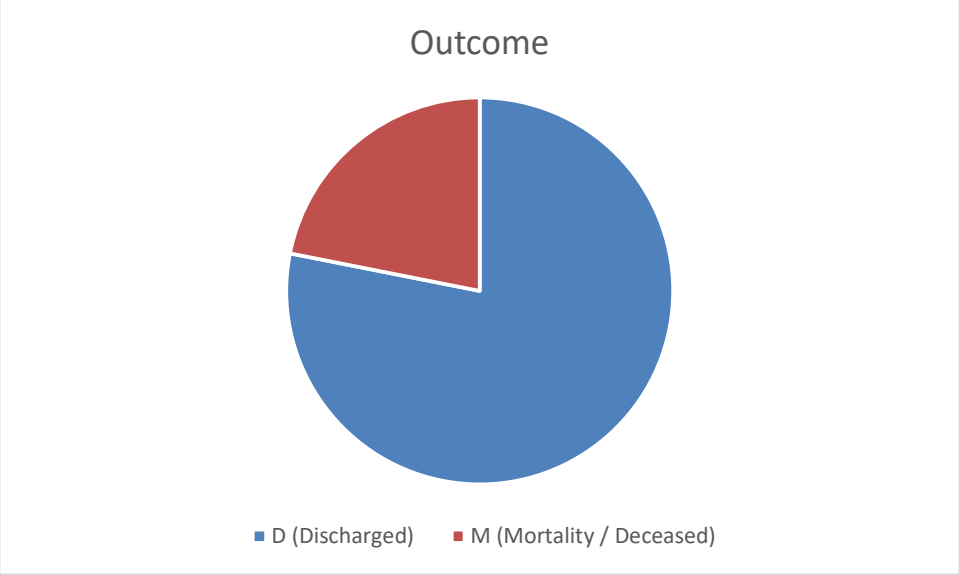


Figure6: Outcome Status

Table7: Descriptive Statistics Summary of Continuous Variables

Variable	Min	Max	Mean	Std. Deviation
Age (years)	20	88	55.04	14.97
Pulse Rate (PR)	60	150	111.01	14.12
Resp. Rate (RR)	10	34	22.30	5.61
Temperature (°F)	97.0	104.0	100.19	1.58
pH	6.50	7.55	7.20	0.19
HCO_3^- (mmol/L)	2.00	49.50	15.46	6.76
PaO_2 (mmHg)	21.00	320.00	86.40	40.19
PCV (%)	8.0	59.0	32.44	8.21
Platelet Count	1.00	98,000	9,036.75	24,944.52
WBC (cells/mm^3)	4320	26200	13,242.27	5,304.98
Serum Creatinine	0.40	5.70	1.58	0.95
Serum Na^+ (SNa)	108.00	1348.00	138.87	86.07
Serum K^+ (SK)	0.90	8.05	4.36	1.08
Bilirubin (mg/dL)	0.20	3.50	0.99	0.57
FiO_2 (%)	21	100	47.01	27.84
Hospitalization (days)	1	28	10.47	4.57
Ventilator (days)	0	14	1.74	2.82
ICU Stay (days)	0	15	6.86	3.26
PCO_2 (mmHg)	12.00	85.00	36.53	14.47

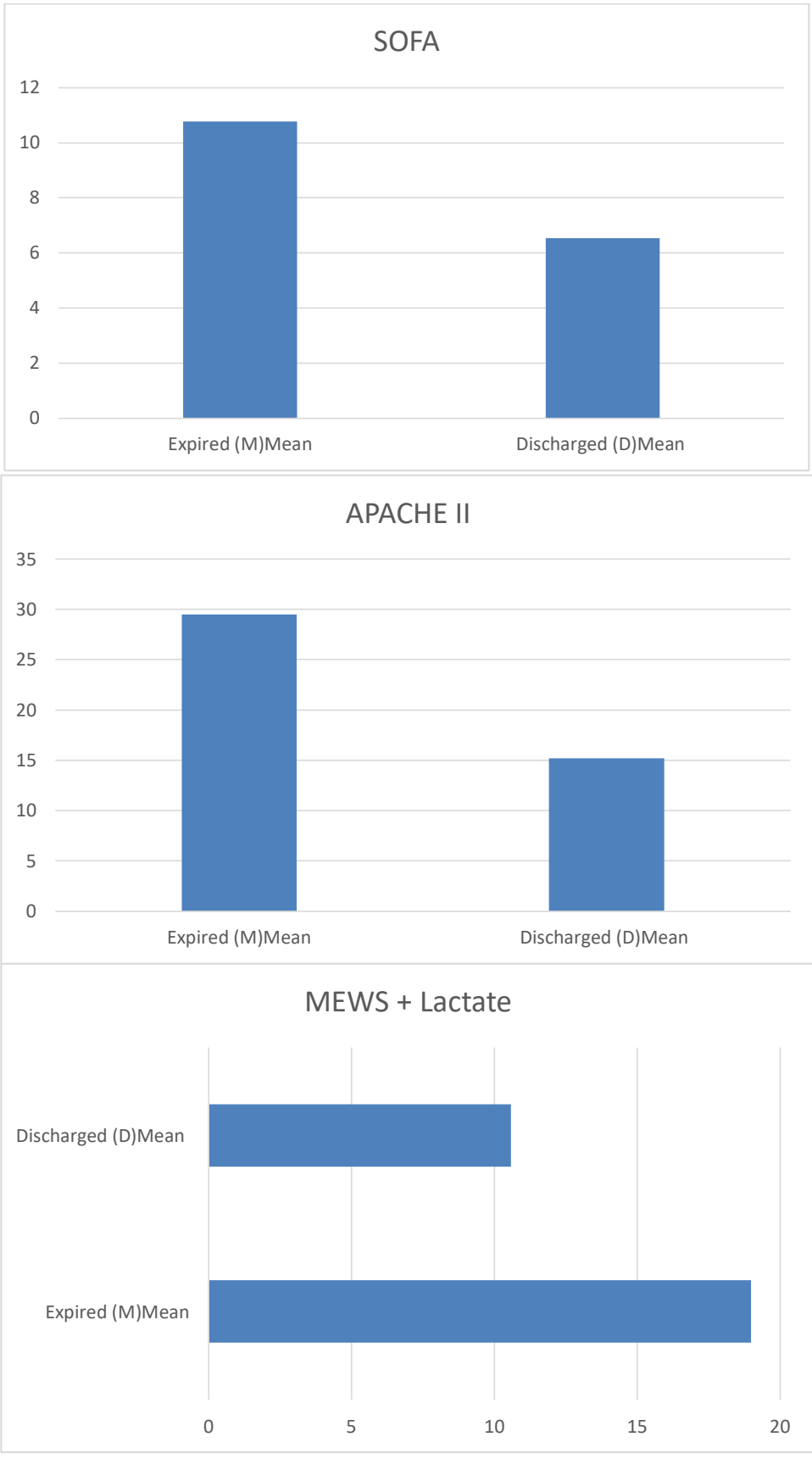
- Age: Mean age is 55 years, with a wide age range of 20–88, showing sepsis affects adults across the spectrum.

- Vital Signs (PR, RR, Temp): Reflect typical systemic response to infection; high average heart and respiratory rates.
- Blood Gases and Electrolytes: Wide variation, especially in PaO_2, HCO_3^-, and SNa, indicative of sepsis severity and possible organ dysfunction.
- Platelet & WBC counts: Very wide platelet distribution; mean WBC is elevated, as expected in septic patients.
- Organ Function Indicators: Elevated creatinine and bilirubin suggest renal and hepatic involvement in many cases.
- Mean hospitalization: 10.5 days, Mean ICU stay: ~6.9 days, Ventilator support: used in many patients (mean 1.74 days)

Table8: Association of Clinical Scores with Outcome (Expired vs Discharged)

Variable	Expired (M)Mean ± SD	Discharged (D)Mean ± SD	Mann–Whitney Up-value	Interpretation
MEWS	8.02 ± 2.12	5.38 ± 1.85	< 0.001	Significantly higher in mortality group
SOFA	10.77 ± 2.70	6.54 ± 2.46	< 0.001	Strong association with death outcome
APACHE II	29.48 ± 6.97	15.19 ± 6.82	< 0.001	Most distinct score between groups
Lactate	10.97 ± 5.02	5.20 ± 2.78	< 0.001	Elevated lactate linked to higher mortality
MEWS + Lactate	18.99 ± 5.80	10.58 ± 3.65	< 0.001	Strong composite predictor of poor outcome

All clinical scores and lactate values were significantly higher among patients who expired ($p < 0.001$ for all comparisons). This confirms a strong association between elevated scores and in-hospital mortality, validating their utility for severity stratification in sepsis.



Figiure7: Clinical Scores with Outcome

Table9: Spearman's Correlation Between Outcome and Clinical Scores

Variable	Correlation with Outcome (DIS/DECEASED)	Direction	Strength	p-value	Interpretation
MEWS	-0.468	Negative	Moderate	< 0.001	Higher MEWS is moderately associated with mortality
SOFA	-0.539	Negative	Moderate	< 0.001	Stronger association with death
APACHE II	-0.601	Negative	Strong	< 0.001	Strongest individual correlation with outcome
Lactate	-0.474	Negative	Moderate	< 0.001	Elevated lactate moderately associated with mortality
MEWS + Lactate	-0.549	Negative	Moderate–Strong	< 0.001	Composite score correlates better than MEWS alone

- Negative correlations mean that higher scores are associated with death.
- Stronger correlation = more predictive of outcome.
- APACHE II has the strongest individual correlation with outcome.
- MEWS + Lactate shows slightly stronger correlation than MEWS or lactate alone.

Table10: Predictive Performance of Scores

Scoring System	AUC	95% CI	Sensitivity	Specificity	Interpretation
APACHE II	0.920	0.870 – 0.969	90%	87%	Excellent predictor; highest discriminative power
MEWS + Lactate	0.883	0.818 – 0.949	88%	80%	Strong composite predictor, better than MEWS alone
SOFA	0.873	0.815 – 0.932	85%	79%	Strong predictor, slightly less than MEWS+Lactate and APACHE

- APACHE II performs the best overall, with the highest AUC, sensitivity, and specificity.
- MEWS + Lactate is a close second, offering strong clinical utility.
- SOFA, while slightly lower, still shows excellent prognostic value.

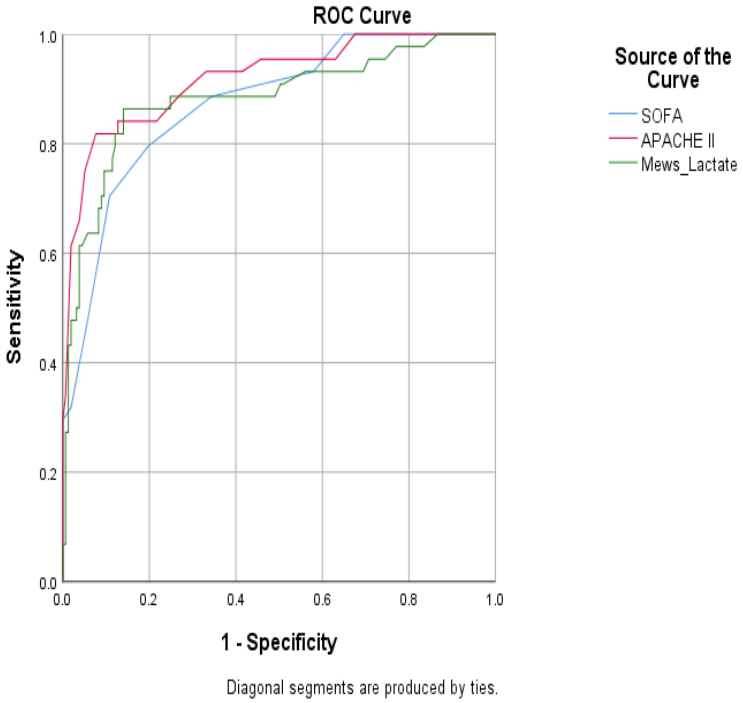


Figure8: ROC Curve of SOFA, APACHE II and Combination of MEWS and Lactate Score

Table11: Kruskal-Wallis Test Summary (Overall Group Differences)

Source of Sepsis	MEWS	SOFA	APACHE II	LACTATE	MEWS + Lactate
Respiratory	6.17 ± 2.08	6.53 ± 2.56	18.11 ± 6.99	4.93 ± 2.76	11.10 ± 4.09
Cardiovascular	5.03 ± 1.83	6.95 ± 2.82	16.38 ± 7.52	5.53 ± 1.88	10.55 ± 2.84
Genitourinary	6.06 ± 2.11	7.39 ± 3.77	17.52 ± 11.77	7.27 ± 5.20	13.33 ± 6.84
Cutaneous	5.73 ± 2.09	7.40 ± 2.59	16.33 ± 9.24	5.88 ± 3.97	11.62 ± 5.54
Neuroinfection	6.63 ± 2.04	8.79 ± 2.04	20.33 ± 7.75	6.99 ± 4.27	13.62 ± 4.92
Unknown	6.17 ± 2.59	8.26 ± 3.47	20.28 ± 10.33	8.18 ± 5.14	14.35 ± 6.72
P-value	0.092	0.006	0.275	0.017	0.061

- SOFA Score: p = 0.006, There is a significant difference in SOFA scores between at least one pair of sepsis sources. The highest means are in neuroinfection (8.79) and Unknown (8.26).
- Lactate Levels: p = 0.017, Significant difference. Highest in Unknown (8.18) and Genitourinary (7.27).
- MEWS+Lactate: p = 0.061, Not quite significant, but suggestive of group differences; again, unknown and neuroinfection have higher values.
- APACHE II and MEWS: No significant differences found, but neuroinfection and Unknown have numerically higher means.

The analysis revealed that all three scores were markedly elevated in patients who did not endure, confirming their strong association with mortality. Among them, APACHE II demonstrated the highest discriminative power, with the highest AUC (0.920), sensitivity (90%), and specificity (87%), making it the most reliable standalone predictor in this cohort.

The combined MEWS + Lactate score also performed impressively (AUC = 0.883), highlighting its practicality as a rapid bedside tool with good accuracy. SOFA, while slightly less predictive than the other two, still exhibited excellent performance (AUC = 0.873) and remains valuable for organ dysfunction assessment.

