



Integrating frailty and acute illness severity scoring for risk stratification in acute geriatric care: Review and practical guide

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Abstract

Managing acute illness in older populations remains a major challenge because of the complex interplay between multimorbidity and reduced physiological reserve. While conventional acute severity scores (*e.g.*, Sequential Organ Failure Assessment, Acute Physiology and Chronic Health Evaluation, and National Early Warning Score) track acute physiological derangements, they often fail to capture the biological heterogeneity seen in geriatric patients. Frailty provides a vital window into the baseline vulnerability of a patient, yet it remains underutilized in acute triage despite its established prognostic value. Current evidence demonstrates that integrating frailty measures, specifically the Clinical Frailty Scale (CFS), with existing severity scores significantly improves mortality prediction and risk stratification in emergency and critical care. This mini-review explores age-related physiological changes as well as the prognostic limitations of standard severity scores while evaluating their integration with validated frailty tools. To bridge current clinical gaps, we propose a practical 4-step clinical triage algorithm integrating a baseline "Two-Week Rule" CFS with acute severity scores *via* a synergistic 2 × 2 risk stratification matrix. By identifying distinct clinical phenotypes, this framework guides precise, person-centered triage, ensuring that treatment escalation and critical care interventions remain medically proportionate to an older adult's attainable functional recovery.

Key Words: Acute geriatric care; Frailty; Clinical Frailty Scale; Sequential Organ Failure Assessment; Acute Physiology and Chronic Health Evaluation; National Early Warning Score; Risk stratification; Triage; Emergency department; Critical care

Core Tip: Conventional acute illness severity scoring often neglects baseline frailty, leading to inaccurate risk stratification in acute and critical care settings. This mini-review proposes a novel framework comprising a synergistic 2×2 risk stratification matrix and a 4-step clinical triage algorithm that integrates acute illness severity scores with the “Two-Week Rule” for baseline Clinical Frailty Scale assessment. By identifying distinct clinical phenotypes, specifically masked resilience and masked risk, clinicians can move beyond one-dimensional triage to make precise, person-centered decisions regarding treatment escalation and goals-of-care planning. This ensures acute geriatric care remains medically proportionate to attainable functional recovery rather than simple physiological stabilization.

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INTRODUCTION

The rapid aging of the global population presents unique clinical challenges for acute care systems. According to the United Nations World Population Prospects 2024, the proportion of the population aged 65 and older is projected to double from 10.3% in 2024 to 20.7% by 2074. This shift means older adults will eventually outnumber children under 18, marking a permanent transition to an aging society[1].

Across the acute care continuum, from emergency departments (EDs) to general acute wards and intensive care units (ICUs), this demographic shift exposes the limitations of traditional risk stratification. For decades, clinicians have relied on scoring systems like the Sequential Organ Failure Assessment (SOFA) or the Acute Physiology and Chronic Health Evaluation (APACHE) to measure the severity of an acute insult. While these tools work well for general populations, they often miss the specific complexities found in geriatric care[2,3]. In older adults, clinical outcomes do not depend solely on the severity of the acute illness (physiological derangement)[4]. Instead, they depend critically on the host's remaining biological capital (physiological reserve)[5]. This reserve correlates imperfectly with chronological age and is best conceptualized through the lens of frailty[6].

This mini-review bridges the gap between traditional physiological scoring and geriatric complexity. It explores the physiological changes that occur with age to explain why it is essential to assess both acute illness severity and frailty status together. We review key acute severity tools [e.g., SOFA, APACHE, National Early Warning Score (NEWS)] alongside validated frailty instruments [specifically the Clinical Frailty Scale (CFS) and Frailty Index (FI)], evaluating the evidence for their integration. Overall, this paper provides clinicians with a practical guide to implement frailty-integrated risk stratification. This represents a vital step toward precise, evidence-based, and person-centered care for a rapidly aging population.

LITERATURE SEARCH STRATEGY AND SELECTION CRITERIA

We conducted a thorough literature search across PubMed/MEDLINE, Scopus, and the Cochrane Database of Systematic Reviews for articles published from January 2000 through April 2026. Our search strategy combined MeSH with free-text keywords focusing on three main areas: (1) Frailty (“Frailty”, “Clinical Frailty Scale”); (2) Acute illness severity (“SOFA”, “APACHE”, “SAPS II”, “NEWS”); and (3) Geriatric populations (“geriatrics”, “older adults”).

We included studies evaluating older adults aged 65 and above within critical care or ED settings. We also considered general adult cohorts if data for age and frailty were clearly isolated. Furthermore, each study was required to assess the prognostic accuracy of acute illness severity scoring systems, either independently or integrated with validated frailty tools. We excluded pediatric groups and populations undergoing purely elective surgery. Studies using subjective or non-standardized frailty assessments were also removed. Finally, we did not include non-English publications or case reports that were not peer-reviewed.

THE PHYSIOLOGY OF AGING AND HOMEOSTATIC RESERVE

Aging is a heterogeneous, progressive process characterized by the accumulation of molecular and cellular damage[7]. This leads to a gradual decline in homeostatic reserve, which is the extra capacity organ systems maintain to function above their basal level during times of stress[8]. In youth, this safety margin is broad. For example, a healthy young adult can increase cardiac output by approximately 3.5 times the resting level during physical exertion[9].

Cellular and molecular mechanisms

With aging, physiological reserve diminishes through a phenomenon termed homeostenosis[10]. This concept describes the progressive narrowing of the homeostatic range within which an individual can maintain their internal stability.

Importantly, this erosion of reserve is often clinically silent. While basal functions (*e.g.*, resting heart rate or serum creatinine) may stay within normal limits, the capacity (physiological reserve) to respond to increased physiological demands (*e.g.*, sepsis, trauma, or surgery) is often severely compromised. This places the older adult on a “compensatory cliff” where even minor stressors can cause a rapid collapse across multiple systems[8,10,11].

At the microscopic level, aging is driven by a confluence of mechanisms including genomic instability, telomere attrition, epigenetic alterations, and the loss of proteostasis[7]. Mitochondrial dysfunction leads to bioenergetic failure, which deprives cells of the critical energy reserves needed to withstand acute stress[12]. Instead of immediate recovery, these stressed cells often transition into a state of permanent arrest known as senescence. The accumulation of senescent cells contributes to a “senescence-associated secretory phenotype”. This releases pro-inflammatory cytokines such as interleukin (IL)-6 and tumor necrosis factor-alpha (TNF- α)[13]. This results in “inflammaging”, a state of chronic, low-grade systemic inflammation that alters the baseline of the immune system and leads to dysregulated responses during acute infection[14]. Finally, oxidative stress and free radicals react with DNA and lipids, leading to cumulative cellular damage that eventually manifests as organ dysfunction[15].

Organ system modifications

The clinical manifestation of these cellular changes is a reduction in the functional reserve of every major organ system.

Cardiovascular system: The aging heart and vasculature undergo structural remodeling characterized by vascular stiffening and myocardial hypertrophy. The aorta and large arteries lose elasticity over time. This increases afterload and pulse wave velocity, which often leads to isolated systolic hypertension[16]. At the myocardial level, prolonged contraction times and delayed relaxation result in diastolic dysfunction. Consequently, the heart relies heavily on the atrial contraction (“atrial kick”) for ventricular filling. This makes the patient very sensitive to changes in fluid status, as the stiff ventricle lacks the compliance to handle both preload depletion and fluid overload[17]. A hallmark of cardiac senescence is a blunted β -adrenergic response. This limits the maximum heart rate [clinically estimated by the Tanaka formula as $208 - (0.7 \times \text{age})$] and reduces the response to circulating catecholamines[16-18]. In the context of acute shock, this prevents the older patient from mounting a compensatory tachycardia, the primary mechanism younger adults use to maintain cardiac output[16].

Respiratory system: Aging causes the chest wall to stiffen due to calcification of costal cartilages, while the lung parenchyma loses its natural elastic recoil[19]. This leads to a state historically termed the “senile lung”, which is characterized by a homogeneous enlargement of alveolar spaces without the fibrosis or alveolar wall destruction seen in clinical chronic obstructive pulmonary disease (COPD)[20]. The respiratory muscle strength also declines. This significantly increases the work of breathing as the patient must overcome both reduced thoracic compliance and the mechanical disadvantage of air trapping[21]. From an acute care perspective, the most critical change is the drop in chemoreceptor sensitivity. The central and peripheral chemoreceptor drive in response to hypoxia or hypercapnia falls by about half in older adults when compared to younger controls[22]. Because of this blunted response, an older patient in acute respiratory failure may not show signs of rapid breathing or “air hunger” typically seen in younger patients until they are on the very edge of physiological collapse[21,23].

