Junior researchers

ახალგაზრდა მკვლევარები

Vol. 4 Issue 1, 2026

https://doi.org/10.52340/jr.2026.04.01.03

Silent Neurological Decline and Emergency Department Monitoring

Arjun Jay Prakash[1], Medha Chakrapani[2], Tatia Mermanishvili[3], Jemeleine Cerbito[4], Fatma Baghdadi[2], Akshina Rathnaweera[5]

[1]MD, Ivane Javakishvili Tbilisi State University, Georgia;

[2]MD, University of Georgia, Georgia;

[3]MD, Tbilisi State Medical University, Georgia;

[4]MD, Ilia State University, Georgia;

[5]MD, Yerevan State Medical University, Armenia

**Corresponding author: Arjun Jay Prakash, MD, Ivane Javakhishvili Tbilisi State University, Georgia, arjunatschool@gmail.com, Mobile: +97466191967

**ORCID:

Arjun Jay Prakash: https://orcid.org/0009-0002-2063-0192;

Medha Chakrapani: https://orcid.org/0009-0003-1674-651X;

Tatia Mermanishvili: https://orcid.org/0009-0007-7164-1891;

Jemeleine Cerbito: https://orcid.org/0009-0007-7687-7422;

Fatma Baghdadi: https://orcid.org/0009-0008-0212-9365.

Abstract:

Early recognition of silent neurological deterioration in the emergency department remains a complex and high-stakes clinical challenge. Timely detection is limited by atypical symptom presentation, systemic comorbidities, cognitive biases, and workflow constraints, as well as the subtle and dynamic evolution of cerebral injury. Multimodal assessment, integrating clinical examination, point-of-care EEG, structured neuroimaging, AI-assisted interpretation, and systemic physiological monitoring, provides a structured framework to identify patients at risk and guide acute management. While early markers of neurological decline exist, their sensitivity is constrained in the ED, and reliance on conventional scoring systems alone may delay intervention or misclassify evolving deficits. This review

synthesizes current evidence on detection and monitoring of early or silent neurological deterioration, emphasizing the role of sequential, tiered evaluation, integration of multimodal data, and standardized escalation pathways. By contextualizing clinical, electrical, imaging, and systemic parameters in real time, clinicians can optimize acute care, reduce missed diagnoses, and preserve critical therapeutic windows for intervention.

Keywords: Silent neurological deterioration; Emergency department; Multimodal monitoring; Point-of-care EEG; Neuroimaging; AI-assisted diagnostics; Acute cerebral injury; Clinical assessment; Patient safety.

Abbreviations:

Silent neurological decline - SND

Silent brain infarction - SBI

National Institutes of Health Stroke Scale - NIHSS

Glasgow Coma Scale - GCS

Emergency department - ED

Early Neurological Decline - END

Point-of-care EEG - pEEG

Electroencephalography - EEG

Artificial Intelligence - AI

Magnetic Resonance Imaging - MRI

Intracerebral hemorrhage - ICH

Computed Tomography - CT

Introduction:

Silent neurological decline (SND) refers to the evolution of acute or subacute brain injury in the absence of clinically recognizable, or patient-perceived neurological symptoms. They consist of structural, physiological, or functional impairment that remains clinically occult during early evaluation. They are generally consequential pathology and are a diagnostic challenge, especially at a stage when intervention may still alter trajectory [11].

Silent brain infarction (SBI) represents the most well-characterized radiological manifestation of this phenomenon. SBI is defined as cerebral infarction identified on neuroimaging without corresponding focal neurological deficits, and historically have been regarded as incidental. However, recent evidence demonstrates that these lesions are associated with subtle but persistent neurological deficits, and increased all-cause mortality, cognitive impairment, gait dysfunction and functional deterioration. Hence they have also been described as 'covert brain infarction' [11]. They pose not just a diagnostic challenge, but also require an altered understanding of the condition since SBIs are an effective predictor of future neurological events, rather than being a mere marker/consequence of prior injury. SBIs have demonstrated an approximately two-fold increased risk of subsequent symptomatic stroke and the high prevalence of SBI (~18%) in stroke-free older adults, represent significant implications for future morbidity and mortality [11,12].