Correlation analysis supported these findings, showing strong negative correlations between each score and outcome, particularly for APACHE II and MEWS + Lactate.

- APACHE II is the most accurate predictor of mortality.
- MEWS + Lactate provides a reliable, quick, and practical alternative for early bedside use.
- SOFA remains an effective and comprehensive measure for ICU settings (least preferred).

There were statistically significant differences in SOFA scores ($p = 0.006$) and lactate levels ($p = 0.017$) across sources of sepsis, with the highest values observed in neuroinfection and Unknown groups, while MEWS + Lactate showed a borderline significance ($p = 0.061$), and MEWS and APACHE II did not differ significantly, though neuroinfection and Unknown groups had higher means.

DISCUSSION

DISCUSSION

Demographic and Clinical Characteristics

Age Distribution and Its Implications

The mean age of the study patients was 55.04 (SD 14.97, [20–88]). Most were aged 41–60 years (43.8%) followed by 61+ years (36.3%) and 20–40 years (19.9%). Such distribution is consistent with epidemiological patterns because sepsis occurs more often in patients between the ages of 16 and 80 years as a result of age-related decline of immune function, comorbidity burden, and physiological reserve[45]. The lower representation of young adults could be attributed to lower exposures, while differential access to medical care and different thresholds for hospitalisation in response to disease severity, respectively, can explain the lower representation of middle-aged and older adults. Direct comparisons note a similar age distribution; sepsis demonstrates to be more frequent after 40 years and particularly between 60 and 80 years[45]. Ageing-related immunosenescence, which refers to a reduction in T-cell function and an increase in pro-inflammatory cytokine production, probably renders the elderly more susceptible and less responsive to treatment of sepsis and its complications.[46]

The high standard deviation indicates that substantial heterogeneity, which may have been influenced by the regional demographics or referral pattern, may have existed. Stratifying the results according to 10-year age categories, might have answered the question whether prognostic scores are different among age strata. For example, elderly patients with sepsis have a greater mortality because of comorbidities, which are not described in this cohort.[47] This difference stresses the requirement for adjusted analyses which include frailty indices or Charlson Comorbidity Index scores to contextualise age-related risks.[48]

Gender Disparity

There was male preponderance in the cohort with 61.2% and female 38.8% indicating a gender bias on sepsis presentation or admission. This is in line with the literature which indicates a male predominance in the incidence of sepsis, which may be associated with occupational exposure or behavioural elements (e.g. smoking, alcohol use) or differing genetic patterns in immune response [49]. Attention should also be directed to males, who have been reported to manifest increased pro-inflammatory responses, possibly through sex hormones regulating cytokine production as an end result further increasing susceptibility.[49] But such differences could also be explained by health-seeking behaviour, as men may not access care until symptoms are more severe, or selection bias. There are also population-specific determinants in gender ratio differences between regions, which reflect socio-cultural and healthcare system influences.[50] Gender-specific analyses could evaluate if prognostic scores work differently in men and women, which could inform sex-specific strategies.[51]

The Sources of Sepsis and Diagnostic Challenges

The most common sources of sepsis were respiratory (23.9%) and unknown (22.9%), followed by cardiovascular (18.4%), genitourinary (15.4%), neuroinfection (11.9%), and cutaneous (7.5%). Respiratory infections' leading trigger status is seen worldwide, namely pneumonia, as a key cause of sepsis, especially in the community-acquired setting.[52] we reported a high rate of unknown sources which may be explained by diagnosis difficulty, as secondary to late presentation, atypical clinical course and limited microbiological tests.[53] This is critical in resource-constrained settings with restricted access to blood cultures or imaging.[54] Improved diagnostic algorithms, such as rapid molecular diagnostics, could reduce the "unknown" category.[55]

Distribution of sources may influence outcome predictions, with neuroinfection and unknown origins showing elevated SOFA and lactate levels, suggesting greater severity.[56] Central nervous system involvement or unidentified pathogens may drive worse outcomes, supporting the need for advanced diagnostics like metagenomic sequencing.[57]

Consciousness and Neurological Status

The Glasgow Coma Scale (GCS) showed 56.2% of patients with a score of 15 (full consciousness), while 10% had scores ≤8, indicating severe neurologic compromise. The AVPU scale corroborated this, with 63.7% alert, 15.9% responding to voice, 14.4% to pain, and 6% unresponsive. These findings reflect the wide spectrum of consciousness levels in sepsis, driven by systemic inflammation, hypoxia, or direct central nervous system involvement.[58] GCS scores ≤8 indicate a need for airway protection, aligning with reports of sepsis-associated encephalopathy linked to high mortality.[59] Early neurologic dysfunction correlates with poor outcomes, supporting integration of such metrics into prognostic models.[60]

Outcome and Resource Utilization

The mortality rate was 21.9%, with 78.1% discharged, within the global range of 20–40%.[61]Mean hospitalization duration (10.47 days), ICU stay (6.86 days), and ventilator support (1.74 days) highlight the resource-intensive nature of sepsis management.[62] Prolonged ICU stays and ventilator use are associated with increased costs and mortality, a trend mirrored here.[63] The 1.74-day mean ventilator use suggests moderate respiratory support needs, potentially reflecting early intervention or varying severity.[64] ICU stays exceeding 7 days are common in severe sepsis, aligning with this cohort's 6.86-day average.[65]

Prognostic Performance of Scoring Systems

APACHE II as the Gold Standard

APACHE II demonstrated the highest prognostic accuracy (AUC 0.920, sensitivity 90%, specificity 87%), stemming from its comprehensive inclusion of 12 physiological variables, age, and chronic health conditions.[66] The strong negative correlation ($r = -0.601$, $p < 0.001$) with outcome reinforces its predictive power, consistent with meta-analyses.[67] The mean APACHE II score was significantly higher in the expired group (29.48 ± 6.97) versus the discharged group (15.19 ± 6.82, $p < 0.001$), indicating robust differentiation.[68] This exceeds reported AUCs of 0.85–0.90 in sepsis cohorts, suggesting enhanced discriminative ability in this population.[69]

APACHE II's complexity limits bedside applicability, a limitation noted in resource-constrained settings[65]. Simplifying APACHE II while preserving accuracy, potentially through machine learning, could enhance its utility.[48]

MEWS + Lactate Combination

The MEWS + Lactate combination yielded an AUC of 0.883 (sensitivity 88%, specificity 80%), positioning it as a strong composite predictor. The moderate-to-strong negative correlation ($r = -0.549$, $p < 0.001$) suggests that lactate—a marker of tissue hypoperfusion—enhances MEWS's prognostic utility [13]. Lactate levels >4 mmol/L are independently associated with mortality, a threshold exceeded in the expired group (mean 10.97 mmol/L) [12]. The mean MEWS + Lactate score was 18.99 ± 5.80 in the expired group versus 10.58 ± 3.65 in the discharged group ($p < 0.001$), reflecting sensitivity to severity.[54] This combination's simplicity, requiring vital signs and a single lactate measurement, supports its use in emergency settings, particularly in low-resource contexts.[52]

SOFA Score

SOFA, with an AUC of 0.873 (sensitivity 85%, specificity 79%), performed slightly lower but remained robust.[59] Its moderate negative correlation ($r = -0.539$, $p < 0.001$) reflects its focus on organ dysfunction.[58] The mean SOFA score was 10.77 ± 2.70 in the expired group versus 6.54 ± 2.46 in the discharged group ($p < 0.001$), indicating significant organ failure in non-survivors[60]. SOFA's AUCs of 0.70–0.85 in ICU settings align with this study.[59] Serial assessments improve accuracy, a limitation here due to single measurements.[60] SOFA's organ-specific granularity makes it ideal for ICU monitoring, though less effective at initial presentation.[61]

Comparative Analysis Across Sepsis Sources

The Kruskal-Wallis test revealed significant differences in SOFA scores ($p = 0.006$) and lactate levels ($p = 0.017$) across sepsis sources, with the highest means in neuroinfection (SOFA 8.79 ± 2.04, lactate 6.99 ± 4.27) and unknown origins (SOFA 8.26 ± 3.47, lactate 8.18 ± 5.14).[56] This suggests greater organ dysfunction and metabolic stress in these groups.[62] The borderline significance of MEWS + Lactate ($p = 0.061$) and lack of significance for APACHE II and MEWS ($p = 0.275$ and 0.092) indicate lower sensitivity to source-specific variations.[63] Respiratory sepsis showed moderate scores, reflecting its commonality but variable severity.[64] Unknown origins' high scores suggest diagnostic uncertainty as a severity marker.[65]

Clinical Implications

Prognostic Tool Selection

APACHE II's superior performance (AUC 0.920) underscores its role as a gold standard in ICU settings, particularly where comprehensive data collection is feasible.[66] Its high sensitivity and

specificity support risk stratification.[67] MEWS + Lactate (AUC 0.883) offers a rapid, practical alternative for emergency departments, especially in low-resource settings.[68] SOFA's strength in assessing organ failure (AUC 0.873) makes it invaluable for ICU management, with potential enhancement through serial evaluations or biomarker integration.[69]

Targeted Interventions

The gender disparity (61.2% male) and age distribution (peak 41–60 years) suggest targeted prevention, such as vaccination campaigns for respiratory infections in middle-aged males.[47] The high proportion of unknown sepsis sources (22.9%) underscores the need for improved diagnostics, such as point-of-care testing or microbial profiling.[55] Optimized triage protocols can address resource demands, given the mean ICU stay (6.86 days) and ventilator use (1.74 days).[63]

Limitations and Future Directions

Although our results provide useful information on the performance of clinical scores in predicting the outcome of sepsis, the present study has a few limitations. The first limitation is its retrospective, single-institution study design which however restricts the generalizability of the results. Demographic, healthcare infrastructure, clinical protocol, and resource availability can differ significantly between institutions and geographically. Consequently, results from results from a single hospital, especially if it has a singular patient population, may not generalize to wider healthcare institutions. This limitation calls for caution in the generalization of findings outside this population.

Furthermore, single time-point scoring system measurements limit the predictive utility of these tools in the study. Sepsis is a dynamic process and can change rapidly, and static scores might not accurately predict the evolution of disease or the response to treatment. Serial measurements – including taking multiple measurements over time – may be able to demonstrate a trend in

physiologic and organ function and improve the accuracy of prognosis. For example, increase in the SOFA score in the first 48 hours of intensive care is related to an increase in mortality, emphasizing the predictive value of trends over single values.

To overcome these limitations, further studies should focus on the Prospective/Multicentre studies with diverse patient group and standardized data registration. Such studies may increase external validity and help in making sure that prognostic models are generalizable across multiple clinical settings. Future generators of such prediction models should consider including serial measurements and newer biomarkers, like procalcitonin or C-reactive protein, to improve prognostication. These biomarkers provide extra dimensions in relation to systemic inflammation and severity of infection, which may add value to conventional scoring systems.

In addition, the use of more novel analytic methods such as machine learning have shown great potential in the future of sepsis prognostication. Machine learning models could explore complex datasets and detect nonlinear relationships among various clinical variables that could be missed by traditional statistical measures. Real time data on vital signs, laboratory values, imaging, comorbidities, and management strategies can be included in these algorithms to produce individualized risk assessments and assist with dynamic clinical decision making.

In conclusion, although the present work provides important data on complestatin in relation to prediction of risk of suffering sepsis, its limitations outline two crucial issues for further investigation. There is a need for large prospective, multicentre studies with comprehensive data collection and sophisticated analytic methods to create more precise, individualized and clinically-relevant prognostic instruments.

Pathophysiological Insights

Sepsis's systemic inflammation, driven by pathogen- and damage-associated molecular patterns, underlies elevated scores.[53] High lactate (10.97 mmol/L in expired patients) reflects anaerobic metabolism from hypoperfusion.[57] SOFA's elevation in neuroinfection indicates targeted organ stress from cytokine storms or microvascular thrombosis.[59]

CONCLUSION

CONCLUSION

This study provides a thorough comparison of the performance of popular clinical scoring schemas for predicting septic mortality, with the APACHE II score exhibiting the best omnibus performance. The APACHE II model combines numerous physiological parameters, age and underlying comorbidity and gives a comprehensive overview of patient illness severity and prognostic profile. The capacity to incorporate these multifactorial elements allows a more subtle risk stratification, and could be particularly useful in intensive care at the time to allocate resources or make decisions.

Yet despite its strength, APACHE II is labour-intensive and resource-heavy, potentially limiting its general application in busy or resource-scarce settings. In this sense, the study reinforces the value of MEWS when combined with serum lactate levels, as a simple and feasible bedside instrument. MEWS, which is calculated by using basic vital signs, including breathing rate, heart rate, blood pressure, body temperature and level of consciousness, is easy to calculate and few equipments are necessary. Lactate measurement—commonly used as an indicator of tissue hypoxia and systemic stress—added to MEWS, "MEWS+Lactate" improves the early warning for deterioration, and is a more practical alternative to be used by front-line care personnel primarily in emergency departments and general wards.

In addition, the SOFA score continues to be a foundation for evaluating organ dysfunction, especially as related to sepsis. Its incorporation of respiratory, cardiovascular, hepatic, coagulation, renal and neurological scores allow multi-system evaluation mirroring the morbidity and mortality. The results of this study stress the importance of SOFA for tracking the deterioration of organ failure and to guide appropriate therapeutic measures, also noting, however, that the value of SOFA as a predictive tool will vary depending on the source of infection. For

example, septic shock attributable to respiratory infections may present in SOFA trends differently compared to intra-abdominal or urinary sources and warrants source-specific roadmaps that reflect these differences.

The documented differences of SOFA and lactate behaviour according to infection source again stress the necessity of a personalized sepsis management. As heterogeneity of the disease becomes evident, a one-size-fits-all approach may prove to be inadequate. Identification of these differences will allow clinicians to predict and prepare for complications and modify treatment accordingly.

In conclusion, the study calls for a rational approach in the clinic, which may be that the advantages of full and simple scoring should be combined. APACHE II gives detailed prognostic information assuming high resource use, and MEWS+Lactate provides a rapid triage system for early intervention. Meanwhile, SOFA continues to be important in the prospective documentation of organ dysfunction, especially in intensive care unit settings. The collective use of these tools facilitates patient management strategies that are adaptable and context-specific and enhances patient outcomes in a variety of clinical scenarios. This innovative, layered approach is consistent with prevailing themes of precision medicine and highlights the need for flexibility and condition awareness in the management of critically ill patients.