Renal system: The aging kidney undergoes significant structural attrition, with renal mass dropping by roughly 30% by the ninth decade, primarily affecting the renal cortex[8,24]. This is accompanied by a progressive loss of functional glomeruli, decreasing from roughly one million before age 40 to about 700,000 by age 65[8]. Consequently, the glomerular filtration rate in healthy adults declines by an average rate of 0.37 to 1.07 mL/minute/1.73 m² each year[25]. This steady erosion of the “renal reserve” compromises the kidney’s capacity to concentrate urine, conserve sodium, and excrete acid. Such changes make older patients highly vulnerable to sudden dehydration, electrolyte disturbances (notably hyponatremia and hyperkalemia), and metabolic acidosis during acute illness[12]. Moreover, sarcopenia often masks this functional decline. Because the reduced skeletal muscle mass lowers baseline creatinine production, serum creatinine levels can appear falsely reassuring while hiding significant renal impairment[26]. Relying on creatinine alone carries a high risk of drug toxicity from the over-prescription or under-adjustment of renally cleared medications, such as aminoglycosides, digoxin, or low-molecular-weight heparin[12].

Neurological system: Age-related neurological changes create a state of high vulnerability to acute brain failure, clinically manifesting as delirium in the older population[27]. This vulnerability stems from increased blood-brain barrier (BBB) permeability and a diminished neurotransmitter reserve[28]. With aging, the structural integrity of the BBB declines, allowing peripheral inflammatory processes (*e.g.*, from infections or surgery) to activate brain parenchymal cells. Once activated, these cells express inflammatory mediators (*e.g.*, C-reactive protein, IL-6, TNF- α , and IL-8)[29] within the central nervous system, leading to profound neuronal and synaptic dysfunction that results in delirium[30]. This profound neuroinflammation is compounded by an age-related “cholinergic deficit” (depleted acetylcholine), which strips the brain of the neurochemical buffer needed to withstand physiological stress[28]. Consequently, delirium presenting as acute confusion or altered consciousness is frequently the sole presenting symptom of a systemic illness in older adults[27].

Immune system: The aging immune system undergoes a profound functional decline termed immunosenescence. This is paradoxically coupled with a state of chronic, low-grade systemic inflammation known as inflammaging[31]. Adaptive immunity is severely compromised because of thymic involution, which is the steady shrinking of the thymus. This drives a critical shift from naive to memory T-cells. This depletion of the naive T-cell significantly impairs the host’s ability to recognize and react effectively to new pathogens[31]. Simultaneously, the innate immune system becomes

dysregulated. While absolute white blood cell counts may remain stable, the functional efficacy (specifically the chemotaxis, phagocytosis, and bactericidal activity) of neutrophils, macrophages, and natural killer cells are significantly diminished[32,33]. Clinically, this “double hit” to both the adaptive and innate responses leave older adults highly susceptible to rapid, overwhelming infections. Because classical signs of infection (*e.g.*, fever and pronounced leukocytosis) are often blunted by this immune dysfunction, clinicians often detect the illness too late. This delay leads to prolonged courses of sepsis and much higher mortality rates in the acute care setting[34].

Differences between younger and older patients in acute illness presentation

The physiological decline of aging described in the preceding sections alters how acute illness presents in the older compared to younger cohorts. While robust young adults mount a “classical” stress response (high fever, tachycardia, localized pain, and leukocytosis), older adults often fail to manifest these dramatic signs due to reduced physiological reserves and multiple comorbidities[35,36].

Rather than showing specific signs related to a single organ, acute illness in the frail older adults typically manifests atypically as non-specific “geriatric syndromes”[35]. These include: (1) Delirium (altered mental state): An acute change in cognition is often the sole presenting sign of an underlying acute stressor (*e.g.*, pneumonia, urinary tract infection, or myocardial infarction); (2) Falls: What looks like a simple “mechanical fall” in a frail older adult is often a syncopal or pre-syncopal event driven by an occult acute illness; and (3) Functional decline: An acute loss of independence, characterized by sudden immobility, an inability to eat or drink, or new-onset urinary incontinence.

These atypical presentations frequently lead to delayed or missed diagnoses. This increases the risk of complications, prolonged hospitalizations, and higher mortality in the acute care setting[35]. Furthermore, since traditional triage systems and early warning scores rely heavily on classical changes in vital signs, this blunted physiological response often leads to dangerous under-triage and delayed resuscitation[37].

Technological advancements and increased longevity

Modern medical breakthroughs have fundamentally changed the way we manage acute illness. Innovative treatments can now override natural physiological limits, allowing older adults with minimal physiological reserve to survive acute insults that once would have been fatal[38]. Procedures such as percutaneous coronary intervention, transcatheter aortic valve replacement, and mechanical thrombectomy have shown great success even in nonagenarians. This evidence suggests that chronological age alone is no longer a valid reason to disqualify an older adult from receiving these life-saving interventions[39,40]. Diseases that were once considered terminal are now frequently managed as chronic conditions[41].

Yet, these advancements have created a new clinical reality: The survivor who lives with multiple chronic illnesses and extreme frailty. While medical technology can support failing organs through dialysis or mechanical ventilation, it cannot fix the fundamental loss of physiological reserve. As a result, ICUs are increasingly occupied by patients who survive the initial medical rescue but lack the reserve needed to regain their independence. This often leads to prolonged dependency, “chronic critical illness”, and complex ethical questions about the true goals of care[42]. This modern paradox highlights the urgent need for assessment tools that can distinguish between a patient who will truly benefit from aggressive technological rescue and one for whom such interventions might only lead to a bridge to nowhere.

ACUTE ILLNESS SEVERITY SCORES: FORMULATIONS AND LIMITATIONS IN THE GERIATRIC POPULATION

Acute illness severity scores are mathematical models that measure how far a patient has drifted from their normal physiological balance. While researchers originally designed these for population-level benchmarking and quality audits, clinicians now frequently employ them at the bedside for individual risk stratification and ED triage. However, applying these standardized scoring systems in the geriatric population has clear limitations. This is largely because these models track physiological derangements that aging organ systems often fail to mount. Using these scores alone is simply not enough. It frequently leads to inaccurate risk stratification and dangerous under-triaging. For a frail patient, this mismatch can delay life-saving interventions until the window for recovery has closed.

The SOFA and quick SOFA

The SOFA score was first developed by the Working Group on Sepsis-Related Problems of the European Society of Intensive Care Medicine during a consensus meeting in 1994 and subsequently validated by Vincent *et al*[43] in 1996. Although originally known as the “Sepsis-Related” Organ Failure Assessment, it was later renamed the “Sequential” Organ Failure Assessment to reflect its applicability to critically ill patients without sepsis[44]. Its original intent was to describe and monitor the severity of organ dysfunction over time in critically ill patients, quantifying morbidity rather than predicting mortality upon admission[43].

Despite these original intentions, the SOFA score and its abbreviated bedside counterpart, the quick SOFA (qSOFA), have gained global recognition as the primary criteria for defining organ dysfunction in sepsis (Sepsis-3 definitions)[45, 46]. Clinicians use an acute increase of 2 or more SOFA points to stratify severity of sepsis, as this change indicates a significantly higher risk of in-hospital mortality[47].

The SOFA score tracks six distinct organ systems by assigning a value between 0 for normal function and 4 for severe failure, leading to a total score from 0 to 24[43]. These components include: (1) Respiratory function: Defined by the $\text{PaO}_2/\text{FiO}_2$ ratio, which captures the efficiency of gas exchange with scores ranging from 0 for a ratio above 400 mmHg to 4 when it drops below 100 mmHg with respiratory support; (2) Coagulation: Measured by platelet count, where thrombocytopenia serves as a sensitive indicator for disseminated intravascular coagulation and worsening sepsis; (3) Hepatic function: Measured by serum bilirubin levels, reflecting hepatic clearance function; (4) Cardiovascular function: Assessed by mean arterial pressure (MAP) and the requirement for vasopressors, a component that incorporates therapeutic interventions (dosage of vasopressors) rather than just physiological variables; (5) Central nervous system: Assessed by the Glasgow Coma Scale (GCS), which is often the most challenging component to score accurately in sedated patients; and (6) Renal function: Measured by serum creatinine or urine output.

To facilitate the rapid identification of patients with suspected infection at high risk of mortality outside the ICU (*e.g.*, in the ED or general wards), the Sepsis-3 task force derived the qSOFA score. This bedside tool uses three simple clinical criteria, with one point assigned for each[45]. These include a respiratory rate of 22 breaths per minute or higher, altered mental status with a GCS below 15, and a systolic blood pressure (SBP) of 100 mmHg or less.

Prognostic performance of SOFA and qSOFA in the general adult population: Validation studies have consistently established that the absolute SOFA score on admission, the delta SOFA (the change in SOFA score over the first 48 hours), and the qSOFA score are powerful predictors of mortality. Specifically: (1) Inside the ICU: Ferreira *et al*[48] demonstrated that an initial SOFA score above 11 is associated with a mortality rate over 90%, while an increasing delta SOFA score significantly correlates with poor clinical outcomes regardless of the baseline score. Furthermore, in the landmark Sepsis-3 retrospective cohort study, Seymour *et al*[45] revealed that the SOFA score was very accurate in predicting mortality within the ICU, yielding an area under the receiver operating characteristic (AUROC) of 0.74; and (2) Outside the ICU: For patients in the ED or general wards, the qSOFA score showed excellent prognostic accuracy (AUROC = 0.81). This statistically matched the performance of the full SOFA score (AUROC = 0.79). Most importantly, a qSOFA score of 2 or higher was associated with a 3- to 14-fold increase in hospital mortality, highlighting how effective the tool is for early sepsis triage[45].