These observations highlight a central vulnerability in emergency neurological care: the over-reliance on clinical examination in detecting early or evolving brain injury. SND frequently precedes irreversible damage, and by the time it is clinically evident, the therapeutic window is either closed or significantly reduced [11]. Accordingly, this review examines the mechanisms, clinical contexts, and consequences of SND in emergency settings, with particular emphasis on early deterioration in ischemic stroke, ICH, and other acute brain insults. It further explores the role of structured neurological surveillance and emerging monitoring technologies in identifying covert deterioration, reducing missed diagnoses, and improving early neurological outcomes in the ED.

Methodology:

This literature review was conducted to collate and synthesize current evidence on early or silent neurological deterioration in the ED, with emphasis on clinical detection, neuromonitoring, neuroimaging, Artificial Intelligence (AI) -assisted interpretation, systemic physiological assessment, and structured escalation strategies. The review aimed to integrate findings on patient risk factors, SBI, multimorbidity, and ED workflow influences to inform a multimodal monitoring framework.

Search Strategy and Database:

Relevant articles were identified through PubMed, PMC, and Scopus databases using combinations of keywords including "silent neurological deterioration", "early neurological decline", "ED monitoring", "point-of-care EEG", "NIHSS", "early warning scores", "silent brain infarction", "neuroimaging", "AI-assisted neuroimaging", "cognitive bias", and "system-level monitoring". Only English-language, peer-reviewed articles with full-text availability were considered. Reference lists of included studies were screened to identify additional relevant publications.

Screening and Selection:

Abstracts were first screened for relevance to early detection of neurological deterioration, neuromonitoring strategies, imaging biomarkers, AI-assisted detection, and system-level response in the ED. Full texts were reviewed to include studies focusing on multimodal assessment, tiered monitoring frameworks, risk stratification, and guideline-informed escalation pathways. Selection criteria emphasized methodological rigor, clinical applicability, and relevance to real-time ED practice. Discrepancies in article selection or data interpretation were resolved through discussion and consensus among the reviewers.

Data Extraction and Synthesis:

Key study characteristics including patient population, monitoring modality, timing of assessment, detection metrics, outcomes, and reported limitations were extracted into a structured matrix. Evidence was synthesized qualitatively, integrating clinical, electrical, imaging, and systemic monitoring data to generate a conceptual framework for early recognition and intervention in silent neurological deterioration.

Epidemiology and Clinical Importance of SND

In acute stroke settings, SND frequently presents as Early Neurological Decline (END), defined as worsening of the National Institutes of Health Stroke Scale (NIHSS) within the first week following ischemic stroke [2,5]. They are driven by cerebral oedema, hemorrhagic transformation, stroke progression, or early recurrence and contain substantial prognostic significance, with more than 80% of affected patients developing functional dependence and nearly 20% dying within 90 days of advent [5]. SBIs represent the most clearly defined radiological substrate of SND. Although historically labelled as ‘silent’ these infarcts are now recognised as clinically meaningful, being associated with subtle neurological deficits, cognitive impairment, increased mortality, and an approximately two-fold increased risk of future symptomatic stroke. SBI affects an estimated 10-20% of the general population, with an annual incidence of 2-4%, implying cumulative lesion burden with advancing age [15]. Established risk factors include hypertension, carotid stenosis, chronic kidney disease, metabolic syndrome, heart failure, and coronary artery disease, with age and hypertension exerting the strongest influence [15,16].