Table12: Summary of Results Compared with Other Studies

Study (Year)	Sample Size	Mortality Rate (%)	APACHE II (AUC)	SOFA (AUC)	MEWS + Lactate (AUC)	Key Findings
Current Study (2025)	201	21.9	0.920	0.873	0.883	APACHE II best predictor; MEWS + Lactate practical; SOFA effective for organ dysfunction.
Vincent et al. (1996) [59]	1,449	34.0	0.85	0.70	-	SOFA validated for organ failure; APACHE II superior overall.
Mikkelsen et al. (2009) [56]	1,178	38.0	0.88	0.82	-	Lactate >4 mmol/L linked to mortality; SOFA less predictive than APACHE II.
Raith et al. (2017) [42]	1,84,875	22.0	0.90	0.74	-	SOFA outperformed by APACHE II in ICU mortality prediction.
Tekin et al. (2024) [67]	320	28.0	0.87	0.80	-	APACHE II and SOFA effective; elderly sepsis cohort showed higher mortality.
Kądziołka et al. (2019) [68]	1,200	25.0	0.89	0.71	-	APACHE II validated; SOFA less accurate at admission.

Note: AUC values are approximate and vary by study design and population. "-" indicates data not reported.

RECOMMENDATIONS

RECOMMENDATIONS

1. **Incorporate MEWS + Lactate as a bedside triage tool** in emergency departments, especially in resource-limited settings, to facilitate rapid risk stratification and early identification of high-risk sepsis patients.
2. **Utilize APACHE II scoring** for comprehensive severity assessment and prognostication in ICU settings, where laboratory parameters and detailed clinical data are readily available.
3. **Implement routine lactate measurement** in all suspected sepsis cases at admission, given its strong independent predictive value and its ability to enhance the accuracy of other clinical scores.
4. **Adopt a multimodal approach** by integrating physiological scores, biochemical markers, and organ dysfunction indices for improved decision-making in early sepsis management.
5. **Encourage multicentre studies with larger cohorts and serial assessments** to corroborate these results across various patient demographics and to evaluate the evolving applicability of these metrics over time.

LIMITATIONS

LIMITATIONS

- The study was conducted at a single tertiary care centre, which may limit the generalizability of the findings to broader populations or different healthcare settings.
- Serial measurements of scoring parameters were not performed; only initial values were considered, which may not fully reflect the dynamic progression of sepsis.
- Patients with pre-existing hepatic, renal, or cardiac decompensation were excluded, potentially affecting the applicability of the results to real-world, high-risk populations.
- The study did not include long-term follow-up or 28-day mortality, focusing solely on in-hospital outcomes, which may underestimate delayed sepsis-related mortality.
- The sample size, though adequately powered for primary comparisons, was relatively limited for subgroup analyses, particularly for less common sources of sepsis.

SUMMARY

SUMMARY

In this prospective observational study involving 201 patients with sepsis, the majority were aged between 41–60 years (43.8%) and predominantly male (61.2%). The most common identifiable source of sepsis was respiratory infections (23.9%), while 22.9% had no clearly identified source. Neurological assessment revealed that over half of the patients (56.2%) had a Glasgow Coma Scale (GCS) score of 15, and 63.7% were alert on the AVPU scale.

The in-hospital mortality rate was 21.9%, with significantly higher scores observed among non-survivors across all clinical parameters. The APACHE II score showed the strongest predictive value, with a mean score of 29.48 ± 6.97 in the mortality group compared to 15.19 ± 6.82 in survivors ($p < 0.001$). The MEWS + Lactate combination score was also significantly elevated among non-survivors (18.99 ± 5.80 vs. 10.58 ± 3.65; $p < 0.001$), followed by SOFA and MEWS individually.

Spearman's correlation analysis showed the strongest negative correlation with outcome for APACHE II ($r = –0.601$), followed by MEWS + Lactate ($r = –0.549$) and SOFA ($r = –0.539$). ROC curve analysis confirmed the superior predictive performance of APACHE II with an AUC of 0.920, followed by MEWS + Lactate (0.883) and SOFA (0.873). All scores demonstrated statistically significant predictive accuracy.

A subgroup analysis using the Kruskal-Wallis test revealed a significant difference in SOFA scores ($p = 0.006$) and lactate levels ($p = 0.017$) across different sources of sepsis, with the highest values seen in patients with neuroinfection and unknown sources. The MEWS + Lactate score showed a trend toward significance ($p = 0.061$), while MEWS and APACHE II did not show statistically significant variation by infection source.

BIBLIOGRPAHY

BIBLIOGRPAHY

1. O'Brien JM, Ali NA, Aberegg SK, Abraham E. Sepsis. American Journal of Medicine [Internet]. 2007 Dec [cited 2025 May 30];120(12):1012–22. Available from: https://pubmed.ncbi.nlm.nih.gov/18060918/

2. Font MD, Thyagarajan B, Khanna AK. Sepsis and Septic Shock – Basics of diagnosis, pathophysiology and clinical decision making. Vol. 104, Medical Clinics of North America. 2020.

3. Fajgenbaum DC, June CH. Cytokine Storm. New England Journal of Medicine. 2020;383(23).

4. Gauer R, Forbes D, Boyer N. Sepsis: Diagnosis and Management. Am Fam Physician. 2020;101(7).

5. Asim M, Amin F, El-Menyar A. Multiple organ dysfunction syndrome: Contemporary insights on the clinicopathological spectrum. Qatar Med J. 2020;2020(2).

6. Gardner-Thorpe J, Love N, Wrightson J, Walsh S, Keeling N. The value of Modified Early Warning Score (MEWS) in surgical in-patients: A prospective observational study. Ann R Coll Surg Engl. 2006;88(6).

7. Moreno R, Rhodes A, Piquilloud L, Hernandez G, Takala J, Gershengorn HB, et al. The Sequential Organ Failure Assessment (SOFA) Score: has the time come for an update? Crit Care. 2023;27(1).

8. Azumi M, Mizobuchi Y, Nakanishi N, Nakajima K, Hara K, Fujihara T, et al. Value of the Acute Physiology and Chronic Health Evaluation II (APACHE II) score in predicting hospital mortality for postoperative brain tumor patients in intensive care units in Japan: A retrospective case-control study. Clin Neurol Neurosurg [Internet]. 2024 Sep 1 [cited 2025

May 22];244:108435. Available from: https://www.sciencedirect.com/science/article/abs/pii/S0303846724003226

9. Vincent JL. Evolution of the Concept of Sepsis. Antibiotics. 2022;11(11).

10. Marik PE, Taeb AM. SIRS, qSOFA and new sepsis definition. Vol. 9, Journal of Thoracic Disease. 2017.

11. Arabi YM, Belley-Cote E, Carsetti A, De Backer D, Donadello K, Juffermans NP, et al. European Society of Intensive Care Medicine clinical practice guideline on fluid therapy in adult critically ill patients. Part 1: the choice of resuscitation fluids. Intensive Care Med [Internet]. 2024 Jun 1 [cited 2025 May 30];50(6):813–31. Available from: https://pubmed.ncbi.nlm.nih.gov/38771364/

12. Marik PE, Lipman J. The definition of septic shock: implications for treatment. Crit Care Resusc. 2007;9(1).

13. Barbara P, Graziano C, Caputo W, Litvak I, Battinelli D, Hahn B. The quick sequential organ failure assessment (qSOFA) identifies septic patients in the out-of-hospital setting. American Journal of Emergency Medicine. 2018;36(6).

14. Grondman I, Pirvu A, Riza A, Ioana M, Netea MG. Biomarkers of inflammation and the etiology of sepsis. Vol. 48, Biochemical Society Transactions. 2020.

15. Guo Q, Qu P, Cui W, Liu M, Zhu H, Chen W, et al. Organism type of infection is associated with prognosis in sepsis: an analysis from the MIMIC-IV database. BMC Infect Dis. 2023;23(1).

16. Burchard KW. FUNGAL SEPSIS. Infect Dis Clin North Am [Internet]. 1992 Sep 1 [cited 2025 May 22];6(3):677–92. Available from: https://linkinghub.elsevier.com/retrieve/pii/S0891552020304694

17. Klastrup V, Hvass AM, Mackenhauer J, Fuursted K, Schønheyder HC, Kirkegaard H, et al. Site of infection and mortality in patients with severe sepsis or septic shock. A cohort study of patients admitted to a Danish general intensive care unit. Infect Dis. 2016;48(10).

18. Jacobi J. Pathophysiology of sepsis. In: American Journal of Health-System Pharmacy. American Society of Health-Systems Pharmacy; 2002.

19. van der Poll T, Opal SM. Host-pathogen interactions in sepsis. Vol. 8, The Lancet Infectious Diseases. 2008.

20. Antonelli M. Sepsis and septic shock: Pro-inflammatory or anti-inflammatory state? In: Journal of Chemotherapy. 1999.

21. Ward NS, Casserly B, Ayala A. The Compensatory Anti-inflammatory Response Syndrome (CARS) in Critically Ill Patients. Vol. 29, Clinics in Chest Medicine. 2008.

22. Cecconi M, De Backer D, Antonelli M, Beale R, Bakker J, Hofer C, et al. Consensus on circulatory shock and hemodynamic monitoring. Task force of the European Society of Intensive Care Medicine. Intensive Care Med. 2014;40(12).

23. Smith N, Lopez RA, Silberman M, Vernon NT. Distributive Shock (Nursing). StatPearls. 2022.

24. Fulton II MR, Zubair M, Taghavi S. Laboratory Evaluation of Sepsis. StatPearls. 2024.

25. Faix JD. Biomarkers of sepsis. Crit Rev Clin Lab Sci [Internet]. 2013 Jan [cited 2025 May 30];50(1):23–36. Available from: https://pubmed.ncbi.nlm.nih.gov/23480440/

26. Inci K, Aygencel G, Gökçe O, Türkoğlu M, Aydın Kaynar L, Can F, et al. Prognostic value of hyperlactatemia and lactate clearance in septic patients with hematological malignancies. Ann

Hematol [Internet]. 2024 Oct 1 [cited 2025 May 29];103(10). Available from: https://pubmed.ncbi.nlm.nih.gov/39235491/

27. Suetrong B, Walley KR. Lactic acidosis in sepsis: It's Not All anaerobic: Implications for diagnosis and management. Vol. 149, Chest. 2016.

28. Jones AE. Lactate clearance for assessing response to resuscitation in severe sepsis. Vol. 20, Academic Emergency Medicine. 2013.

29. Khan A, Sarma D, Gowda C, Rodrigues G. The role of modified early warning score (Mews) in the prognosis of acute pancreatitis. Oman Med J. 2021;36(3).

30. Santiago González N, García-Hernández M de L, Cruz-Bello P, Chaparro-Díaz L, Rico-González M de L, Hernández-Ortega Y. Modified Early Warning Score: Clinical Deterioration of Mexican Patients Hospitalized with COVID-19 and Chronic Disease. Healthcare (Switzerland). 2023;11(19).

31. Marmiere M, D'Amico F, Monti G, Landoni G. Mastering the Sequential Organ Failure Assessment Score: Critical Choices of Score Statistic, Timing, Imputations, and Competing Risk Handling in Major Trials - A Systematic Review. Crit Care Med [Internet]. 2024 May 1 [cited 2025 May 29];53(5). Available from: https://pubmed.ncbi.nlm.nih.gov/39631051/

32. Pölkki A, Pekkarinen PT, Takala J, Selander T, Reinikainen M. Association of Sequential Organ Failure Assessment (SOFA) components with mortality. Acta Anaesthesiol Scand. 2022;66(6).

33. Niskanen M, Kari A, Nikki P, Iisalo E, Kaukinen L, Rauhala V, et al. Acute Physiology and Chronic Health Evaluation (APACHE II) and Glasgow Coma Scores as predictors of outcome from intensive care after cardiac arrest. Crit Care Med. 1991;19(12).

34. Kattan E, Hernández G. The role of peripheral perfusion markers and lactate in septic shock resuscitation. Vol. 2, Journal of Intensive Medicine. 2022.

35. Khwannimit B, Bhurayanontachai R, Vattanavanit V. Comparison of the accuracy of three early warning scores with SOFA score for predicting mortality in adult sepsis and septic shock patients admitted to intensive care unit. Heart and Lung [Internet]. 2019 May 1 [cited 2025 May 29];48(3):240–4. Available from: https://pubmed.ncbi.nlm.nih.gov/30902348/

36. Chen YX, Li CS. Arterial lactate improves the prognostic performance of severity score systems in septic patients in the ED. American Journal of Emergency Medicine. 2014;32(9).

37. Yoo JW, Lee JR, Jung YK, Choi SH, Son JS, Kang BJ, et al. A combination of early warning score and lactate to predict intensive care unit transfer of inpatients with severe sepsis/septic shock. Korean Journal of Internal Medicine. 2015;30(4).

38. Luo X pan, Wang H hong, Hu S fei, Wu S jing, Xie G hao, Cheng B li, et al. Comparison of three different organ failure assessment score systems in predicting outcome of severe sepsis. Zhonghua Wai Ke Za Zhi. 2009;47(1).

39. Jaiswal P, Agrawal S, Kumar S, Acharya S, Wanjari A, Bawankule S, et al. A two-year cross-sectional study on the impact of serial serum Lactate in comparison with APACHE IV and SOFA Scores in predicting outcomes in patients of sepsis at limited resources rural setup. Journal of Emergency Medicine, Trauma and Acute Care. 2022;2022(5).

40. Su X, Zhang H, Yuan W, Yi M, Fu C, Jiang J, et al. Comparison of four early warning scores in predicting the prognosis of critically ill patients in secondary hospitals. Chinese Critical Care Medicine. 2023;35(10).

41. Morkar DN, Dwivedi M, Patil P. Comparative Study of Sofa, Apache Ii, Saps Ii, as a Predictor of Mortality in Patients of Sepsis Admitted in Medical ICU. J Assoc Physicians India. 2022;70(4).

42. Başpınar B, Erdat EC, Ersoy Ortaç E, Topeli A. Comparison of early warning and sepsis scores for mortality prediction in patients with suspected infection admitted to medical intensive care units. Journal of Health Sciences and Medicine. 2024;7(1).