Prognostic failure of SOFA and qSOFA in the geriatric population: While SOFA and qSOFA perform exceptionally well in general populations, this robust performance does not universally translate to older adults. This is evidenced by: (1) Limitations of the full SOFA score: A prospective cohort study by Falsetti *et al*[49] focused on older patients with suspected infection admitted from the ED to internal medicine step-down units highlighted that the ability of the SOFA score to predict mortality falls sharply, with an AUROC of only 0.686. This drop in accuracy highlights a critical challenge in geriatric care. The SOFA score inherently assumes a baseline organ dysfunction score of zero and was designed to detect acute physiological changes. Yet, many older adults live with chronic organ dysfunction and have a normal baseline state that already scores a SOFA 1 or 2 (*e.g.*, due to chronic kidney disease or cognitive impairment). Consequently, the score fails to distinguish between an older patient who is acutely deteriorating from sepsis and one who is simply frail with a high burden of chronic disease; and (2) Limitations of the abbreviated qSOFA score: A comparative study by Ishikawa *et al*[50] highlighted a sharp decline in how well qSOFA predicts hospital mortality as patients get older. The sensitivity of a qSOFA score ≥ 2 drops from 80% in younger adults (< 75 years) to just 50% in the older adults (≥ 75 years). This was accompanied by a significant reduction in the AUROC from 0.85 to 0.61. Similarly, Bastoni *et al*[51] evaluating the Sepsis-3 criteria specifically within acute geriatric wards (mean age > 83 years) confirmed that qSOFA has a weak prognostic capacity in frail groups, yielding an AUROC of only 0.67 for hospital mortality.

Therefore, relying solely on qSOFA can create a false sense of security that often leads to delayed recognition of sepsis in patients at high risk of mortality. This failure stems directly from the altered baseline physiology of the aging population: (1) Masked hypotension: Older adults frequently have baseline chronic hypertension. If a patient's baseline SBP is 160 mmHg, a drop to 105 mmHg will not trigger a point for qSOFA despite a significant 35% decrease in perfusion pressure. This demonstrates that the fixed-point threshold of $\text{SBP} \leq 100$ mmHg in qSOFA fails to catch profound relative hypotension; and (2) Masked delirium: The high background prevalence of cognitive impairment (*e.g.*, dementia) can lead to false positives if altered mental status is not strictly defined as an acute change. On the other hand, spotting delirium superimposed on dementia is exceptionally difficult, as subtle acute changes in cognition are often ignored or mistaken for the patient's usual baseline behavior.

NEWS2

To address the issue of clinicians missing early signs of clinical deterioration on general hospital wards, the Royal College of Physicians in the United Kingdom introduced the NEWS in 2012, later updating it to NEWS2 in 2017. While the SOFA score relies on laboratory values to measure organ dysfunction, NEWS2 works as a bedside track-and-trigger system derived entirely from routine vital signs[52].

The NEWS2 combines six physiological parameters, assigning a score from 0 to 3 for each based on their deviation from normal ranges. These include: (1) Respiratory rate; (2) Oxygen saturation: NEWS2 uniquely incorporates two different scales. Scale 1 is for the general population, while Scale 2 is specifically for patients with hypercapnic respiratory failure (*e.g.*, COPD); (3) SBP; (4) Pulse rate; (5) Level of consciousness: Assessed using the Alert, Confusion, Voice, Pain, Unresponsive scale, prioritizing new-onset confusion; and (6) Temperature.

Prognostic performance of NEWS2 in the general adult population: Recognised for its practicality and extensive validation, NEWS2 provides a "common language" for clinical deterioration and is used as a standard bedside triage tool worldwide[53]. Verma *et al*[54] found that NEWS2 significantly outperforms qSOFA in predicting mortality for sepsis patients in the ED. Furthermore, a meta-analysis by Wei *et al*[55] evaluating prehospital and ED settings confirmed that

the tool accurately identifies patients at risk for early mortality, with an excellent pooled AUROC of 0.88. In practice, a high combined NEWS2 of 5 or more, or a “red score” for any individual parameter, acts as a critical threshold to trigger escalation of care and mandate urgent clinician review[52,55].

Prognostic failure of NEWS2 in the geriatric population: Despite its success in general population, the “one-size-fits-all” approach of NEWS2 often fails to account for the specific vulnerabilities of the older adult[56]. Rønningen *et al*[57] highlights this reduced prognostic performance. In frail patients with coronavirus disease 2019, the AUROC for predicting mortality dropped to 0.61 compared to 0.73 in non-frail patients. Sensitivity also dropped from 86% to 61%. Similarly, Kemp *et al*[58] found that NEWS2 performed poorly in predicting hospital admissions and 30-day revisits. These statistics suggest that the NEWS2 cannot reliably distinguish between an acute deterioration and the chronically altered baselines often seen in frailty.

This failure stems from three physiological traps: (1) Chronic physiological derangements and alarm fatigue: Frail patients with multimorbidity often have vital signs that are always outside the normal range (*e.g.*, chronic hypoxia, atrial fibrillation, or baseline cognitive impairment). These patients can easily generate a “stable” baseline NEWS2 score of 4 or 5. This leads to constant alerts for stable patients, resulting in alarm fatigue. Eventually, clinicians become desensitized and this makes it harder to detect truly acute event[56]; (2) Immunosenescence: Because the febrile response is often blunted, up to 30% of older adults might score a zero for temperature even during severe sepsis[59]; and (3) Blunted compensatory responses: Chronotropic incompetence and the widespread use of rate-limiting medications (*e.g.*, beta-blockers) often mask compensatory tachycardia, keeping the heart rate below critical trigger thresholds even during shock. In the end, relying on the rigid thresholds of NEWS2 leads to the over-triage of stable chronic frailty and the under-triage of atypical, acute illness in the older adults.

APACHE

The APACHE scoring system, first published by Knaus *et al*[60] in 1981, offers a comprehensive evaluation of illness severity for patients admitted to the ICU. APACHE II (1985) remains the most widely used because it is practical, while APACHE IV (2006) utilizes a more complex variable set and larger database to improve its calibration[60,61].

Both versions calculate a score based on the worst physiological values recorded during the first 24 hours of ICU admission. This total score is derived from three distinct domains[60,61]: (1) Acute physiology score: A weighted sum of multiple physiological variables (12 in APACHE II; 17 in APACHE IV) including heart rate, MAP, temperature, respiratory rate, oxygenation, and laboratory values such as arterial pH and creatinine; (2) Age points: Categorical points that increase with chronological age to reflect diminished physiological reserve; and (3) Chronic health evaluation: Points assigned for pre-existing severe organ failure (*e.g.*, hepatic, cardiovascular, or renal) or an immunocompromised state.

Prognostic performance of APACHE in the general adult population: In general critical care, APACHE scores demonstrate excellent performance in predicting hospital mortality. A large-scale multi-center study by Rowan *et al*[62] found that APACHE II yielded a robust AUROC of 0.83 for this outcome, while Zimmerman *et al*[61] demonstrated that the more modern APACHE IV achieved an AUROC of 0.88. Furthermore, dynamic evaluations (especially the day-3 APACHE II score) serve as optimal prognostic biomarkers, with a threshold greater than 17 defining a high mortality risk [63]. Given this accuracy and clinical utility, these tools are widely considered the benchmark for standardizing patient severity in clinical research and evaluating ICU performance.

Prognostic failure of APACHE in the geriatric population: Even though it includes specific “Age points”, APACHE scores often struggle to predict the outcome for the oldest-old accurately. The main issue is that APACHE treats chronological age as a linear risk factor. This creates a prognostic mismatch where mortality risk is overestimated for fit older adults but underestimated for those who are biologically vulnerable. This failure is driven by the following factors: (1) Fixed age weighting and poor calibration: APACHE II assigns the exact same six age points to a healthy 80-year-old as it does to an 80-year-old who is bed bound and frail. Because it cannot account for the biological heterogeneity seen in frailty, this rigid weighting leads to poor mathematical calibration. Markgraf *et al*[64] demonstrated that the tool significantly underestimates mortality in geriatric cohorts, with a standardized mortality ratio (SMR) of 1.17. Bagshaw *et al*[65] confirmed this trend in the oldest-old with an SMR of 1.28. Because of this, a landmark multi-center study by Flaatten *et al*[66] proved that baseline frailty is a much better predictor of survival than chronological age alone; and (2) Over-reliance on acute physiology: APACHE prioritizes acute derangements rather than baseline vulnerability, with acute laboratory values driving nearly 68% of its predictive power compared to only 8.4% for comorbidities[67]. Because frail older adults often exhibit blunted physiological responses to acute illness (*e.g.*, severe pneumonia), these lab-heavy models lose their predictive accuracy in patients 75 and older[68]. Ultimately, while these standardized scoring systems effectively capture the acute physiological insult, they fundamentally fail to account for the older patient’s depleted functional reserve.