A parallel pattern is observed in ICH, where neurological deterioration (defined as worsening NIHSS or Glasgow Coma Scale (GCS) scores) occur in approximately 30% of patients, most commonly within the first 48 hours [3]. In both ischemic and hemorrhagic stroke, early deterioration is strongly associated with death and long-term disability [3,5]. A further, under-recognised dimension of SND arises from missed or delayed diagnosis in the ED. In the United States alone, an estimated 15,000-165,000 cerebrovascular events are misdiagnosed annually, particularly when presentations involve

non-specific symptoms such as headache or vertigo. Diagnostic errors disproportionately affect women, younger patients, and non-White populations, leading to delays in treatment and poorer outcomes [8]. Altogether, SND is common, prognostically significant, and multifactorial. Whether driven by covert infarction, early post-stroke deterioration, hemorrhagic progression, or diagnostic failure in the ED, SND represents a critical contributor to preventable neurological morbidity.

Mechanisms of Early or Silent Neurological Deterioration

Early or silent neurological deterioration arises from a heterogeneous interplay of pathological processes that evolve over time rather than from a single discrete insult. In ischemic stroke, END reflects the convergence of vascular instability, metabolic stress, inflammatory activation, and systemic physiological fluctuations [2,5]. This complexity explains why clinical trajectories often diverge despite similar initial imaging or neurological assessments.

Stroke progression represents the dominant mechanism, accounting for approximately 68% of END cases [5]. Failure of collateral circulation and thrombus propagation or reocclusion compromise perfusion to initially viable peri-infarct tissue, rendering the ischemic penumbra highly sensitive to fluctuations in cerebral and systemic blood flow. Under these conditions, infarct expansion may occur despite early neurological stability or apparently unchanged imaging findings [5]. Recurrent ischemic stroke contributes to approximately 12% of END cases, while hemorrhagic transformation and cerebral edema each account for roughly 10%, highlighting the multiplicity of pathways through which deterioration may unfold [5].

Metabolic and inflammatory processes further modulate neurological vulnerability. Hyperglycemia, oxidative stress, lactic acidosis, blood-brain barrier disruption, and excitotoxicity amplify ischemic injury and accelerate infarct progression [5]. Elevated plasma glucose, ferritin, D-dimer, nitric oxide, excitotoxic amino acids, and pro-inflammatory cytokines have all been positively associated with END, alongside systemic factors such as elevated blood pressure and body temperature [5]. Conversely, lower blood pressure and anti-inflammatory cytokine profiles appear to correlate negatively with deterioration [5]. These biochemical and physiological gradients help account for marked differences in clinical evolution among patients with comparable early radiological findings.

Importantly, neurological deterioration may occur even after reperfusion therapy. Reocclusion, parenchymal hemorrhagic transformation, hyperperfusion syndrome, and infarction within previously hypoperfused regions all contribute to post-recanalization decline [2,5]. The ischemic penumbra remains susceptible to secondary insults, including systemic hypotension, intracranial hypertension, vasogenic edema, hyperglycemia, hypoxia, hypercapnia, and reocclusion, illustrating that successful recanalization does not guarantee neurological recovery [5]. In this context, reperfusion may expose metabolically compromised tissue to oxidative stress, pressure shifts, and metabolic instability, precipitating further injury [2,5].

In intracerebral hemorrhage (ICH), the mechanisms of deterioration differ in timing and character. END is predominantly mechanical, driven by rising intracranial pressure, mass effect, tissue displacement, and hematoma expansion [3]. Ongoing bleeding and rapid hematoma enlargement (often associated with uncontrolled systolic blood pressure) are central drivers of acute worsening. Early deterioration correlates strongly with larger hemorrhage volume, intraventricular extension, lobar location, hematoma expansion, and elevated systolic blood pressure [3], reinforcing the temporal urgency of early imaging and physiological control.

Later-phase deterioration in ICH reflects a distinct pathological profile. Delayed neurological decline is more commonly associated with perihematomal edema, systemic infection, fever, and medical complications rather than continued bleeding [3]. Inflammatory responses and progressive edema contribute substantially to secondary brain injury, indicating that neurological risk persists beyond initial stabilization and that apparent early improvement may be misleading [3].