43. Lokesh SB, Latha V. Comparison of Acute Physiology and Chronic Health Evaluation II and Sequential Organ Failure Assessment intensive care unit scoring system as mortality predictors in intensive care unit patients with sepsis. Asian J Med Sci [Internet]. 2024 Dec 4;15(12):124–9. Available from: https://www.nepjol.info/index.php/AJMS/article/view/71012

44. Badrinath K, Shekhar M, Sreelakshmi M, Srinivasan M, Thunga G, Nair S, et al. Comparison of various severity assessment scoring systems in patients with sepsis in a tertiary care teaching hospital. Indian Journal of Critical Care Medicine. 2018;22(12).

45. Angus DC, Linde-Zwirble WT, Lidicker J, Clermont G, Carcillo J, Pinsky MR. Epidemiology of severe sepsis in the United States: Analysis of incidence, outcome, and associated costs of care. Crit Care Med. 2001;29(7).

46. Schröder J, Kahlke V, Staubach KH, Zabel P, Stüber F. Gender differences in human sepsis. Archives of Surgery. 1998;133(11).

47. Martin GS, Mannino DM, Eaton S, Moss M. The Epidemiology of Sepsis in the United States from 1979 through 2000. New England Journal of Medicine. 2003;348(16).

48. Oh N, Cha WC, Seo JH, Choi SG, Kim JM, Chung CR, et al. ChatGPT Predicts In-Hospital All-Cause Mortality for Sepsis: In-Context Learning with the Korean Sepsis Alliance

Database. Healthc Inform Res [Internet]. 2024 Jul 1 [cited 2025 May 29];30(3):266–76. Available from: https://pubmed.ncbi.nlm.nih.gov/39160785/

49. Van Vught LA, Scicluna BP, Wiewel MA, Hoogendijk AJ, Klouwenberg PMCK, Ong DSY, et al. Association of gender with outcome and host response in critically ill sepsis patients. Crit Care Med. 2017;45(11).

50. Fleischmann C, Scherag A, Adhikari NKJ, Hartog CS, Tsaganos T, Schlattmann P, et al. Assessment of global incidence and mortality of hospital-treated sepsis current estimates and limitations. Am J Respir Crit Care Med. 2016;193(3).

51. Schuetz P, Christ-Crain M, Thomann R, Falconnier C, Wolbers M, Widmer I, et al. Effect of procalcitonin-based guidelines vs standard guidelines on antibiotic use in lower respiratory tract infections: The ProHOSP randomized controlled trial. JAMA. 2009;302(10).

52. Todi S, Chatterjee S, Sahu S, Bhattacharyya M. Epidemiology of severe sepsis in India: an update. Crit Care. 2010;14(Suppl 1).

53. Hotchkiss RS, Karl IE. The Pathophysiology and Treatment of Sepsis. New England Journal of Medicine. 2003;348(2).

54. Fuchs A, Tufa TB, Hörner J, Hurissa Z, Nordmann T, Bosselmann M, et al. Clinical and microbiological characterization of sepsis and evaluation of sepsis scores. PLoS One [Internet]. 2021 Mar 1 [cited 2025 May 31];16(3 March). Available from: https://pubmed.ncbi.nlm.nih.gov/33661970/

55. Seymour CW, Liu VX, Iwashyna TJ, Brunkhorst FM, Rea TD, Scherag A, et al. Assessment of clinical criteria for sepsis for the third international consensus definitions for sepsis and septic shock (sepsis-3). JAMA - Journal of the American Medical Association. 2016;315(8).

56. Mikkelsen ME, Miltiades AN, Gaieski DF, Goyal M, Fuchs BD, Shah C V., et al. Serum lactate is associated with mortality in severe sepsis independent of organ failure and shock. Crit Care Med. 2009;37(5).

57. Jansen TC, Van Bommel J, Woodward R, Mulder PGH, Bakker J. Association between blood lactate levels, Sequential Organ Failure Assessment subscores, and 28-day mortality during early and late intensive care unit stay: A retrospective observational study. Crit Care Med. 2009;37(8).

58. Raith EP, Udy AA, Bailey M, McGloughlin S, MacIsaac C, Bellomo R, et al. Prognostic accuracy of the SOFA score, SIRS criteria, and qSOFA score for in-hospital mortality among adults with suspected infection admitted to the intensive care unit. JAMA - Journal of the American Medical Association [Internet]. 2017 Jan 17 [cited 2025 May 31];317(3):290–300. Available from: https://pubmed.ncbi.nlm.nih.gov/28114553/

59. Vincent JL, Moreno R, Takala J, Willatts S, De Mendonça A, Bruining H, et al. The SOFA (Sepsis-related Organ Failure Assessment) score to describe organ dysfunction/failure. Intensive Care Med. 1996;22(7).

60. Lopes Ferreira F, Peres Bota D, Bross A, Mélot C, Vincent JL. Serial evaluation of the SOFA score to predict outcome in critically ill patients. JAMA. 2001;286(14).

61. Singer M, Deutschman CS, Seymour C, Shankar-Hari M, Annane D, Bauer M, et al. The third international consensus definitions for sepsis and septic shock (sepsis-3). Vol. 315, JAMA - Journal of the American Medical Association. American Medical Association; 2016. p. 801–10.

62. Neumann F, Lobitz O, Fenk R, Bruns I, Köstering M, Steiner S, et al. The sepsis-related Organ Failure Assessment (SOFA) score is predictive for survival of patients admitted to the

intensive care unit following allogeneic blood stem cell transplantation. Ann Hematol. 2008;87(4).

63. Dellinger RP, Levy MM, Carlet JM, Bion J, Parker MM, Jaeschke R, et al. Surviving sepsis campaign: International guidelines for management of severe sepsis and septic shock: 2008. Crit Care Med. 2008;36(1).

64. Bota DP, Melot C, Ferreira FL, Ba VN, Vincent JL. The Multiple Organ Dysfunction Score (MODS) versus the Sequential Organ Failure Assessment (SOFA) score in outcome prediction. Intensive Care Med. 2002;28(11).

65. Zimmerman JE, Kramer AA, McNair DS, Malila FM. Acute Physiology and Chronic Health Evaluation (APACHE) IV: Hospital mortality assessment for today's critically ill patients. Crit Care Med. 2006;34(5).

66. Knaus WA, Draper EA, Wagner DP, Zimmerman JE. APACHE II: A severity of disease classification system. Crit Care Med. 1985;13(10).

67. Tekin B, Kiliç J, Taşkin G, Solmaz I, Tezel O, Başgöz BB. Comparison of scoring systems: SOFA, APACHE-II, LODS, MODS, and SAPS-II in critically ill elderly sepsis patients. J Infect Dev Ctries. 2024;18(1).

68. Kądziolka I, Świstek R, Borowska K, Tyszecki P, Serednicki W. Validation of APACHE II and SAPS II scales at the intensive care unit along with assessment of SOFA scale at the admission as an isolated risk of death predictor. Anaesthesiol Intensive Ther. 2019;51(2).

69. Subbe CP, Kruger M, Rutherford P, Gemmel L. Validation of a modified early warning score in medical admissions. QJM: An International Journal of Medicine. 2001;94(10).

ANNEXURES

ANNEXURE I

Sequential Organ Failure Assessment (SOFA) score						
Organ system		Score				
		0	**1**	**2**	**3**	**4**
Respiratory	PaO2/FiO2(kPa)	>53.3	<53.3	<40	<26.7	<13.3
Renal	S.Creatinine	<110	110-170	171-299	300-440	>440
Hepatic	Bilirubin	<20	20-32	33-101	102-204	>204
Haematological	Platelet count	>150	<150	<100	<50	<20
Neurological	Glasgow Coma Score	15	13-14	10-12	6-9	<6
Cardiovascular		MAP >70mmHg	MAP<70mmHg	Dopamine <5 or Dobutamine	Dopamine 5.1-15, Epinephrine<0.1or Norepinephrine <0.1	Dopamine>15 or Epinephrine >0.1 or Norepinephrine >0.1

APACHE II - Acute Physiology Score									
	4	**3**	**2**	**1**	**0**	**1**	**2**	**3**	**4**
Body temperature	<29.9	30-31.9	32-33.9	34-35.9	36-38.4	38.5-38.9		39-40.9	>41
Mean BP	<49		50-69		70-109		110-129	130-159	>160
Pulse	<39	40-54	55-69		70-109		110-139	140-179	>180
Respiratory rate	<5		6-9	10-11	12-24	25-34		35-49	>50
A-a DO2 PaO2	<55	55-60		61-70	<200 >70		200-349		
Arterial Blood pH No ABG data- HCO3	<7.15 <15	7.15-7.24 15-17.9	7.25-7.32 18-21.9		7.33-7.49 22-31.9	7.50-7.59 32-40.9		7.60-7.69 41-51.9	>7.70 >52
Serum sodium	<110	111-119	120-129		130-149	150-154	155-159	160-179	>180
Serum potassium	<2.5		2.5-2.9	3-3.4	3.5-5.4	5.5-5.9		6-6.9	>7
Hematocrit	<20		20-29.9		30-45.9	46-49.9	50-59.9		>60
WBC(x10^3/mm^3)	<1		1-2.9		3-14.9	15-19.9	20-39.9		>40
Glasgow coma scale	15-Glasgow coma scale								

Modified Early Warning Score (MEWS)							
Score	**3**	**2**	**1**	**0**	**1**	**2**	**3**
Respiratory rate		<9		9-14	15-20	21-29	>30
Heart rate		<40	41-50	51-100	101-110	111-129	>130
Systolic blood pressure	<70	71-80	81-100	101-199		>200	
Temperature		<35		35-38.4		>38.5	
AVPU				Alert	reacting to Voice	reacting to Pain	unresponsive

PROFORMA

"A STUDY TO COMPARE COMBINATION OF MODIFIED EARLY WARNING SCORE (MEWS) AND LACTATE WITH SEQUENTIAL ORGAN FAILURE ASSESSMENT (SOFA)SCORE AND ACUTE PHYSIOLOGY AND CHRONIC HEALTH EVALUATION (APACHE)II SCORE IN PREDICTING SEVERITY IN SEPSIS PATIENTS".

Serial No: Date:

UHID No:

1. NAME :
2. AGE/ SEX :
3. DATE OF ADMISSION :
4. DATE OF DISCHARGE :
5. PROBABLE SOURCE OF SEPSIS :

RESPIRATORY	☐	CARDIOVASCULAR	☐
GENITO URINARY	☐	CUTANEOUS	☐
NEURO INFECTION	☐	UNKNOWN	☐

VITAL PARAMETERS

PULSE RATE: SYSTOLIC BLOOD PRESSURE:

DIASTOLIC BLOOD PRESSURE: MEAN ARTERIAL PRESSURE:

RESPIRATORY RATE: TEMPERATURE:

GCS: AVPU:

ARTERIAL BLOOD GAS ANALYSIS

pH : HCO3 :

PaO2 :

COMPLETE BLOOD COUNT

HEMATOCRIT : PLATELET COUNT :

WBC COUNT :

RENAL FUNCTION TEST

SERUM CREATININE : SERUM Na :

SERUM K :

LIVER FUNCTION TEST

BILIRUBIN :

RESPIRATORY SYSTEM

FiO2 :

Serum Lactate :

Number of days of Hospitalization :

Number of days on Mechanical Ventilator :

Number of days of ICU stay :

Got discharged / Expired :

ANNEXURE – II: PATIENT INFORMATION SHEET

STUDY TITLE: A STUDY TO COMPARE COMBINATION OF MODIFIED EARLY WARNING SCORE (MEWS) AND LACTATE WITH SEQUENTIAL ORGAN FAILURE ASSESSMENT (SOFA)SCORE AND ACUTE PHYSIOLOGY AND CHRONIC HEALTH EVALUATION (APACHE)II SCORE IN PREDICTING SEVERITY IN SEPSIS PATIENTS.

Study site: R.L Jalapa Hospital and Research Centre attached to Sri Devaraj Urs Medical College, affiliated to Sri Devaraj Urs Academy Higher Education & Research,

Aim: To prognosticate the severity and outcome of patients with sepsis based on Combination of Modified Early Warning score with lactate, in comparison with SOFA and APACHE II scores.

This information is intended to give you the general background of the study. Please read the following information and discuss with your family members. You can ask any question regarding the study. If you agree to participate in the study, we will collect information (as per proforma) from you or a person responsible for you or both. Relevant history will be taken. General physical examination, systemic and local examination will be done. We will collect the treatment and relevant details from your hospital record. This information collected will be used for only the dissertation and publication.

All information collected from you will be kept confidential and will not be disclosed to any outsider. Your identity will not be revealed. This study has been reviewed by the Institutional Ethics Committee and you are free to contact the member of the Institutional Ethics Committee.

There is no compulsion to agree to this study. The care you will get will not change if you don't wish to participate. You are required to sign/ provide thumb impression only if you voluntarily agree to participate in this study. The investigations needed are part of routine ICU treatment.