FRAILITY ASSESSMENT TOOLS: QUANTIFYING BASELINE RESERVE IN THE GERIATRIC POPULATION

If acute illness severity scores measure the physiological “load” placed on a patient, then frailty tools measure the “chassis” bearing that load. Frailty is conceptually defined as a multidimensional state of increased vulnerability to stressors due to decreased homeostatic reserves (Figure 1)[5,6,69]. In medical literature, we generally define frailty through two main models: (1) Frailty phenotype model (developed by Fried *et al*[69]); and (2) Cumulative deficit model

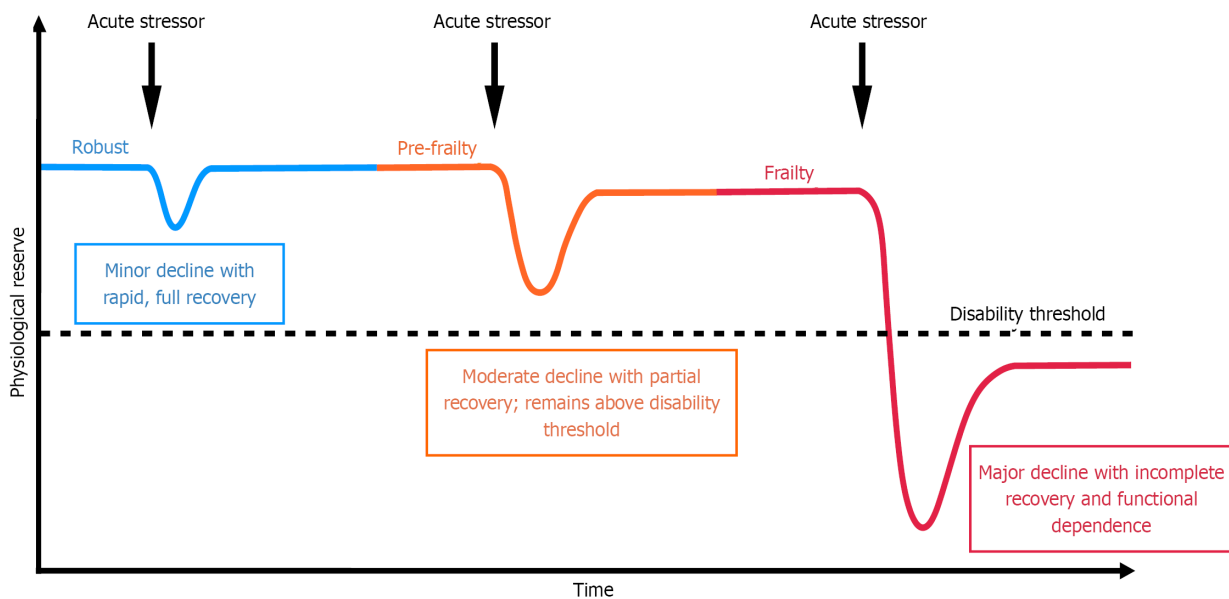


Figure 1 Comparison of functional recovery trajectories following an acute physiological stressor[5]. A: Robust phenotype (blue line) with high physiological reserve experiences a transient decline and a rapid, full recovery to baseline; B: Pre-frailty phenotype (orange line) demonstrating reduced reserve with partial recovery that remains above the disability threshold; C: Frailty phenotype (red line) showing a severe, disproportionate decline following an acute stressor (arrow) with a failure to recover above the disability threshold (dotted line), resulting in new-onset functional dependence. Citation: Dent E, Morley JE, Cruz-Jentoft AJ, Woodhouse L, Rodríguez-Mañas L, Fried LP, Woo J, Aprahamian I, Sanford A, Lundy J, Landi F, Beilby J, Martin FC, Bauer JM, Ferrucci L, Merchant RA, Dong B, Arai H, Hoogendijk EO, Won CW, Abbatecola A, Cederholm T, Strandberg T, Gutiérrez Robledo LM, Flicker L, Bhasin S, Aubertin-Leheudre M, Bischoff-Ferrari HA, Guralnik JM, Muscedere J, Pahor M, Ruiz J, Negm AM, Reginster JY, Waters DL, Vellas B. Physical Frailty: ICFSR International Clinical Practice Guidelines for Identification and Management. *J Nutr Health Aging* 2019; 23: 771-787. Copyright© The Authors 2019. Published by Elsevier. The article is open access (Supplementary material).

(developed by Mitnitski *et al*[70]).

The Fried frailty phenotype, which relies on physical metrics like grip strength and walking speed, is foundational to understanding the frailty syndrome in community settings[69]. However, it is entirely impractical in acute care setting, as it is impossible to perform physical assessments on a patient who is acutely ill, delirious, or on a ventilator[71]. Our review focuses entirely on tools derived from the cumulative deficit model. This model defines frailty as the progressive accumulation of health deficits across multiple domains (functional, cognitive, and comorbid). Because this model does not require a physical test and can be derived retrospectively *via* collateral history or medical records, it is the most practical choice for acute and critical care environments. Acute care relies on two primary methodologies rooted in this specific model: (1) Rapid clinical judgment through the CFS[72]; and (2) Chart-derived indices such as the FI[70].

The CFS

Developed by Rockwood *et al*[72], the CFS summarizes a patient's overall trajectory of fitness and frailty into a 9-point visual scale. Rather than assessing the patient's current state of physiological instability, it relies on clinical judgment and collateral history of the patient's function two weeks prior to the acute illness[4]. CFS is a preferred bedside tool across the ED, ICU, and general acute wards because it is rapid and seamlessly integrates comorbidity, physical disability, and cognitive impairment into a single metric[4,73]. The CFS is a robust, independent predictor of mortality and adverse discharge outcomes across both geriatric trauma and acute medical admissions[74,75].

The FI and automated tools

For patients without available collateral history, the FI offers an objective alternative. Based on the cumulative deficit model proposed by Mitnitski *et al*[70], the FI views frailty as a mathematical proportion of defects[76]. FI is calculated by taking the number of deficits present in a patient and dividing that figure by the total number of potential deficits measured. Because the FI generates a score on a continuous scale, the FI provides highly granular risk stratification. Generally, a score above 0.25 indicates frailty[77]. Interestingly, scores rarely climb above 0.67. This value represents the theoretical limit of human survival, as reaching this point suggests that the body can no longer sustain life[78]. Because manual calculation in an acute setting is often impractical, clinicians now frequently utilize the automated versions of the tool. Systems such as the Hospital Frailty Risk Score and the electronic FI extract historical diagnostic data directly from electronic health records (EHR)[79,80]. This allows clinicians to quantify a patient's baseline vulnerability and clearly distinguish their pre-existing functional reserve from the sudden physical stress caused by their current illness[81]. To facilitate clinical integration, Table 1 provides a structured comparison of acute illness severity scores and frailty assessment frameworks, highlighting their specific strengths, limitations, and complementary roles in geriatric care.

Table 1 Comparison of acute illness severity scores and frailty assessments: Characteristics, limitations, and complementary roles in geriatric care

Comparative domain	Acute illness severity scores	Frailty assessments
Core concept	Measures the physiological “load” (the acute insult)	Measures the biological “chassis” (the baseline reserve)
Primary clinical variables	SOFA: Organ dysfunction labs, hemodynamics, and GCS[43]. qSOFA: Rapid bedside clinical signs[45]. NEWS2: Routine bedside vital signs[52]. APACHE: Worst physiological values during the first 24 hours of ICU admission[60,61]	CFS: Clinical judgment and collateral history of the patient’s baseline function [72]. FI/eFI: Mathematical accumulation of health deficits extracted <i>via</i> medical records or EHR[70,79]
Temporal focus	Current physiological state (real-time or 24-hour window)	Pre-existing baseline state (<i>e.g.</i> , functional status 2 weeks prior for CFS, or accumulated lifetime deficits for FI)
Key strengths	Highly standardized; universal language for triage; excellent for detecting rapid physiological deterioration	Captures biological age rather than chronological age; predicts capacity for functional recovery and long-term trajectory
Limitations in the geriatric population	SOFA: Pre-existing chronic organ dysfunction masks the true acute insult. qSOFA: Fixed SBP thresholds miss relative hypotension; GCS confounded by dementia/delirium. NEWS2: Chronic vital derangements cause alarm fatigue; blunted physiological responses lead to false negatives. APACHE: Linear age-weighting ignores biological reserve; lab-heavy scoring misses severe illness when systemic responses are blunted	CFS: Relies heavily on the availability and accuracy of collateral history. FI/eFI: Manual calculation is impractical during emergencies without automated EHR integration
Complementary role in integration	Identifies the immediate, life-threatening physiological derangement requiring urgent resuscitation	Determines the underlying biological reserve to withstand the acute insult, predicting functional recovery and guiding goals of care

APACHE: Acute Physiology and Chronic Health Evaluation; CFS: Clinical Frailty Scale; EHR: Electronic health records; FI: Frailty index; eFI: Electronic frailty index; GCS: Glasgow Coma Scale; ICU: Intensive care unit; NEWS2: National Early Warning Score 2; qSOFA: Quick Sequential Organ Failure Assessment; SBP: Systolic blood pressure; SOFA: Sequential Organ Failure Assessment.

SYNERGISTIC RISK STRATIFICATION: INTEGRATING FRAILTY WITH ACUTE ILLNESS SEVERITY SCORES

When managing the care of older adults, we should not look at acute illness severity and baseline frailty as separate issues. While physiological scoring systems tell us what a patient needs right now for resuscitation, frailty tools reveal if the patient has the biological capacity to survive both the illness and the intense stress of medical interventions. Recent literature suggests that we must evaluate these two domains together to achieve a high level of clinical precision. Whether we apply this to early warning scores (*e.g.*, NEWS) and sepsis screening tools (*e.g.*, qSOFA) in the ED, or critical care prognostic models (*e.g.*, SOFA, APACHE II) in the ICU, research confirms that adding the CFS greatly improves our ability to predict who will need an intensive care admission[82] and what their overall risk of mortality might be[82-84]. This multidimensional approach transforms standardized scoring from a blunt biological metric into a highly individualised, person-centered tool. It serves as a direct guide for clinicians when they must decide on emergency triage, the intensity of treatment, and early goals-of-care (GOC) discussions.

EDs and initial triage

As our population ages rapidly, EDs are increasingly managing older adults with multimorbidity and high levels of vulnerability. Acute illnesses, ranging from sepsis to cardiopulmonary events, frequently present atypically in older adults due to diminished physiological reserve. This clinical complexity makes accurate risk stratification in the ED essential to safely guide decision-making and optimize resources allocation. However, standard early warning and triage scores rely predominantly on acute physiological parameters that often underestimate true illness severity in this frail group[57]. Baseline frailty is a vital complement that acts as a strong predictor of adverse clinical outcomes, as demonstrated by Ellis *et al*[85] in a study of over 68000 ED visits. Therefore, integrating CFS directly into the triage workflow alongside these acute severity scores is key to construct an accurate, multidimensional risk profile. This approach is strongly supported by the expert clinical commentary of Vardy *et al*[86]. Recent multi-center studies demonstrate that integrated models consistently outperform standard early warning scores alone across several critical ED endpoints.

Predicting ICU admission and in-hospital mortality: Chung *et al*[82] demonstrated that integrating the CFS with standard acute scores [NEWS2, qSOFA, and Rapid Emergency Medicine Score (REMS)] significantly improved the AUROC for predicting ICU admission and in-hospital mortality.

Improving short- and long-term mortality prognostication: Wretborn *et al*[83] confirmed that the systematic use of the CFS alongside NEWS and standard triage tools markedly improved 30-day mortality prognostication. Providing the statistical rationale, Engvig *et al*[87] demonstrated a significant statistical interaction effect between the CFS and NEWS2. Their work revealed that the impact of frailty on a patient's outlook is even more significant when the acute illness is severe. These findings confirm that baseline vulnerability must be factored into front-door risk stratification to accurately predict survival trajectories.

Predicting in-hospital cardiac arrest: Biuzzi *et al*[88] looked at using NEWS2 and frailty assessments together to predict cardiac arrest in the hospital. This integrated approach successfully identified older patients at high risk for sudden deterioration. This is a catastrophic outcome that standard physiological monitoring often fails to capture in frail cohorts.

Ultimately, these integrated models prove that triage based on frailty transforms one-dimensional physiological scores into precise tools for prognosis. This multidimensional approach empowers clinicians to rapidly identify and allocate life-saving resources to highly vulnerable patients, even when their illness is masked by atypical presentations and low biological reserve.

ICU and critical care prognostication

Accurate risk stratification becomes even more critical when older adults are admitted to the ICU. Standard critical care scoring systems, such as the APACHE and the SOFA, rely entirely on acute physiological parameters. This often underestimates mortality in older adults because these tools ignore the baseline biological reserve. Fundamentally, baseline frailty acts as a crucial prognostic complement that dictates how well a patient can endure both the critical illness and the stress of intensive therapy. This is supported by foundational meta-analyses by Muscedere *et al*[89], which showed that frailty acts as a powerful, independent predictor of mortality irrespective of admission acute severity scores. Therefore, experts in critical care now strongly advocate for the formal integration of the CFS alongside these standard ICU severity scores to accurately predict survival and functional trajectories[90]. Landmark and contemporary large-scale studies show that concurrent assessment of acute illness and frailty consistently outperforms standard critical care scores alone.

The necessity of dual-axis risk stratification: Le Maguet *et al*[2] and Bagshaw *et al*[91] identified that frailty is a robust, independent predictor of both short- and long-term mortality. It acts as a necessary complement to traditional physiological indices [Simplified Acute Physiology Score (SAPS) II, APACHE II, SOFA]. Within the very old intensive care patients network, Guidet *et al*[92] confirmed that the CFS and the SOFA score act as powerful, concurrent predictors of short-term mortality across general ICU admissions. Furthermore, Haas *et al*[93] proved these metrics concurrently predict 6-month mortality in septic cohorts. Most definitively, Bruno *et al*[94] meta-analysis of nearly 24000 geriatric patients confirmed that frailty remains a powerful, independent predictor of ICU mortality alongside the SOFA score. They identified a “frailty continuum”, whereby mortality risk increases progressively with each CFS category.

Superior predictive accuracy of integrated scoring models: Zeng *et al*[95] showed that incorporating an FI to capture premorbid functional data increased the AUROC for mortality from 0.86 to 0.92 for APACHE II, and from 0.88 to 0.93 for APACHE IV. Similarly, Szűcs *et al*[84] demonstrated that combining the CFS with acute illness scoring improved the prognostic accuracy for mortality of APACHE II from 0.72 to 0.80, and SAPS II from 0.79 to 0.84.

Aligning prognosis and treatment intensity in nonagenarians: Suh *et al*[96] demonstrated in over 8200 critically ill nonagenarians that the APACHE III-J and CFS independently predict mortality and prolonged hospitalization.

Methodological challenges in integration: Despite strong consensus supporting the combination of these tools, the improvements in predictive power are not always consistent. For example, Langlais *et al*[97] found that integrating the CFS with the SOFA score *via* a regression-derived model did not significantly outperform the SOFA alone in predicting hospital mortality. In their study, the AUROC of the SOFA-CFS score was 0.66 compared to 0.63 for the SOFA alone, which was not statistically significant. This likely happens because acute physiological changes can be so dominant in smaller cohorts, that they effectively “drown out” the prognostic signal of baseline frailty in standard models. This gap in the results highlights a clear need for better integration. Although measuring both dimensions is essential, we need more advanced mathematical models to ensure that the prognostic value of frailty is not simply overshadowed by acute physiological data.

Ultimately, integrating the CFS into ICU prognostication helps clinicians differentiate between a reversible acute illness and an irreversible decline. This ensures that life-sustaining interventions align with the patient's biological reserve, to effectively guide goals-of-care discussions. A summary of key primary literature evaluating the concurrent assessment and formal integration of baseline frailty and acute illness severity scores across acute care settings is provided in Table 2.

A PRACTICAL GUIDE TO FRAILTY-INTEGRATED RISK STRATIFICATION

Moving from theory to clinical practice, this mini-review provides a practical guide for frailty-integrated risk stratification to individualize acute care for older adults. We have designed this framework for clinicians working across the full spectrum of acute care, encompassing the ED, acute medical wards, and the ICU. Relying exclusively on standard acute severity scores frequently leads to inaccurate risk stratification in older adults. In contrast, our proposed algorithm proactively identifies distinct clinical phenotypes. This helps guide multidisciplinary decisions regarding critical care escalation, ward-based management, and the establishment of appropriate ceilings of care.

Table 2 Key primary studies evaluating the dual-axis assessment and prognostic integration of baseline frailty and acute illness severity scores