For any further clarification you can contact the study investigator

Dr. Aravind S R

Mobile No: 8129498234

e-mail id: aravindsr54@gmail.com

ಅನುಬಂಧ - II: ರೋಗಿಯ ಮಾಹಿತಿ ಹಾಳೆ

ಅಧ್ಯಯನದ ಶೀರ್ಷಿಕೆ: ಮಾರ್ಪಡಿಸಿದ ಆರಂಭಿಕ ಎಚ್ಚರಿಕೆ ಸ್ಕೋರ್ (MEWS) **ಮತ್ತು ಲ್ಯಾಕ್ಟೇಟ್ ಸಂಯೋಜನೆಯನ್ನು ಅನುಕ್ರಮ ಅಂಗ ವೈಫಲ್ಯ ಮೌಲ್ಯಮಾಪನ (**SOFA) **ಸ್ಕೋರ್ ಮತ್ತು ತೀವ್ರ ಶರೀರಶಾಸ್ತ್ರ ಮತ್ತು ದೀರ್ಘಕಾಲದ ಆರೋಗ್ಯ ಮೌಲ್ಯಮಾಪನ (**APACHE) II **ನೊಂದಿಗೆ ಹೋಲಿಸುವ ಅಧ್ಯಯನ.**

ಅಧ್ಯಯನ ತಾಣ: ಶ್ರೀ ದೇವರಾಜ್ **ಅರಸ್** ವೈದ್ಯಕೀಯ ಕಾಲೇಜಿಗೆ ಲಗತ್ತಿಸಲಾದ ಆರ್.ಎಲ್. ಜಾಲಪ್ಪ ಆಸ್ಪತ್ರೆ ಮತ್ತು ಸಂಶೋಧನಾ ಕೇಂದ್ರ, ಶ್ರೀ ದೇವರಾಜ್ **ಅರಸ್** ಅಕಾಡೆಮಿ ಉನ್ನತ ಶಿಕ್ಷಣ ಮತ್ತು ಸಂಶೋಧನೆಗೆ ಸಂಯೋಜಿತವಾಗಿದೆ,

ಉದ್ದೇಶ: **SOFA** ಮತ್ತು **APACHE II** ಸ್ಕೋರ್‌ಗಳಿಗೆ ಹೋಲಿಸಿದರೆ, ಮಾರ್ಪಡಿಸಿದ ಆರಂಭಿಕ ಎಚ್ಚರಿಕೆ ಸ್ಕೋರ್ ಅನ್ನು ಲ್ಯಾಕ್ಟೇಟ್‌ನೊಂದಿಗೆ ಸಂಯೋಜಿಸುವ ಆಧಾರದ ಮೇಲೆ ಸೆಪ್ಸಿಸ್ ರೋಗಿಗಳ ತೀವ್ರತೆ ಮತ್ತು ಫಲಿತಾಂಶವನ್ನು ಮುನ್ಸೂಚಿಸುವುದು.

ಈ ಮಾಹಿತಿಯು ನಿಮಗೆ ಅಧ್ಯಯನದ ಸಾಮಾನ್ಯ ಹಿನ್ನೆಲೆಯನ್ನು ನೀಡಲು ಉದ್ದೇಶಿಸಲಾಗಿದೆ. ದಯವಿಟ್ಟು ಕೆಳಗಿನ ಮಾಹಿತಿಯನ್ನು ಓದಿ ಮತ್ತು ನಿಮ್ಮ ಕುಟುಂಬ ಸದಸ್ಯರೊಂದಿಗೆ ಚರ್ಚಿಸಿ. ಅಧ್ಯಯನದ ಕುರಿತು ನೀವು ಯಾವುದೇ ಪ್ರಶ್ನೆಯನ್ನು ಕೇಳಬಹುದು. ನೀವು ಅಧ್ಯಯನದಲ್ಲಿ ಭಾಗವಹಿಸಲು ಒಪ್ಪಿದರೆ, ನಾವು ನಿಮ್ಮಿಂದ ಅಥವಾ ನಿಮ್ಮ ಜವಾಬ್ದಾರಿಯುತ ವ್ಯಕ್ತಿಯಿಂದ ಅಥವಾ ಇಬ್ಬರಿಂದಲೂ (ಪ್ರೊಫಾರ್ಮಾ ಪ್ರಕಾರ) ಮಾಹಿತಿಯನ್ನು ಸಂಗ್ರಹಿಸುತ್ತೇವೆ. ಸಂಬಂಧಿತ ಇತಿಹಾಸವನ್ನು ತೆಗೆದುಕೊಳ್ಳಲಾಗುತ್ತದೆ. ಸಾಮಾನ್ಯ ದೈಹಿಕ ಪರೀಕ್ಷೆ, ವ್ಯವಸ್ಥಿತ ಮತ್ತು ಸ್ಥಳೀಯ ಪರೀಕ್ಷೆಯನ್ನು ಮಾಡಲಾಗುತ್ತದೆ. ನಿಮ್ಮ ಆಸ್ಪತ್ರೆಯ ದಾಖಲೆಯಿಂದ ಚಿಕಿತ್ಸೆ ಮತ್ತು ಸಂಬಂಧಿತ ವಿವರಗಳನ್ನು ನಾವು ಸಂಗ್ರಹಿಸುತ್ತೇವೆ. ಸಂಗ್ರಹಿಸಿದ ಈ ಮಾಹಿತಿಯನ್ನು ಪ್ರಬಂಧ ಮತ್ತು ಪ್ರಕಟಣೆಗೆ ಮಾತ್ರ ಬಳಸಲಾಗುತ್ತದೆ.

ನಿಮ್ಮಿಂದ ಸಂಗ್ರಹಿಸಲಾದ ಎಲ್ಲಾ ಮಾಹಿತಿಯನ್ನು ಗೌಪ್ಯವಾಗಿಡಲಾಗುತ್ತದೆ ಮತ್ತು ಯಾವುದೇ ಹೊರಗಿನವರಿಗೆ ಬಹಿರಂಗ ಪಡಿಸಲಾಗುವುದಿಲ್ಲ. ನಿಮ್ಮ ಗುರುತನ್ನು ಬಹಿರಂಗ ಪಡಿಸಲಾಗುವುದಿಲ್ಲ. ಈ ಅಧ್ಯಯನವನ್ನು ಸಾಂಸ್ಥಿಕ ನೀತಿ ಸಮಿತಿಯು ಪರಿಶೀಲಿಸಿದೆ ಮತ್ತು ನೀವು ಸಾಂಸ್ಥಿಕ ನೀತಿ ಸಮಿತಿಯ ಸದಸ್ಯರನ್ನು ಸಂಪರ್ಕಿಸಲು ಮುಕ್ತರಾಗಿದ್ದೀರಿ.

ಈ ಅಧ್ಯಯನಕ್ಕೆ ಒಪ್ಪಿಕೊಳ್ಳಲು ಯಾವುದೇ ಬಲವಂತವಿಲ್ಲ. ನೀವು ಭಾಗವಹಿಸಲು ಬಯಸದಿದ್ದರೆ ನೀವು ಪಡೆಯುವ ಆರೈಕೆ ಬದಲಾಗುವುದಿಲ್ಲ. ನೀವು ಈ ಅಧ್ಯಯನದಲ್ಲಿ ಸ್ವಯಂಪ್ರೇರಣೆಯಿಂದ ಭಾಗವಹಿಸಲು ಒಪ್ಪಿದರೆ ಮಾತ್ರ ನೀವು ಸಹಿ/ಹೆಬ್ಬೆರಳಿನ ಗುರುತನ್ನು ಒದಗಿಸಬೇಕಾಗುತ್ತದೆ. ಅಗತ್ಯವಿರುವ ತನಿಖೆಗಳು ದಿನನಿತ್ಯದ ಐಸಿಯು ಚಿಕಿತ್ಸೆಯ ಭಾಗವಾಗಿದೆ.

ಯಾವುದೇ ಹೆಚ್ಚಿನ ಸ್ಪಷ್ಟೀಕರಣಕ್ಕಾಗಿ ನೀವು ಅಧ್ಯಯನ ತನಿಖಾಧಿಕಾರಿಯನ್ನು ಸಂಪರ್ಕಿಸಬಹುದು

ಡಾ. ಅರವಿಂದ್ ಎಸ್ ಆರ್

ಮೊಬೈಲ್ ಸಂಖ್ಯೆ: **8129498234**

ಇ-ಮೇಲ್ ಐಡಿ: **aravindsr54@gmail.com**

ANNEXURE – III: WRITTEN INFORMED CONSENT

WRITTEN INFORMED CONSENT

"A study to compare combination of modified early warning score (MEWS) and lactate with sequential organ failure assessment (SOFA)score and acute physiology and chronic health evaluation (APACHE)II score in predicting severity in sepsis patients".

If you agree to participate in the study, we will collect information (as per proforma) from you or a person responsible for you or both. We will collect the treatment and relevant details from your hospital record. This information collected will be used for only dissertation and publication. This study has been reviewed by the Institutional Ethics Committee. There is no compulsion to agree to this study. The care you will get will not change if you don't wish to participate. You are required to sign/ provide a thumb impression only if you voluntarily agree to participate in this study.

I understand that I remain free to withdraw from the study at any time and this will not change my future care. I have read or have been read to me and understand the purpose of the study, the risk and benefits associated with my involvement in the study and the nature of information that will be collected and disclosed during the study. I have had the opportunity to ask my questions regarding various aspects of the study and my questions were answered to my satisfaction. I, the undersigned agree to participate in this study and authorize the collection and disclosure of my personal information for dissertation.

Subject name: DATE:

Signature / Thumb impression:

Parents / Guardians name:

ಅನುಬಂಧ - III: ಲಿಖಿತ ಮಾಹಿತಿಯುಕ್ತ ಒಪ್ಪಿಗೆ

ಲಿಖಿತ ಮಾಹಿತಿಯುಕ್ತ ಒಪ್ಪಿಗೆ

"ಮಾರ್ಪಡಿಸಿದ ಆರಂಭಿಕ ಎಚ್ಚರಿಕೆ ಸ್ಕೋರ್ (MEWS) ಮತ್ತು ಲ್ಯಾಕ್ಟೇಟ್ ಸಂಯೋಜನೆಯನ್ನು ಅನುಕ್ರಮ ಅಂಗ ವೈಫಲ್ಯ ಮೌಲ್ಯಮಾಪನ (SOFA) ಸ್ಕೋರ್ ಮತ್ತು ತೀವ್ರ ಶರೀರಶಾಸ್ತ್ರ ಮತ್ತು ದೀರ್ಘಕಾಲದ ಆರೋಗ್ಯ ಮೌಲ್ಯಮಾಪನ (APACHE) II ಸ್ಕೋರ್‌ನೊಂದಿಗೆ ಸೆಪ್ಸಿಸ್ ರೋಗಿಗಳಲ್ಲಿ ತೀವ್ರತೆಯನ್ನು ಊಹಿಸುವಲ್ಲಿ ಹೋಲಿಸಲು ಒಂದು ಅಧ್ಯಯನ".

ನೀವು ಅಧ್ಯಯನದಲ್ಲಿ ಭಾಗವಹಿಸಲು ಒಪ್ಪಿದರೆ, ನಾವು ನಿಮ್ಮಿಂದ ಅಥವಾ ನಿಮಗೆ ಜವಾಬ್ದಾರರಾಗಿರುವ ವ್ಯಕ್ತಿಯಿಂದ ಅಥವಾ ಇಬ್ಬರಿಂದಲೂ ಮಾಹಿತಿಯನ್ನು (ಪ್ರೊಫಾರ್ಮಾ ಪ್ರಕಾರ) ಸಂಗ್ರಹಿಸುತ್ತೇವೆ. ನಿಮ್ಮ ಆಸ್ಪತ್ರೆ ದಾಖಲೆಯಿಂದ ನಾವು ಚಿಕಿತ್ಸೆ ಮತ್ತು ಸಂಬಂಧಿತ ವಿವರಗಳನ್ನು ಸಂಗ್ರಹಿಸುತ್ತೇವೆ. ಸಂಗ್ರಹಿಸಿದ ಈ ಮಾಹಿತಿಯನ್ನು ಪ್ರಬಂಧ ಮತ್ತು ಪ್ರಕಟಣೆಗಾಗಿ ಮಾತ್ರ ಬಳಸಲಾಗುತ್ತದೆ. ಈ ಅಧ್ಯಯನವನ್ನು ಸಾಂಸ್ಥಿಕ ನೀತಿ ಸಮಿತಿಯು ಪರಿಶೀಲಿಸಿದೆ. ಈ ಅಧ್ಯಯನಕ್ಕೆ ಒಪ್ಪಿಕೊಳ್ಳಲು ಯಾವುದೇ ಬಲವಂತವಿಲ್ಲ. ನೀವು ಭಾಗವಹಿಸಲು ಬಯಸದಿದ್ದರೆ ನೀವು ಪಡೆಯುವ ಆರೈಕೆ ಬದಲಾಗುವುದಿಲ್ಲ. ನೀವು ಸ್ವಯಂಪ್ರೇರಣೆಯಿಂದ ಈ ಅಧ್ಯಯನದಲ್ಲಿ ಭಾಗವಹಿಸಲು ಒಪ್ಪಿಕೊಂಡರೆ ಮಾತ್ರ ನೀವು ಸಹಿ/ಹೆಬ್ಬೆರಳಿನ ಗುರುತನ್ನು ಒದಗಿಸಬೇಕಾಗುತ್ತದೆ.

ನಾನು ಯಾವುದೇ ಸಮಯದಲ್ಲಿ ಅಧ್ಯಯನದಿಂದ ಹಿಂದೆ ಸರಿಯಲು ಸ್ವತಂತ್ರನಾಗಿರುತ್ತೇನೆ ಮತ್ತು ಇದು ನನ್ನ ಭವಿಷ್ಯದ ಆರೈಕೆಯನ್ನು ಬದಲಾಯಿಸುವುದಿಲ್ಲ ಎಂದು ನಾನು ಅರ್ಥಮಾಡಿಕೊಂಡಿದ್ದೇನೆ. ನಾನು ಓದಿದ್ದೇನೆ ಅಥವಾ ನನಗೆ ಓದಿ ಹೇಳಿದ್ದೇನೆ ಮತ್ತು ಅಧ್ಯಯನದ ಉದ್ದೇಶ, ಅಧ್ಯಯನದಲ್ಲಿ ನನ್ನ ಭಾಗವಹಿಸುವಿಕೆಗೆ ಸಂಬಂಧಿಸಿದ ಅಪಾಯಗಳು ಮತ್ತು ಪ್ರಯೋಜನಗಳು ಮತ್ತು ಅಧ್ಯಯನದ ಸಮಯದಲ್ಲಿ ಸಂಗ್ರಹಿಸಲಾದ ಮತ್ತು ಬಹಿರಂಗಪಡಿಸಲಾದ ಮಾಹಿತಿಯ ಸ್ವರೂಪವನ್ನು ಅರ್ಥಮಾಡಿಕೊಂಡಿದ್ದೇನೆ. ಅಧ್ಯಯನದ ವಿವಿಧ ಅಂಶಗಳ ಕುರಿತು ನನ್ನ ಪ್ರಶ್ನೆಗಳನ್ನು ಕೇಳಲು ನನಗೆ ಅವಕಾಶ ಸಿಕ್ಕಿದೆ ಮತ್ತು ನನ್ನ ಪ್ರಶ್ನೆಗಳಿಗೆ ನನಗೆ ತೃಪ್ತಿಯಾಗುವಷ್ಟು ಉತ್ತರಿಸಲಾಗಿದೆ. ಕೆಳಗೆ ಸಹಿ ಮಾಡಿರುವ ನಾನು ಈ ಅಧ್ಯಯನದಲ್ಲಿ ಭಾಗವಹಿಸಲು ಒಪ್ಪುತ್ತೇನೆ ಮತ್ತು ಪ್ರಬಂಧಕ್ಕಾಗಿ ನನ್ನ ವೈಯಕ್ತಿಕ ಮಾಹಿತಿಯನ್ನು ಸಂಗ್ರಹಿಸಲು ಮತ್ತು ಬಹಿರಂಗಪಡಿಸಲು ಅಧಿಕಾರ ನೀಡುತ್ತೇನೆ.