Ref.	Study design (n)	Scoring systems	Key findings
Emergency department			
Chung <i>et al</i> [82], 2025	Retrospective multi-center (n = 932)	CFS and (NEWS2, qSOFA, REMS)	Combined assessment significantly improved AUROC for predicting hospital admission, ICU admission, and in-hospital mortality (mortality AUROCs 0.77-0.82) compared to isolated scores
Wretborn <i>et al</i> [83], 2024	Prospective multi-center (n = 1832)	CFS and (NEWS, TEWS, RETTS)	Combined assessment significantly improved the AUROC for short-term mortality prognostication (0.53 to 0.82) compared to isolated early warning and triage tools (<i>e.g.</i> , NEWS)
Engvig <i>et al</i> [87], 2022	Prospective single-center (n = 195)	CFS and NEWS2	Combined assessment revealed a significant interaction effect ($P = 0.003$), demonstrating that the prognostic impact of frailty is greater at higher levels of acute illness severity compared to isolated scores
Biuzzi <i>et al</i> [88], 2026	Retrospective single-center (n = 70)	CFS and NEWS2	Combined assessment successfully identified highly frail patients at risk for in-hospital cardiac arrest who presented with deceptively low acute physiological scores
Intensive care unit			
Le Maguet <i>et al</i> [2], 2014	Prospective multi-center (n = 196)	FP and SAPS II, CFS and SOFA	Multivariate analysis demonstrated that FP and SAPS II independently predict short-term mortality, while CFS and SOFA concurrently predict long-term mortality
Bagshaw <i>et al</i> [91], 2014	Prospective multi-center (n = 421)	CFS and (SOFA, APACHE II)	Multivariable analysis confirmed that frailty is independently associated with higher in-hospital and long-term mortality after adjusting for SOFA and APACHE II scores. Highlighted mortality increased in a dose-dependent manner with each incremental increase in the frailty score
Guidet <i>et al</i> [92], 2020	Prospective multinational (n = 3920)	CFS and SOFA	Both scores act as powerful, independent predictors of short-term mortality, reinforcing the necessity of dual-axis risk stratification
Haas <i>et al</i> [93], 2021	Prospective multinational (n = 532; sepsis subset of VIP2)	CFS and SOFA	Within the septic sub-cohort, SOFA and CFS scores act as robust, independent predictors of long-term mortality
Bruno <i>et al</i> [94], 2023	IPD meta-analysis (n = 23989)	CFS and SOFA	In geriatric cohort (≥ 65 years), frailty remains a robust, independent predictor of ICU mortality alongside the SOFA score. Highlighted a “frailty continuum” where risk increases progressively across CFS categories
Zeng <i>et al</i> [95], 2015	Prospective single-center (n = 155)	FI and (APACHE II, APACHE IV)	FI captures essential premorbid functional data, increasing the AUROC for mortality from 0.86 to 0.92 (APACHE II) and 0.88 to 0.93 (APACHE IV)
Szűcs <i>et al</i> [84], 2025	Prospective single-center (n = 212)	CFS and (APACHE II, SAPS II)	Integrating the CFS significantly increased the AUROC for mortality from 0.72 to 0.80 (APACHE II) and from 0.79 to 0.84 (SAPS II)
Suh <i>et al</i> [96], 2025	Retrospective multi-center (n = 8220)	CFS and APACHE III-J	Both tools remained strong independent predictors of mortality and prolonged hospitalization in critically ill nonagenarians
Langlais <i>et al</i> [97], 2018	Prospective single-center (n = 189)	CFS and SOFA	Integration of CFS and SOFA yielded a non-significant AUROC improvement for mortality (0.63 to 0.66, $P = 0.082$)

APACHE: Acute Physiology and Chronic Health Evaluation; AUROC: Area under the receiver operating characteristic curve; CFS: Clinical Frailty Scale; ED: Emergency department; FI: Frailty Index; FP: Frailty phenotype; ICU: Intensive care unit; IPD: Individual patient data; NEWS: National Early Warning Score; NEWS2: National Early Warning Score 2; qSOFA: Quick Sequential Organ Failure Assessment; REMS: Rapid Emergency Medicine Score; RETTS: Rapid emergency triage and treatment system; SAPS II: Simplified Acute Physiology Score II; SOFA: Sequential Organ Failure Assessment; TEWS: Triage Early Warning Score.

Operationalizing the airway, breathing, circulation, disability, exposure, and frailty framework: From primary survey to risk-stratified level-of-care decisions

Recent consensus in emergency and intensive care medicine has advocated for a fundamental update to the initial patient assessment: Evolving the traditional primary survey into an Airway, Breathing, Circulation, Disability, Exposure, and Frailty (ABCDE-F) framework[98]. This 2025 recommendations elevates frailty assessment to the same level of clinical urgency as securing an airway or stabilizing hemodynamics.

However, identifying frailty during the primary survey is only the first step. Once clinicians address immediate life-threatening conditions, they must translate this baseline assessment into an actionable clinical pathway. While the ABCDE-F framework highlights what to assess during resuscitation, it does not provide a mechanism to integrate frailty with acute illness severity scores to determine the optimal level of care. To bridge this gap, we propose a four-step clinical triage algorithm and the synergistic 2×2 risk stratification matrix. This model provides a clear pathway to transition the patient from initial resuscitation to appropriate critical care escalation, ward-based management, or comfort-focused

palliative care.

Step 1: Establishing the baseline frailty (the “two-week rule” CFS)

The rationale for using the CFS in acute triage: As we have discussed, the CFS is uniquely suited for Step 1 because it relies on an overall assessment of baseline functional status. Unlike other tools, it does not require physical performance metrics or exhaustive medical record reviews. This allows clinicians to rapidly identify a patient’s intrinsic physiological reserve and separate it from the acute decompensation they are currently facing.

Operationalizing the “two-week rule”: To successfully operationalize the “F” in the ABCDE-F primary survey, the CFS must be calculated strictly based on the patient’s functional status and level of dependency two weeks prior to the onset of the acute illness[4]. Clinicians must actively avoid scoring the acute presentation. A frequent mistake involves assigning a high frailty score to a previously independent patient who is now bed bound or delirious because of their current illness. Since acute illness frequently masks the true baseline, obtaining collateral history from caregivers or primary care records is a mandatory step. This ensures the CFS score accurately reflects the patient’s true biological reserve rather than a transient, acute functional decline.

Feasibility and reliability in practice: In time-constrained environments like the ED and ICU, the CFS proves to be exceptionally practical. Multi-center studies show that it can be reliably administered by frontline personnel, including triage nurses and non-geriatricians, with good inter-rater reliability (weighted kappa 0.74)[99]. Furthermore, the CFS is exceptionally efficient. Evaluations report a mean completion time of only 24 seconds, making it the fastest diagnostic tool for acute triage[100]. Using structured classification trees also helps with rapid and accurate categorization[73]. This ensures that frailty screening seamlessly integrates into standard hospital workflows without adding to the clinical workload.

Step 2: Quantifying the acute insult (NEWS2, SOFA, and/or qSOFA)

Once the premorbid “F” (Frailty) is established, the clinician must measure the severity of the acute physiological insult. We use established scoring systems to provide an objective measure of the acute “hit”. To ensure our synergistic matrix remains practical at the bedside, we split the acute severity axis into two clear categories based on validated triggers for clinical escalation: (1) High acute severity: Defined as an aggregate NEWS2 score of 5 or more, a single “Red” parameter score of 3[52], or a SOFA or qSOFA score of 2 or more[45,47]. These thresholds represent validated “red zones” associated with significantly increased mortality and the need for urgent intervention; and (2) Low acute severity: Defined as scores that fall below these specific triggers, such as NEWS2 between 0 and 4 or a SOFA or qSOFA score of 0 or 1. These patients typically represent a lower immediate risk of physiological collapse, provided “silent severity” is not present.

Recognizing “masked risk” in the frail older adults: When calculating these scores for older adults, clinicians must maintain a high index of suspicion for masked risk. Due to age-related physiological changes and polypharmacy (*e.g.*, beta-blockers), frail patients often do not show a traditional “textbook” response to a severe illness. A reassuringly low acute severity score (*e.g.*, NEWS2 < 5) in a frail patient (*e.g.*, CFS ≥ 5) is frequently a clinical red herring. This misleading result can hide a serious emergency. This silent severity may manifest as: (1) Blunted tachycardia: A major infection or shock may present without a fast heart rate due to beta-blockade or aging conduction system; (2) Afebrile sepsis: A patient may present with normothermia or even hypothermia instead of the expected febrile response during an infection; and (3) Relative hypotension: A “normal” blood pressure reading might actually represent profound shock for a patient with a baseline chronic hypertension.

Consequently, while our matrix provides objective numerical thresholds, a low severity score in a frail patient should never be seen as a definitive “safe zone”. In these cases, clinicians should have a low threshold for further testing (*e.g.*, measuring lactate levels and screening for sepsis), as the raw physiological data may significantly underestimate the true severity of the acute insult.

Step 3: Identifying clinical phenotypes via the synergistic 2 × 2 risk stratification matrix

By intersecting the premorbid CFS (Step 1) and the acute severity score (Step 2) onto the synergistic 2 × 2 risk stratification matrix, the clinician can identify one of four distinct clinical phenotypes. This visualization moves beyond a one-size-fits-all approach to scoring. It generates a personalized risk profile by intersecting the patient’s baseline biological reserve with the weight of the acute “hit”. As illustrated in [Figure 2](#), this approach transforms numerical data into a four-quadrant model.

Low risk (low frailty and low acute severity): These patients possess preserved biological reserve and minimal acute physiological derangement. They represent the most stable cohort, where the acute illness is unlikely to exceed the patient’s intrinsic compensatory mechanisms.

Masked resilience (low frailty and high acute severity): This phenotype presents with significant acute derangement but possess a robust biological reserve. They are often capable of tolerating intensive resuscitation. The primary challenge here is “pessimism bias”, where life-saving treatment might be prematurely withheld based on chronological age or high acute severity scores rather than the patient’s actual biological potential for reversibility.