ವಿಷಯದ ಹೆಸರು: ದಿನಾಂಕ:

ಸಹಿ/ಹೆಬ್ಬೆರಳಿನ ಗುರುತು:

ಪೋಷಕರು/ಪೋಷಕರ ಹೆಸರು:

MASTER CHART

Sl.No	Age	Sex	Source of sepsis	PR	SBP	DBP	MAP	RR	TEMP	GCS_	GCS	AVPU	pH	HCO3	PaO2	PCV	PLT	WBC	SCr	SNa	SK	bilirubin	FiO2	Hospitalization	Ventilator	ICU	DIS/DECEASED	MEWS	SOFA	APACHE II	PCO2	LACTATE
1	48	M	N	102	50	30	37	24	101	10	E2V3M5	P	7.1	16.5	70	32	1.2	16000	1.2	130	3.7	1.2	52	15	7	11	D	8	7	26	40	4.6
2	35	F	R	104	60	30	40	26	100	15	15	A	7.28	18.5	79.6	22	1.4	7000	0.7	133	3.8	1.7	37	11	0	7	D	6	5	13	38	5.27
3	40	M	N	110	80	40	53	24	100.2	12	E3V4M5	A	7.42	17.2	86.2	34	2.5	17240	0.8	140	4	0.9	52	8	0	5	D	5	7	13	28	3
4	65	M	O	116	70	40	50	26	100.4	15	15	A	7.35	19.1	84.2	37	1.4	14560	3.3	129	5.2	1.1	29	11	0	6	D	7	8	19	30	2
5	60	M	C	102	70	40	50	28	99.9	15	15	A	7.23	12.8	67.7	30	2.1	6000	0.8	138	3.7	2	21	10	0	3	D	6	4	14	40	3
6	40	M	R	130	40	20	27	28	101	15	15	A	7.12	12	90	26	80000	10970	1.2	133	3.6	1	70	12	5	9	D	8	9	19	56	2.47
7	55	M	R	120	80	60	67	24	99.6	7	E1VtM1	P	7.18	18.5	79.1	41	46000	11400	1.4	147	4.3	0.7	100	4	4	4	M	8	13	30	40	12
8	80	F	C	98	90	60	70	20	98	15	15	A	7.43	19.8	71.5	26	2	11000	2.7	115	5.1	2.2	21	16	0	3	D	2	6	18	42	2.2
9	52	M	U	120	100	70	80	30	99	12	E3V4M5	P	6.9	14	72	36	1.4	13500	3.5	129	5	0.8	100	12	6	9	M	8	10	30	32	8
10	65	M	C	110	90	60	70	24	101	15	15	A	6.9	5	77	34	3.3	15600	1.4	146	4.3	1.4	43	10	6	10	M	4	6	12	28	9.4
11	45	M	G	128	60	40	47	28	100	14	E4V4M6	A	7.19	12.1	47.1	32	3.6	14200	1.6	122	3.8	1.3	35	20	4	12	D	7	7	25	30	6.2
12	75	M	C	96	80	50	60	20	98	15	15	A	7.52	23.3	87.8	30	3.89	18380	2.5	128	3.6	1.4	21	22	0	4	D	3	5	18	43	2.4
13	53	M	U	110	70	50	57	24	100	15	15	A	7.45	16	65.5	36	2.68	18410	4.8	119	6.2	1	21	16	0	10	D	6	5	25	24	3
14	69	M	R	112	50	30	37	28	99	15	15	A	7.55	20	79.3	26	2.7	12200	1.7	130	3.6	1.1	47	12	0	7	D	7	7	21	24	1.3
15	65	F	R	96	80	40	53	30	97.8	11	E3V3M5	V	7.11	13.1	89	27	1.69	18410	1.2	125	4.4	0.6	56	13	4	8	M	6	9	26	52	4
16	36	M	U	148	50	30	37	32	98	10	E3V2M5	P	6.8	11.4	72	33	2.12	9000	3	134	5.87	3.2	100	2	2	2	M	11	14	32	28	8.2
17	66	M	R	116	70	40	50	28	100.1	15	15	A	7.31	12	80	32	1.48	6000	0.7	144	4.7	1.3	70	16	14	14	M	9	7	19	36	6.43
18	41	M	U	132	70	50	57	20	101	15	15	A	7.14	9	70	30	1.54	7200	1.8	138	5.3	1.7	35	7	0	3	D	7	7	15	32	2.43
19	65	F	C	114	70	40	50	18	98.9	15	15	A	7.43	16	76	24	1.4	18000	5.7	128	5.6	2.3	47	28	0	3	D	6	12	22	36	6
20	85	F	R	96	80	60	67	28	100.2	15	15	A	7.52	19.4	70	34	1.54	6050	0.9	121	3.9	1.1	39	10	0	5	D	4	4	17	38	3.4
21	82	M	C	96	100	60	73	18	98	15	15	A	7.31	10.1	140	23.7	1.6	6700	1.3	136	3.4	0.8	21	8	0	3	D	2	2	15	30	1.8
22	64	M	C	150	100	70	80	22	99	15	15	A	7.47	13.9	78	25	1.45	10230	1.6	122	4.7	2	21	10	0	4	D	6	5	16	36	5.83
23	36	F	U	88	80	40	53	22	98.8	15	15	A	7.1	16	69	23	1.38	18500	2.1	127	5	2.4	29	7	0	2	D	4	9	18	44	1.89
24	80	F	R	102	110	70	83	28	99	15	15	A	7.35	17.1	97	44	98000	9740	1	136	4.5	0.8	43	9	0	5	D	3	4	11	36	1.9
25	60	F	C	120	90	70	77	24	102	13	E4V3M6	A	7.05	4.7	150	45	1.89	19670	1.4	138	5	0.6	21	10	0	3	D	7	2	16	23	5.76
26	39	F	G	116	90	60	70	20	100	15	15	A	7.15	10.2	76.5	36	1.54	19620	0.8	140	4.3	2.7	29	8	0	5	D	4	5	9	21	1.06
27	38	F	G	142	50	30	37	26	100	13	E3V4M6	V	6.8	8.3	83	32	2.4	17200	4.5	120	5.4	2.2	70	18	5	12	D	9	12	31	19	18.98
28	35	F	U	100	100	70	80	18	101	15	15	A	7.36	22	88	42	1.9	18500	0.8	138	3.7	3.5	21	10	0	2	D	2	2	4	37	8.59
29	65	F	R	110	90	50	53	24	98	15	15	A	7.3	20	76	23.9	1	12000	1	128	3.6	1.4	29	5	0	0	D	4	6	19	35	2.56
30	85	M	C	110	100	70	80	22	99	15	15	A	7.38	16.9	77.2	23.7	1.2	14000	2	116	4.5	2.2	21	7	5	7	M	4	6	20	27	3
31	24	M	C	88	80	40	53	26	99	11	E3V3M5	V	7.2	7.6	89.5	30	2	18000	3.2	122	7.13	3	35	12	0	7	D	5	11	26	28	7.32
32	52	F	G	60	40	20	27	14	103	4	E1V2M1	U	7	9.5	40	23	89000	22000	2	134	5.1	0.6	100	7	7	7	M	8	15	39	15	13.81
33	35	F	R	128	40	20	27	24	99.8	15	15	A	7.42	14.7	200	26	1.19	15600	1.8	133	3.04	0.7	51	8	0	5	D	7	7	15	29	9.02
34	55	M	G	120	80	60	67	22	100	15	15	A	7.22	9.2	69	25	1.55	10900	0.9	138	4.5	1.6	21	8	0	4	D	6	3	17	25	4.21
35	72	M	C	110	160	100	120	24	102	14	E4V4M6	A	7.49	19.6	85	22	2.3	16400	0.7	110	3.99	2.5	21	7	0	0	D	5	3	21	17	6
36	58	F	R	140	100	60	73	30	100.2	15	15	A	7.09	17	80	27.5	2.2	12300	0.9	140.1	3.15	0.9	60	9	0	5	D	7	2	20	39	10
37	60	M	C	128	40	20	27	28	102	12	E3V4M5	V	6.8	9.5	56	14.7	1.3	12250	4.2	130	6.67	1.1	100	7	7	7	M	10	14	41	12	7.88
38	40	M	G	113	50	30	37	32	101	15	15	A	6.93	4.2	43.6	32	1.4	9780	1.2	129.2	3.69	0.5	60	14	0	9	D	8	8	20	20	11.55
39	34	M	U	102	80	60	67	30	99.7	9	E2V2M5	P	6.73	2.3	56	22	1.19	12320	3.2	129	6.2	1	100	4	4	4	M	8	15	34	48	14
40	75	M	U	90	100	50	67	22	98.9	15	15	A	7.3	10.8	96.1	42	2.1	15400	1.9	128	5.98	2.5	39	9	0	4	D	3	9	19	38	1.85
41	45	M	G	102	80	60	67	26	98	15	15	A	7.18	14.5	199	27.5	1.45	14610	0.8	131	4.2	0.9	39	6	0	3	D	5	5	14	40	8.6
42	32	M	U	110	90	50	63	22	97	15	15	A	7.2	12.4	88	20	2	12350	1.4	133.1	5.9	1.3	21	7	0	3	D	4	3	14	30	8.91
43	51	F	O	122	60	40	47	26	100	15	15	A	7	10	75	18.5	1.4	5600	2	127	5	0.8	60	9	0	5	D	7	7	28	20	10
44	32	M	U	96	100	50	67	16	102	13	E3V4M6	A	6.9	7.4	82	24	1	17430	2	130	3.8	1.5	21	15	0	9	D	4	7	18	29	15.5
45	72	F	N	98	90	50	63	12	98	9	E2V2M5	P	7.1	14	76	24	3.2	8600	1.2	112	3.7	0.8	100	6	6	6	M	3	8	30	42	12.8
46	59	M	R	116	80	60	67	32	99.8	13	13	A	7.2	21	53	28.6	2.5	7650	1.4	127.6	3.1	0.7	56	10	0	7	D	7	5	20	40	
47	76	M	R	112	70	40	50	30	99	15	15	A	6.9	4	137	36	1	5400	1.3	119	6.4	1	100	20	9	14	M	8	8	29	19	11.85
48	43	M	U	110	100	60	73	18	100	15	15	A	7.42	17.4	90	30	2	16000	1.8	133	3.9	1.7	21	12		8	D	4	2	5	38	6
49	50	M	C	104	80	50	60	22	98	12	E3V4M5	P	7.13	7.1	157.6	24	1.2	13450	2	120.8	5.37	1.3	43	12	0	5	D	7	10	22	17	5.83
50	43	M	R	126	100	60	73	30	100	12	E3V4M5	V	6.98	17.4	79.9	18.8	2.38	8650	1	136	3.7	0.7	100	13	2	8	D	7	8	22	75	13.17