Masked risk (high frailty and low acute severity): This is the clinical “danger zone”. These patients appear stable on paper but possess a very low biological reserve. They frequently exhibit silent severity, which is a lack of overt physiological derangement despite significant underlying illness. This phenotype is at high risk for atypical deterioration,

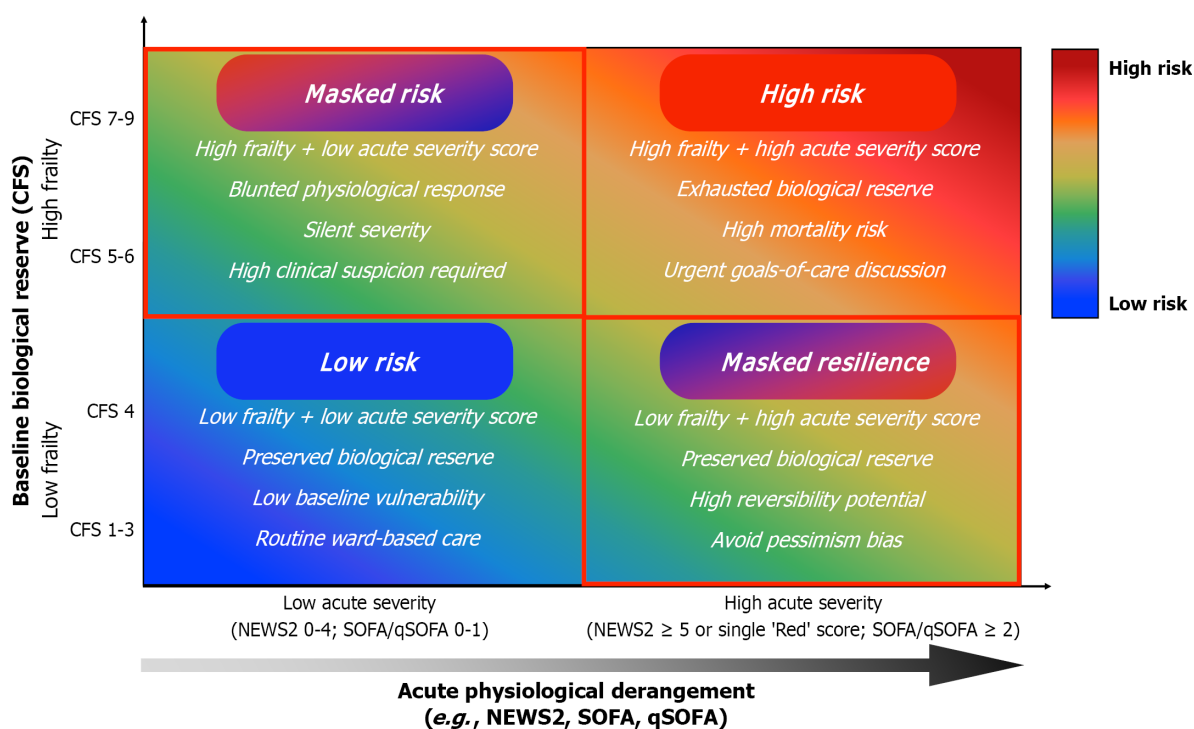


Figure 2 The synergistic 2 × 2 risk stratification matrix. This conceptual model illustrates the prognostic value of intersecting baseline biological reserve (Y-axis) with acute physiological derangement (X-axis). Baseline reserve is stratified by the Clinical Frailty Scale (CFS)[4,72], dichotomized into low frailty (CFS 1-4) and high frailty (CFS 5-9). The X-axis represents an escalating gradient of acute illness severity, categorized as low acute severity [National Early Warning Score 2 (NEWS2) 0-4; Sequential Organ Failure Assessment (SOFA)/quick SOFA (qSOFA) 0-1] or high acute severity (aggregate NEWS2 ≥ 5 or a single 'Red' parameter score of 3[52]; SOFA/qSOFA ≥ 2[45,47]). The background color gradient reflects the escalating risk of mortality and functional decline. The discordant quadrants identify critical clinical phenotypes: Masked Risk (where high frailty blunts physiological response, leading to "silent severity") and Masked Resilience (where robust biological reserve allows for survival of severe acute insults, provided "pessimism bias" is avoided). CFS: Clinical Frailty Scale; NEWS2: National Early Warning Score 2; SOFA: Sequential Organ Failure Assessment; qSOFA: Quick Sequential Organ Failure Assessment.

delirium, and rapid functional decline if the underlying insult is underestimated.

High risk (high frailty and high acute severity): This phenotype represents the most challenging scenario. It involves a severe acute derangement occurring in a body with a severely depleted biological reserve. In this cohort, the clinical focus shifts toward determining the proportionality of care. Clinicians must identify whether the patient has the biological capacity to withstand the intense stress of invasive medical interventions.

The role of Comprehensive Geriatric Assessment in the triage framework: While the 2 × 2 matrix serves as the initial triage engine at the "front door", the Comprehensive Geriatric Assessment (CGA) remains the gold standard for any older adult in the acute setting. These two approaches work in tandem rather than in isolation. While the matrix identifies the urgency and intensity of care, a CGA performed early in the admission identifies the multidimensional needs and the reversible factors that triggered the acute presentation[101].

Step 4: Integrated decision-making: Triage, treatment intensity, and goals of care

This final step puts the clinical phenotypes from Step 3 into action by creating clear care pathways. By integrating frailty, clinicians can look beyond a single dimension of illness severity. This allows for individualized decision-making across three vital areas of hospital care.

Triage and hospital disposition: The intersection of frailty and acute severity provides a roadmap for hospital placement. It helps direct patients to the environment most capable of managing their specific phenotype: (1) Intensive care/high dependency unit (HDU): Indicated for masked resilience group to facilitate intensive resuscitation. This level of care may also be considered for high risk patients where the goal is rapid stabilization or a time-limited trial (TLT) within the ICU. By identifying masked resilience, clinicians can advocate for critical care for biologically robust older adults who might otherwise be denied escalation due to ageist assumptions; (2) Specialized acute geriatric unit (AGU): Mandatory for the masked risk phenotype and highly appropriate for high risk patients undergoing a ward-based TLT. These patients possess a "silent" or "overt" severity that is too unstable for a general ward but may not benefit from invasive technology. The AGU provides the multidisciplinary vigilance (e.g., the ACE model) required to manage acute illness while simultaneously preventing delirium and functional decline[102-104]; and (3) Standard ward triage: Appropriate for low risk patient. Management focuses on routine ward-based care. However, even in this group, an early CGA remains essential to mitigate the risk of hospital-associated disability (HAD) and functional decline.

Titrating treatment intensity and defining success: Integrating frailty assessment with acute severity scores ensures proportionality of care by shifting the definition of clinical success based on the patient's specific biological reserve: (1) Masked resilience: High treatment intensity is justified regardless of chronological age. Success is defined as a return to the robust premorbid functional baseline through intensive resuscitation and early ICU/HDU escalation; (2) Masked risk: Success is defined by the preservation of current function. This requires high-vigilance monitoring and early specialized geriatric interventions to avoid "hidden" decompensation and iatrogenic harm; and (3) High risk: Treatment intensity is titrated *via* a TLT of high-intensity care (typically 48-72 hours). Success is defined by a dignified "pivot" to palliative care if physiological improvement is not observed within the TLT window, protecting the patient from non-beneficial, invasive interventions.

Navigating the "dual-axis reality" in goals-of-care discussions: The dual-axis reality serves as a conceptual bridge for shared decision-making during GOC discussions. This framework helps families distinguish between the physiological success of treating an illness (the acute axis) and the functional recovery of the person (the chronic axis). While a GOC discussion is mandatory for all acutely ill older adults, the focus of the conversation shifts based on the clinical phenotype: (1) Masked resilience (the "green light"): In this scenario, the conversation focuses on justification. Clinicians explain how a robust biological reserve (chronic axis) justifies intensive medical escalation, even when the patient has a severe physiological presentation (acute axis); (2) Masked risk (the "warning"): Here, the focus shifts to proactive vigilance. Clinicians explain that while the patient may appear stable (acute axis), their depleted reserve (chronic axis) makes them highly vulnerable. This discussion prepares the family for a potentially slow recovery, the risk of delirium, and the need for specialized geriatric monitoring; and (3) High risk (the "threshold"): In this challenging group, the focus is on proportionality and limits. Clinicians clarify that while medical technology may be successful in treating the illness (acute axis), it cannot restore an exhausted biological reserve (chronic axis). This facilitates discussions regarding TLT and a dignified transition to comfort-focused care if the biological limit is reached.

Ultimately, Step 4 ensures our synergistic matrix triages patients not just into hospital beds, but into individualized care plans that respect both biological limits and personal dignity. These phenotype-specific clinical implications are summarized in [Table 3](#), while the comprehensive four-step decision-making process is put into practice through our clinical triage algorithm ([Figure 3](#)).

CLINICAL VIGNETTES: APPLYING THE PROPOSED 4-STEP ALGORITHM

To illustrate the clinical utility of this integrated approach, we present two contrasting cases of older adults presenting to the ED.

Case A: The masked resilience phenotype

Patient: An 82-year-old male presenting with suspected urosepsis.

Step 1 (the "two-week rule" baseline CFS): Two weeks prior to admission, this patient was an avid gardener, drove daily, and lived independently (CFS 2: Fit).

Step 2 (acute hit): His NEWS2 was 9 (tachycardia, hypotensive, febrile).

The clinical trap: Based on chronological age and a high NEWS2 score, a traditional "linear" triage model might prematurely suggest a "ceiling of care" or a palliative approach. This happens because of a perceived vulnerability that is actually not present.

Step 3 (synergistic matrix): Low frailty combined with high acute severity identifies masked resilience. This reveals a robust biological reserve that is capable of surviving a severe acute illness.

Step 4 (shared decision-making and disposition): Using the premorbid functional baseline as justification, the clinician advocates for full escalation to ICU. The patient underwent intensive resuscitation and was successfully discharged back to his baseline functional status within 7 days.