Sl.No	Age	Sex	Source of sepsis	PR	SBP	DBP	MAP	RR	TEMP	GCS_	GCS	AVPU	pH	HCO3	PaO2	PCV	PLT	WBC	SCr	SNa	SK	bilirubin	FiO2	Hospitalization	Ventilator	ICU	DIS/DECEASED	MEWS	SOFA	APACHE II	PCO2	LACTATE
51	66	M	N	110	60	40	47	24	101	9	E2V3M4	P	7.1	20	62	32	2.6	13230	0.8	110	3.6	1.2	35	15	3	9	D	8	10	26	56	8.2
52	20	F	U	120	80	50	60	28	98.7	14	E4V4M6	A	7.21	5.4	79.6	26.8	2.92	20950	0.77	129	3.17	0.97	70	10	0	8	D	6	6	19	14	17.2
53	56	M	R	110	90	50	63	26	99	15	15	A	7.16	8.4	38.5	20	3	6700	3	133.4	4.37	1	60	11	0	7	D	4	5	22	24	4
54	65	M	R	116	100	60	73	32	103	15	15	A	7	17	60.2	28	1.4	6000	2.2	118	5.8	1.5	70	14	0	8	D	8	6	32	43	5.2
55	42	F	G	110	90	50	63	18	100.7	15	15	A	7.3	11	86	28	2.4	11420	0.8	144.8	4.1	1	21	5	0	3	D	3	1	8	38	6.42
56	65	F	U	124	80	50	60	22	102.2	13	13	V	6.8	9.4	77.8	42	1.5	16850	2.7	140.9	5.7	1.3	70	4	4	4	M	9	9	31	49	18.2
57	70	M	R	118	90	60	70	32	100	11	E4V2M5	A	7.12	18	52	42	90000	17660	1.3	142	4.8	0.7	100	3	3	3	M	6	10	26	38	2.8
58	42	F	R	114	100	50	67	28	98.9	15	15	A	7.32	30	72	38	72000	11000	0.9	1348	3.7	0.6	35	9	0	6	D	5	5	7	42	2.6
59	50	M	C	102	70	40	50	20	102	15	15	A	7.2	15	80	26	2.4	19830	3.4	137	6	0.9	21	12	0	7	D	5	6	25	35	7.23
60	48	F	N	98	100	50	67	18	103	12	E3V4M5	V	7.43	25.7	80.7	38	1.8	8930	0.9	130	4.1	1.4	21	11	0	9	D	5	7	10	40	7.72
61	85	F	R	106	90	50	70	26	100	12	E3V4M5	V	7.12	22	68.2	26	2.4	7500	2	132	4.5	1.4	43	10	0	6	D	5	8	25	27	3.86
62	38	M	U	110	100	60	73	22	102	15	15	A	7.32	28	72	36	1.8	14720	1.1	142	4	0.7	21	14	0	10	D	6	1	5	32	6.8
63	65	M	C	112	60	40	47	24	101.6	15	15	A	6.9	10.2	82	42	1.6	13450	2.8	132	5.2	0.8	21	13	0	10	D	9	7	22	30	3
64	59	M	C	100	90	60	70	16	100.8	15	15	A	7.48	32	76	24	68000	13210	1.7	137	3.7	1.4	21	7	0	5	D	2	5	7	37	7.33
65	48	M	R	124	100	70	80	20	99.4	15	15	A	7.28	26.6	110	30	1.8	10250	1.5	129	3.9	2	35	12	0	7	D	4	4	10	36	3
66	48	M	U	110	110	60	77	16	101.7	15	15	A	7.31	15	74	45	2.8	19500	1.2	121	4	1.4	21	10	0	7	D	2	3	10	39	8
67	66	M	U	116	50	0	23	28	101	13	13	V	6.92	11.2	75	37	2	22340	1.5	116	5.1	0.7	21	14	0	10	D	8	7	30	32	6.2
68	47	F	R	100	80	50	60	30	99.5	15	15	A	7.28	22	52	28	1.8	6700	0.9	147	2.8	1.1	43	12	0	9	D	5	5	19	26	4.2
69	73	F	R	128	50		23	32	99.8	11	E4V2M5	A	7.21	21	54	21	1.4	7256	1.1	133	2.53	1	70	5	5	5	M	8	11	31	71	2.2
70	67	M	N	120	70	50	57	20	103	7	E1V2M4	P	7.52	49.5	49.2	21	2.3	9852	0.8	134	2.56	1.4	100	3	3	3	M	10	11	29	61	2.3
71	69	M	R	112	90	50	63	30	99	12	E3V4M5	V	7.17	15.3	187	26	2.8	7000	1.4	132	4.6	0.8	70	14	4	10	D	7	7	20	54	4.1
72	31	M	G	108	80	50	60	18	98.2	15	15	A	7.46	18.9	95.5	39	2.6	14564	1	133.7	3.85	1.3	21	8	0	7	D	4	3	2	27	2.95
73	27	F	C	118	80	60	67	24	101	15	15	A	7.37	9.9	101.1	37	3	13452	1.4	137.3	4.83	1	43	10	0	8	D	6	5	4	17	3.63
74	70	M	U	102	70	40	50	20	100	15	15	A	7.37	13.1	78.4	29	2.8	10844	1.8	134.2	4.2	0.8	21	11	0	9	D	5	5	11	23	4.85
75	73	M	N	128	50		23	30	101	12	12	V	7.12	10	67	41	2.2	18452	1.2	136	3.9	0.9	60	10	10	10	M	9	10	27	40	6.09
76	70	F	U	104	90	60	70	16	98	15	15	A	7.27	13.3	70	14	1	5670	1.7	131.6	6.04	0.8	21	18	0	14	D	3	3	22	30	7.82
77	60	F	R	100	90	50	63	28	101	15	15	A	7.3	20	70	33	3.2	8676	1.1	138	3.8	0.7	43	11	0	6	D	3	4	9	44	3.4
78	57	M	G	102	80	60	67	18	98	15	15	A	7.36	20.6	77.9	41	3.5	9678	0.9	138	3.65	1.4	21	8	0	6	D	4	3	5	37	2.8
79	42	M	G	116	50		23	22	98.8	12	E3V4M5	V	7.18	20.2	94	42	1.1	18262	1	132.4	4.36	0.7	30	17	0	12	D	8	8	13	44	3.1
80	55	M	R	102	90	50	63	30	99	15	15	A	7.15	5.2	268	29	1.3	8790	1.3	120.6	5.94	0.9	70	14	0	10	D	5	4	16	15	6.5
81	60	F	G	102	60	40	47	20	100	15	15	A	7.45	16.4	65.2	35	3	9856	0.8	127	2.88	1.2	21	12	0	10	D	5	4	12	24	7.93
82	65	M	G	104	80	40	53	18	100	15	15	A	7.21	8.4	107	53	4	6058	2.4	127.8	4.14	1	43	13	0	7	D	4	7	20	21	7.2
83	42	M	N	114	100	50	67	16	102	10	E2V3M5	P	7.04	7.6	58.4	37	98000	6500	0.8	127.4	5.91	1	43	16	0	12	D	8	7	20	29	11.16
84	24	F	O	108	80	60	67	16	99	15	15	A	7.35	18	78	30	3.2	11234	0.7	132.6	4.36	1.2	21	6	0	5	D	4	3	2	38	2.4
85	36	M	N	100	90	50	63	16	100	12	E3V4M5	V	7.46	22	80	32	82000	7250	0.8	136	3.9	0.9	21	14	0	8	D	3	6	5	21	4
86	73	M	U	112	70	50	57	12	98	6	E1V2M2	U	6.97	3.6	123	27	1.24	7000	3	118	5.2	1.3	100	2	2	2	M	8	13	40	16	16.27
87	61	M	R	122	70	40	50	32	99	12	E4V3M5	A	7.23	19	56	31	2.5	5080	1.7	142	5	0.8	43	17	0	12	D	8	8	19	28	3.32
88	51	M	R	130	80	50	60	34	99	15	15	A	7.43	15.8	84.9	36	1.7	6000	1.5	129.4	4.7	0.5	70	14	0	10	D	8	6	14	24	3.41
89	67	F	O	118	50		23	24	102	11	E3V3M5	V	6.91	9.5	68	39	90000	8654	2.4	146	5.5	1	100	4	4	4	M	10	14	33	48	14.56
90	63	F	U	126	70	50	57	20	103	12	E3V4M5	V	7.26	17.9	67	41	2.3	7000	4.5	136.2	6.6	0.6	21	19	3	14	D	9	9	29	40	8.08
91	45	M	G	102	60	40	47	20	99	15	15	A	7.34	12.7	80.6	19	80000	6000	0.9	125	2.89	0.8	21	8	0	6	D	5	6	12	24	2.4
92	51	M	R	110	100	40	60	28	100	15	15	A	7.4	19.3	63	37	3.5	5000	1	134.2	3.9	0.7	43	10	0	7	D	4	3	8	27	3
93	48	M	C	108	50		23	22	98	15	15	A	7.29	8	86.8	16	1.5	21000	1.7	132.6	5.18	0.9	21	16	0	13	D	6	5	17	17	5.85
94	55	M	O	110	100	50	67	22	102	14	14	A	6.98	6.2	92	28	3.1	14240	2.6	133.7	5.71	1.3	43	12	3	8	D	6	7	22	27	4.2
95	56	M	G	110	80	50	60	24	98	15	15	A	7.45	18.1	67.1	38	2.34	10122	1.1	136	3.49	0.9	21	8	0	7	D	7	3	9	26	3.96
96	55	M	O	126	90	50	63	20	102.5	15	15	A	7.41	15.9	102	13	60000	4320	1	133.2	2.56	0.8	70	8	0	5	D	6	5	19	25	4.75
97	70	M	C	98	80	50	60	28	101	9	E2V2M5	P	7.25	16.1	62	31	1.1	24234	1.5	130	3.48	1.3	70	22	7	14	D	5	11	25	37	4.69
98	35	F	G	112	70	40	50	20	102	14	E4V4M6	A	7.44	21.8	75.1	35	2	6542	0.8	124.3	4.15	0.7	21	10	0	8	D	8	5	8	33	4.34
99	82	M	N	96	70	50	57	16	100	8	E1V2M5	P	7.19	17	78	29	2.5	11000	1	118.9	5.2	1.4	100	16	6	12	D	6	11	27	42	5.44
100	50	F	N	98	80	50	60	20	100	11	E3V3M5	V	7.29	8.2	145	38	1.8	16700	1.2	138.2	3.5	1.5	70	10	0	6	D	5	7	14	17	6.5

Sl.No	Age	Sex	Source of sepsis	PR	SBP	DBP	MAP	RR	TEMP	GCS_	GCS	AVPU	pH	HCO3	PaO2	PCV	PLT	WBC	SCr	SNa	SK	bilirubin	FiO2	Hospitalization	Ventilator	ICU	DIS/DECEASED	MEWS	SOFA	APACHE II	PCO2	LACTATE
101	70	M	U	120	70	40	50	30	99.8	8	E2V2M4	P	7.1	7.9	122.6	37	1.4	22000	0.8	147.5	2.98	1.4	100	2	2	2	M	10	11	34	26	18.52
102	62	M	G	112	40	20	27	10	98	4	E1V2M1	U	7.42	14	85	47	45000	13800	1.7	138	2.9	1.2	100	9	9	9	M	8	15	30	23	6.12
103	58	M	U	118	60	40	47	22	98	11	E3V3M5	V	7.12	18	76	50	1.2	14500	1.8	132	3.4	1.1	21	8	0	4	D	8	8	22	48	3.48
104	67	F	U	120	50	30	37	22	99	15	15	A	7.16	10	70	36	2.51	12600	0.7	140	3.9	0.2	21	9	0	6	D	7	4	15	32	3.9
105	68	M	R	122	100	40	60	30	100	15	15	A	7.15	30	67	34	2	13000	0.8	138	4	0.4	56	9	1	5	D	6	5	15	80	4
106	66	F	R	128	60	40	47	28	101	15	15	A	7.26	22	84	39	1.8	16600	1	133	2.4	0.6	56	8	0	4	D	7	5	21	54	2
107	33	M	N	120	80	50	60	22	102	9	E2V2M5	P	7.35	17	320	28	1.94	15000	0.8	141	4.5	1.7	100	10	3	7	D	10	8	15	31	3.6
108	20	F	U	92	40	20	27	18	98	9	E2V2M5	P	7.24	16.3	121	24	2	18600	1	130	3.62	0.4	50	10	2	6	D	6	9	16	38	2
109	69	F	N	112	90	50	63	12	101	9	E2V2M5	P	7.03	19	123	35	1.1	14700	0.4	132	3.2	0.5	60	6	6	6	M	5	7	24	75	3
110	60	M	U	90	80	50	60	20	99	15	15	A	7.25	14.5	117	22	1.4	14500	0.8	131	4.08	0.7	40	7	0	4	D	3	6	9	33	2.3
111	56	M	C	100	70	50	57	20	101	15	15	A	7.19	14	74	36	1.3	20000	1	141	4.6	0.3	43	10	0	5	D	4	6	10	41	4.1
112	35	M	G	116	90	60	70	18	100	15	15	A	7	11	70	32	2.5	16500	1.2	138	3.8	0.4	21	6	0	5	D	4	4	8	38	2.3
113	30	F	U	130	60	40	47	24	99	9	E2V2M5	P	7.2	10.3	220	20	1.8	21000	3	131	5.6	0.3	100	7	7	7	M	10	10	26	27	8.1
114	70	M	O	120	80	50	60	16	103	15	15	A	7.1	12	56	38	3	6600	1	133	3.9	0.5	21	9	0	5	D	7	5	19	33	3.1
115	61	F	R	102	90	60	70	24	100	15	15	A	7.22	22	121	37	3.7	12700	1	141	4.4	0.3	40	10	0	6	D	4	1	6	55	2.8
116	28	M	N	118	90	50	63	14	102	9	E2V3M4	P	7.35	24	53	40	3.8	16780	0.7	133	3.3	0.4	21	13	4	7	D	7	8	17	38	3.4
117	77	M	G	122	40	20	27	12	97	7	E1V2M4	U	6.7	2	311	8	1.8	24000	2	139	4.7	0.5	100	1	1	1	M	10	11	38	14	18.84
118	50	M	C	114	80	50	60	16	101	15	15	A	7.1	12	79.7	34	3	21500	1	136	5.8	0.6	21	10	0	6	D	5	7	15	32	5.94
119	62	M	R	100	70	40	50	24	97	11	E3V3M5	V	7.15	18.6	61	44	1	18200	1.3	141	4.8	0.4	52	12	0	10	D	6	10	19	53	5.3
120	59	F	U	122	80	40	53	18	102	15	15	A	7.39	17.4	87	38	3.1	12700	1.2	132	3.4	0.9	21	9	0	4	D	7	7	9	29	4.1
121	29	F	U	118	90	70	77	16	101	15	15	A	7.35	20	70	38	3	16400	0.9	139	4.1	0.3	21	7	0	3	D	4	6	4	34	3.3
122	52	F	C	100	80	60	67	16	100	15	15	A	7.23	9.1	96.3	59	2.8	17400	1.1	137	3.36	0.5	40	12	0	8	D	3	7	11	22	6.8
123	80	M	R	128	80	50	60	30	98	8	E1V2M5	P	7.18	28	67	34	2.5	12600	0.7	140	3.9	0.2	60	20	9	15	D	9	10	23	80	3.9
124	88	M	R	136	NR	NR	NR	34	99	7	E1V2M4	U	7.1	20	85	36	1.95	18900	1.3	145	3.8	0.2	100	7	7	7	M	12	10	30	85	6.8
125	57	F	U	102	70	50	57	18	97	15	15	A	7.29	23	82	36	1.27	7000	1.5	133	4.5	1	21	8	0	5	D	5	8	9	48	4.4
126	60	M	C	118	90	50	63	26	100	15	15	A	7.17	18	60	33	2	18200	0.8	135	3	0.6	21	10	0	7	D	5	7	16	44	6.5
127	38	M	O	110	70	50	57	16	101	15	15	A	7.38	17.5	104	38	3	10200	0.6	136	4.5	2.8	40	7	0	3	D	3	9	4	30	3.4
128	42	M	G	116	80	60	67	18	102	15	15	A	7.25	17	60	38	2.9	15300	0.7	143	3.8	0.6	21	11	0	6	D	6	7	11	35	4.9
129	68	F	G	130	NR	NR	NR	14	100	9	E2V2M5	P	7.07	11	57	42	1.5	21000	1.9	119	5	0.7	80	7	7	7	M	8	10	31	76	14
130	48	F	O	96	100	50	67	18	102	15	15	A	7.38	19.4	60.9	37	2.5	6700	0.7	136	3.1	0.4	21	5	0	2	D	4	7	7	33	2.5
131	50	M	C	110	80	50	60	16	103	15	15	A	7.2	17	77	39	90000	19300	1.4	131	0.9	0.7	21	14	0	5	D	6	4	17	30	5.2
132	70	M	R	80	90	50	63	26	99	15	15	A	7.32	18	111	40	90000	9500	1.1	140	4.5	1.2	100	19	10	15	D	3	3	14	36	4
133	81	M	R	130	70	40	50	32	104	11	E3V3M5	V	7.12	18	209	50	45000	13500	1.7	138	2.9	2.2	100	10	10	10	M	12	8	31	56	9.6
134	80	M	N	108	70	50	57	12	102	5	E1V1M3	U	6.5	3.5	51.7	32	1.2	7300	3	114	7.8	1	60	1	1	1	M	9	14	35	40	19.76
135	60	F	U	116	80	50	60	18	99	15	15	A	7.32	19	68	30	2.8	17200	1.4	140	4.6	0.5	21	8	0	4	D	5	8	11	36	2.6
136	66	F	N	100	70	50	57	16	102	11	E3V3M5	V	7.03	19	123	39	1.21	6000	1.5	133	4.5	1	60	14	14	14	M	7	10	20	45	16.4
137	48	M	C	128	90	50	63	16	103	15	15	A	7.28	20	70	42	2.7	17000	1.1	138	4.9	0.7	21	13	0	10	D	6	7	13	42	5.1
138	60	F	O	120	70	50	57	12	100	5	E1V1M3	U	6.9	4	118	36	1.8	5700	1	127	3.2	0.5	100	3	3	3	M	8	11	28	38	12.4
139	62	F	R	110	80	50	60	20	99	15	15	A	7.42	14	85	47	45000	9600	1.1	140	3.9	1.1	60	9	0	5	D	4	3	10	23	3.8
140	56	M	N	122	90	50	63	14	100	13	E3V4M6	V	7.45	30.4	71	46	1.67	17900	0.9	142	4.8	0.5	44	9	0	6	D	4	8	11	44	4.6
141	52	F	U	96	70	40	50	18	101	13	E4V4M5	A	7.19	18	68	38	3.4	11200	1	141	5.8	0.3	21	11	0	6	D	4	8	11	48	4.8
142	55	M	C	100	90	60	70	16	100.8	15	15	A	7.48	32	76	24	68000	13210	1.7	137	3.7	1	21	8	0	5	D	2	4	7	38	7.1
143	64	M	R	120	90	60	70	24	98	15	15	A	7.32	18	209	40	61000	11000	0.9	141	4.8	0.9	50	13	3	8	D	5	3	7	36	5
144	70	M	R	132	80	40	53	24	100	15	15	A	7.3	17	143	49	29000	7500	0.9	141	4.4	2	40	16	5	12	D	7	5	12	30	8.9
145	48	M	C	108	50		23	22	98	15	15	A	7.27	8	80	16	1.5	21000	1.7	132.6	5	0.9	21	15	0	12	D	6	8	16	18	5.85
146	28	F	U	138	70	50	57	20	101	12	E3V4M5	V	7.28	17.3	87	24	74000	7430	3.6	137	5.8	0.4	40	10	6	10	M	8	8	16	37	6.63
147	38	M	N	112	90	50	63	16	99	12	E3V4M5	V	7.2	28	120	34	59000	6900	0.7	136	5.4	1.1	60	14	0	10	D	5	5	12	52	6
148	69	F	U	90	80	50	60	34	103	9	E2V3M4	P	6.9	15	67	38	1.7	21300	2	134	4.8	0.6	21	8	3	8	M	9	12	27	46	8
149	58	M	R	126	90	50	63	30	101	15	15	A	7.21	22	90	30	2.3	8700	0.8	141	3.4	0.7	50	10	0	6	D	6	7	14	38	4.2
150	38	F	G	100	70	40	50	18	100	15	15	A	7.44	21.8	75.2	35	2.9	8200	1	142	3.7	0.4	21	8	0	5	D	4	7	2	33	4.34

Sl.No	Age	Sex	Source of sepsis	PR	SBP	DBP	MAP	RR	TEMP	GCS_	GCS	AVPU	pH	HCO3	PaO2	PCV	PLT	WBC	SCr	SNa	SK	bilirubin	FiO2	Hospitalization	Ventilator	ICU	DIS/DECEASED	MEWS	SOFA	APACHE II	PCO2	LACTATE
151	42	F	G	110	70	50	57	20	101	15	15	A	7.33	12.7	80.6	19	80000	7600	0.9	125	2.9	0.7	21	9	0	6	D	5	3	12	44	2.9
152	45	M	N	100	100	60	73	14	102	9	E2V2M5	P	7.48	27	116	40	4	11700	0.6	130	4	0.4	60	10	3	7	D	5	9	11	28	4.6
153	54	M	R	118	100	50	67	26	101	15	15	A	7.15	30	67	34	2.6	12700	0.7	141	3.9	0.2	60	9	2	6	D	5	7	12	76	3.4
154	55	M	O	134	80	50	60	20	103	15	15	A	7.2	16.1	48.5	40	3.5	22000	0.9	132	3	0.6	21	15	0	9	D	8	8	20	41	7.05
155	49	F	U	94	60	40	47	18	98.7	12	E3V4M5	V	7.44	19	73	29	1.1	26200	0.8	145	4.6	0.5	21	13	0	7	D	5	9	13		3.4
156	55	F	R	116	90	50	63	30	98	15	15	A	7.1	25	108	34	1.16	23000	0.6	147	4.9	0.5	60	12	0	7	D	6	7	16	62	4.7
157	32	M	G	108	80	50	60	18	99	15	15	A	7.2	19	90	38	2.3	16000	1.7	133	3.2	0.7	21	5	0	1	D	4	8	9	40	2.8
158	29	M	O	112	90	50	63	18	101	15	15	A	7.21	18	88	42	3.1	17000	1.1	135	3.6	0.8	21	7	0	1	D	4	7	8	35	2.4
159	48	M	C	104	70	40	50	20	99.9	15	15	A	7.23	13	67.7	30	2.1	9000	0.8	138	3.7	0.9	21	12	0	4	D	5	7	8	30	5
160	36	M	R	110	90	50	63	24	102	15	15	A	7.2	16	56	28	4.1	14300	0.8	135	3.8	0.6	21	8	0	4	D	6	7	13	40	4
161	63	M	U	110	80	50	60	22	98	11	E3V3M5	V	6.9	10	46.7	40	3.2	11200	1.4	130	3.74	0.6	21	6	4	6	M	6	11	19	45	12.88
162	57	M	G	124	80	60	67	18	100	15	15	A	7.31	21.2	77.9	42	3.4	9600	0.9	138	3.65	0.9	21	8	0	6	D	5	7	9	37	3
163	42	M	O	114	90	40	57	22	101	15	15	A	7.12	14	78	35	1.4	7800	1	137	4.2	1	52	10	0	7	D	5	7	10	28	6
164	58	M	N	114	100	50	67	20	103	6	E1V2M3	U	7.25	20	87.8	38	2	18900	1	140	4	0.6	44	14	5	10	D	9	10	22	46	6.55
165	40	F	G	110	60	40	47	28	103	13	E3V4M6	V	6.92	5	80	40	2.3	15700	2.3	129	5.7	0.8	52	12	3	8	D	9	10	25	37	7.63
166	73	M	U	114	70	50	57	12	98	5	E1V2M2	U	6.95	3.9	123	27	1.24	7000	3	118	5.2	0.7	100	3	3	3	M	8	13	35	18	17
167	52	M	C	100	90	60	70	17	101	15	15	A	7.34	15	78	37	2.2	6800	1	136	3.4	0.8	21	9	0	3	D	2	7	3	34	3
168	59	F	U	148	NR	NR		26	99	9	E2V3M4	P	6.9	9	96	25	1.45	10230	1.6	129	5.1	0.3	100	5	5	5	M	7	10	31	30	11
169	48	F	C	90	80	50	60	24	100	11	E3V3M5	V	7.18	8	86	32	2.5	18600	2.8	130	6	1	35	14	0	7	D	5	11	25	40	7.6
170	60	F	G	60	40	20	27	14	103	4	E1V2M1	U	6.8	7	42	23	88000	19300	2	132	5.4	0.3	100	3	3	3	M	8	10	37	17	15.7
171	42	F	U	90	80	60	67	16	98	15	15	A	7.33	17	70	30	1.9	9200	1.5	132	6	0.8	21	14	0	10	D	1	8	8	30	6.2
172	50	M	R	110	90	50	63	26	99	15	15	A	7.16	12	54	40	3	6700	3	133	4.6	0.6	60	11	0	6	D	4	9	14	25	4.8
173	32	F	R	116	100	60	73	32	103	15	15	A	7	17	60.2	30	1.4	6000	2.2	118	5.8	0.5	70	13	0	9	D	8	8	20	43	5.6
174	38	M	G	98	100	50	67	18	101	15	15	A	7.42	22	70	36	2.9	9600	0.8	140	3.1	0.6	21	7	0	5	D	2	7	4	37	3
175	65	F	U	124	80	50	60	26	103	13	13	V	6.8	8	77.8	42	1.5	16850	2.9	141	5.7	1.3	70	4	4	4	M	9	9	31	40	18
176	64	M	C	98	100	70	80	22	99	15	15	A	7.48	28	78	28	1.45	13500	1.6	122	4.7	0.4	21	10	0	6	D	3	7	9	36	5.83
177	66	M	N	108	60	40	47	20	101	9	E2V3M4	P	7.1	18	62	36	2.1	14200	0.8	118	3.8	0.3	35	16	5	9	D	7	10	23	58	8
178	27	F	C	118	80	60	67	26	101.2	15	15	A	7.37	9.9	101.1	37	3	13400	1.4	138	4.83	0.6	43	10	0	8	D	6	8	13	18	3.64
179	70	M	U	102	70	40	50	20	100	15	15	A	7.1	11	78.4	29	2.8	10800	1.8	135	4.2	0.8	21	11	0	9	D	4	8	15	23	4.9
180	56	F	R	120	70	50	57	28	99	15	15	A	7.32	19	66	28	2	14500	1.2	138	3.7	0.5	21	8	0	6	D	7	7	13	46	4
181	60	F	N	78	60	40	47	14	102	8	E2V2M4	P	7.5	20	67	44	3.2	7890	0.9	108	1.2	1	100	6	6	6	M	7	10	28	74	7.2
182	44	M	C	112	70	40	50	18	101	15	15	A	7.22	18	70	38	3	21000	1.4	139	4.1	0.6	21	16	0	9	D	6	8	10	32	5.1
183	52	F	N	90	90	50	63	24	102	9	E2V3M4	P	7.17	12.5	80	39	4	16500	1.2	128.2	3.64	0.7	36	17	3	12	D	7	10	17	35	5.07
184	70	M	U	140	70	40	50	30	98	4	E1V2M1	U	6.88	8.9	21	16	2.5	25670	2.3	138.8	3.31	1	60	3	3	3	M	12	16	38	48	14.22
185	74	F	U	128	80	50	60	28	100	9	E2V3M4	P	6.9	3.9	48.1	30	1.1	22340	4.3	129.2	8.05	0.9	100	2	2	2	M	8	14	36	17	16
186	54	M	R	102	90	50	63	34	100.8	12	E4V3M5	A	7.007	20	76.2	22	2.7	15600	1.1	133	4	0.7	56	10	3	8	D	4	9	17	54	6
187	66	F	U	98	90	60	70	30	99.8	12	E4V3M5	A	7.01	3.3	64	30	3.3	18200	2	130	5	0.9	21	16	0	10	D	4	11	18	32	5.6
188	58	F	C	100	70	50	57	18	102	15	15	A	7.3	18	70	31	1.8	22000	1.6	131	4.9	0.8	21	24	0	7	D	6	8	13	31	7.1
189	36	F	G	108	80	40	53	20	98	15	15	A	7.19	17	60	42	1	19800	2	146.2	3.8	1	21	15	0	12	D	4	9	12	45	6
190	40	M	C	108	90	60	70	16	101.6	15	15	A	7.1	13	58	37	3.1	21500	2	137	5.1	0.6	40	16	0	8	D	5	9	13	37	8
191	47	M	O	110	90	50	63	20	101	15	15	A	7.52	22	68	28	3.6	8900	0.9	140	4.2	0.7	21	10	0	6	D	3	7	14	22	4.1
192	60	F	G	60	40	20	27	20	103	4	E1V2M1	U	7	9.5	40	23	89000	21000	2	134	5.1	0.6	100	6	6	6	M	8	15	39	14	14.3
193	64	M	O	110	90	60	70	28	101	15	15	A	6.9	5	77	34	3.3	15700	1.6	140	4.3	1.4	43	10	6	10	M	4	6	12	28	9.4
194	55	F	C	122	NR	NR	NR	24	97	12	E3V4M5	V	7.12	11.2	93.8	22	1.5	22000	5	144.7	6.9	1.2	60	16	2	12	D	5	12	29	34	8
195	46	M	C	112	50		23	22	99	15	15	A	7.29	8	86.8	16	1.5	19000	1.5	132.6	5.2	0.9	21	16	0	13	D	6	5	17	17	6.43
196	58	F	U	124	70	50	57	20	103	12	E3V4M5	V	7.21	17.9	67	41	2.3	6000	4.5	134	6.6	0.7	21	19	3	15	D	9	9	29	40	7.8
197	48	M	U	96	80	50	60	18	101.2	15	15	A	7.28	14.2	110	33	3.1	17200	3	138.4	4.9	0.9	36	14	0	10	D	3	9	10	33	4.2
198	70	F	N	110	70	40	50	20	100	8	E2V2M4	P	7.53	19.3	99.7	27	2.5	5000	1.4	128.9	3.2	1	100	18	5	12	D	7	11	26	20	7.8
199	84	M	G	130	70	50	57	18	99	10	E2V3M5	V	6.9	11	102	39	4	17600	3	118	2.3	0.7	60	2	2	2	M	8	11	32	28	14
200	85	F	R	106	90	50	70	28	100	12	E3V4M5	V	7.12	20	68.2	26	2.4	8000	2	132	4.5	1.4	43	11	0	7	D	5	8	25	27	3.86
201	75	F	U	102	NR	NR	NR	12	102	7	E1V2M4	P	6.8	10	121	32	3	4500	0.7	127	3	0.8	100	4	4	4	M	5	9	30	62	11