Case B: The masked risk phenotype

Patient: An 85-year-old female presenting with suspected pneumonia.

Step 1 (the "two-week rule" baseline CFS): Two weeks prior to admission, this patient with pre-existing dementia of moderate severity required assistance with showering and dressing, though she remained mobile with a walking frame (CFS 6: Moderate frailty).

Step 2 (acute hit): Her NEWS2 was 2 (slight tachypnea, but otherwise "stable" hemodynamics).

The clinical trap: Because physiological responses are often blunted by frailty, a standard triage system would categorize her as "low risk". This leads to routine ward placement and delayed intervention.

Step 3 (synergistic matrix): High frailty combined with low acute severity identifies masked risk. This points to silent severity. Even a minor acute hit can lead to catastrophic collapse in a patient with depleted biological reserve.

Table 3 Phenotype-specific clinical implications: Triage, treatment intensity, and goals of care

Clinical phenotype	Triage and disposition	Treatment intensity	GOC strategy
Low risk	Standard ward	Routine monitoring: Focus on preventing functional decline	Goal alignment: Align treatment with baseline patient goals and functional priorities
Masked resilience	ICU/HDU	Full escalation: Aim for return to robust baseline	Justification: Explain how robust reserve justifies intensive care
Masked risk	AGU	High vigilance: Preserve function; prevent delirium	Proactive warning: Highlight “silent severity” and the high risk of rapid functional decline
High risk	ICU/HDU or AGU	Proportionality: TLT of intensive care	Thresholds: Shift focus to dignity if biological limits are met

GOC: Goals of care; HDU: High dependency unit; AGU: Acute geriatric unit; ICU: Intensive care unit; TLT: Time-limited trial.

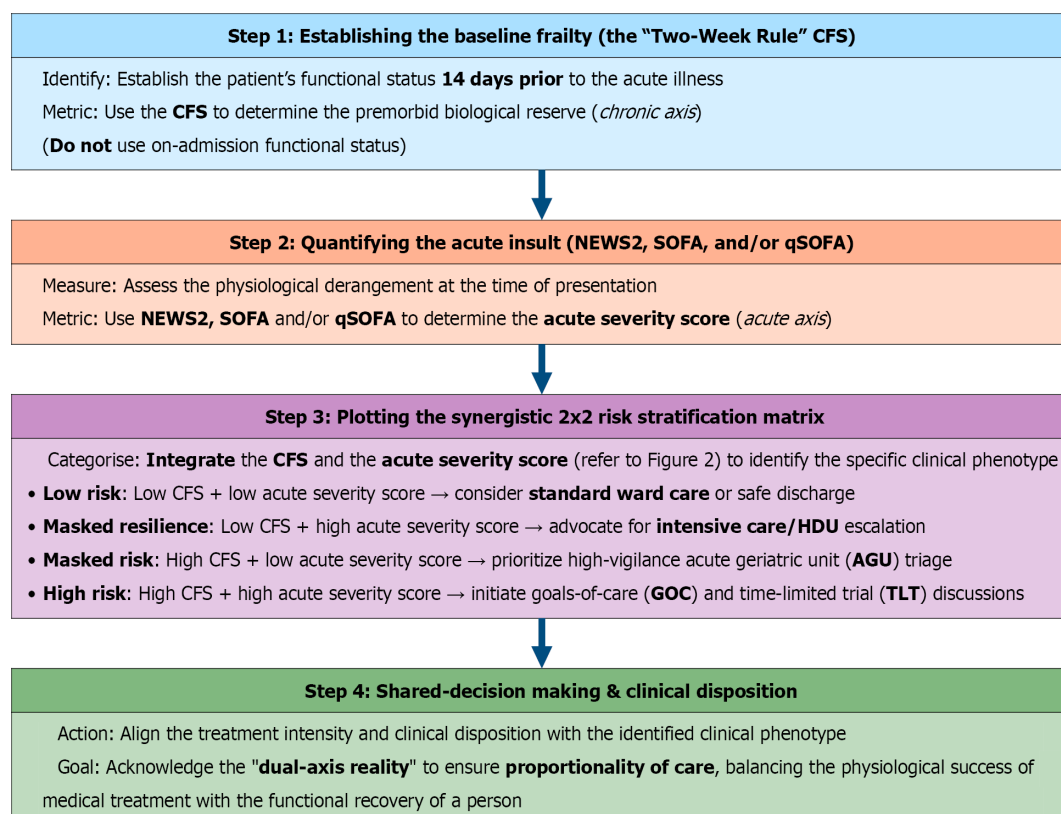


Figure 3 Proposed 4-step clinical triage algorithm for acute geriatric care. The algorithm dictates integration of the “Two-Week Rule” (baseline Clinical Frailty Scale) and acute physiological derangement (National Early Warning Score 2, Sequential Organ Failure Assessment and/or quick SOFA) to guide phenotype-driven triage and shared decision-making. CFS: Clinical Frailty Scale; NEWS2: National Early Warning Score 2; SOFA: Sequential Organ Failure Assessment; qSOFA: Quick Sequential Organ Failure Assessment; HDU: High dependency unit; AGU: Acute geriatric unit; GOC: Goals-of-care; TLT: Time-limited trial.

Step 4 (shared decision-making and disposition): Recognizing the “dual-axis reality”, the clinician triages the patient to a specialized AGU for high-vigilance monitoring and delirium prevention. A GOC discussion is initiated to establish a TLT with the family. The focus is on non-invasive support that honors her functional baseline and personal dignity.

LIMITATIONS AND FUTURE DIRECTIONS

While the proposed 4-step algorithm provides a logical framework for acute geriatric triage, several limitations must be acknowledged to ensure its safe and effective application in clinical practice.

Reliance on collateral history

The success of the “two-week rule” is entirely dependent on the availability and accuracy of collateral history. In cases of

social isolation or delirium where no primary caregiver is present, establishing a precise premorbid CFS remains a significant clinical challenge. This difficulty is even more apparent in environments with weak healthcare infrastructure, incomplete medical records, or limited family support. In rural areas or developing countries, reliable primary care data may be entirely inaccessible, making it difficult to distinguish between a patient's true reserve and the functional decline precipitated by the current medical crisis.

Inter-rater reliability

Although the CFS is a validated tool, its inter-rater reliability can vary between clinicians of different specialties or experience levels. Surkan *et al* [105] highlighted that while inter-rater reliability agreement is generally good, discrepancies between geriatricians and other specialists, such as intensivists, often occur. This is particularly true in the “grey zone” of moderate frailty. These findings underscore the need for standardized training and the rigorous application of the “two-week rule” to minimize subjective bias during the initial assessment.

Need for prospective validation

While the synergistic matrix utilizes established independent metrics (NEWS2, SOFA, and CFS), the integrated 2×2 model itself requires formal prospective validation. Future research must focus on evaluating combined models that integrate acute severity scores with baseline frailty assessments. Specifically, we need large-scale prospective cohort studies and randomized clinical trials to explore how these integrated, dual-axis models impact clinical outcomes compared to standard one-dimensional triage. Key outcomes of interest should include 30-day mortality, length of hospitalization, HAD, and the appropriateness of ICU resource utilization. Furthermore, future research should investigate whether the implementation of such phenotype-driven algorithms actually changes clinician behavior, improves the timeliness of GOC discussions, and ultimately increases the proportion of older adults receiving care that aligns with their personal goals.

Future directions in digital integration

Embedding the synergistic matrix directly into EHR is the necessary next step in transitioning from manual assessment to real-time decision support. Automated identification of specific clinical phenotypes, such as masked risk and masked resilience, would allow geriatricians and acute care teams to receive real-time alerts at the point of care. However, the path to implementation is complicated by the fragmented health information systems and the lack of standardized geriatric data fields across different platforms. Furthermore, the success of these digital tools will hinge on meticulous calibration. Our goal must be to provide meaningful diagnostic insights that improve clinical flow, rather than overwhelming staff through “alert fatigue”.

CONCLUSION

Navigating the interplay between acute physiological derangement and baseline frailty remains a primary challenge in acute geriatric care. Traditional triage systems often rely on acute severity scores that fail to capture the biological heterogeneity of older adults. This practical guide proposes a 4-step clinical triage algorithm to bridge this clinical gap. By applying the “Two-Week Rule” to establish a baseline CFS and integrating it with acute severity scores (such as NEWS2, SOFA, and/or qSOFA) *via* a synergistic 2×2 risk stratification matrix, clinicians can move from reacting to a crisis toward using proactive triage.

This integrated approach helps identify distinct clinical phenotypes. It specifically reduces the under-triage of resilient patients who require intensive care and the over-triage of the terminally frail to life-sustaining interventions that are not beneficial. By transitioning to this phenotype-driven approach, clinicians can better guide goals-of-care discussions. They can focus on what represents an attainable functional recovery to ensure acute and critical care is medically proportionate and truly person-centered.

Finally, it must be explicitly noted that this proposed algorithm reflects an expert consensus rather than level 1 clinical evidence. Therefore, prospective, multi-center validation studies are needed to prove its impact on hospital outcomes and to establish its generalizability across diverse healthcare systems.

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