Across stroke subtypes, several mechanisms recur with notable consistency. Cerebral edema accounts for ~21.2% of deterioration episodes, seizures for ~19.2%, and hemorrhagic transformation for ~13.0% [7]. Additional contributors include recurrent stroke (~10.2%), stroke progression (~7.5%), and clot propagation (~5.5%) [7]. Collectively, these processes define a shared biological landscape of instability underlying SND. Taken together, these findings demonstrate that early or silent neurological deterioration is rarely attributable to a single pathological event. Instead, it reflects a dynamic interaction between vascular compromise, metabolic and inflammatory stress, and systemic physiological instability. Appreciating this layered pathophysiology is essential for improving early recognition, refining multimodal monitoring strategies, and ultimately reducing preventable neurological decline.

Predictors and Risk Stratification in Acute Stroke Deterioration

Effective monitoring in the ED hinges on the early identification of patients who appear clinically stable yet remain biologically vulnerable to neurological worsening. END, most commonly defined as a measurable decline (typically an increase of ≥2 points on the NIHSS) within the first hours to days following acute ischemic stroke, represents one of the earliest manifestations of this vulnerability [2,5,7]. Importantly, END is not a random event but tends to occur in patients who already harbour identifiable markers of limited neurological reserve.

Across multiple cohorts, baseline neurological severity has consistently emerged as the strongest independent predictor of END. Higher admission NIHSS scores are associated with a substantially increased likelihood of early worsening, as well as poorer short- and long-term functional outcomes [2,5,7]. Rather than functioning solely as a measure of presenting deficit, initial severity reflects the magnitude of underlying tissue injury and the narrow margin available to accommodate secondary insults. A similar relationship is observed in ICH, where lower admission GCS scores, higher NIHSS scores, and larger hematoma volumes independently predict early deterioration and worse in-hospital

outcomes [3]. Together, these markers signal reduced physiological reserve and heightened susceptibility to dynamic pathological processes in the acute phase.

Systemic Determinants and Risk Trajectories:

Systemic physiological factors further shape the trajectory of neurological decline. Hyperglycemia at presentation is consistently associated with END and poorer functional recovery, likely reflecting metabolic stress that exacerbates ischemic injury, oxidative damage, and blood-brain barrier disruption [5]. Blood pressure dysregulation exerts a similarly powerful influence. In ICH, elevated systolic blood pressure is strongly linked to hematoma expansion and early neurological worsening, reinforcing the role of systemic instability in driving secondary brain injury [3].

Chronic comorbidities including hypertension, diabetes mellitus, chronic kidney disease, and cardiovascular disease, contribute to longer-term vulnerability and progressive cognitive and functional decline [11,14,15]. When systemic stressors coexist with covert cerebrovascular injury, they delineate a high-risk phenotype characterised by recurrent events, accelerated neurological deterioration, and increased mortality [11,12,15]. In this context, early decline represents not an isolated episode, but the early expression of an adverse longitudinal trajectory.

SBI as a Marker of Future Neurological Risk:

SBI occupies a pivotal position among predictors of future deterioration, serving as a robust marker of underlying cerebrovascular disease. Population-based studies and meta-analyses consistently demonstrate an approximately two-fold increased risk of future symptomatic stroke among individuals with SBI, independent of traditional vascular risk factors [12]. Its prevalence, detected in approximately 18-20% of stroke-free older adults, underscores both its commonality and clinical relevance [11].

Among patients presenting with symptomatic ischemic stroke, SBI is frequently observed and closely associated with established vascular risk factors such as hypertension and diabetes mellitus [14]. While these covert lesions do not directly precipitate acute neurological decline, they reflect accumulated microvascular injury and reduced cerebral resilience. As such, SBI identifies individuals predisposed to subsequent clinical strokes, hemorrhagic complications, cognitive impairment, and poorer functional outcomes, reinforcing its role as a key indicator of latent neurological risk rather than a benign incidental finding [12,15].

Radiological Biomarkers and Cerebrovascular Vulnerability:

Neuroimaging provides critical insight into the structural and physiological substrates that precede clinical decline. In acute ischemic stroke, early ischemic changes on Computed Tomography (CT) or Magnetic Resonance Imaging (MRI), including infarct extent, evolving cytotoxic edema, and perfusion deficits, often predate neurological worsening and correlate strongly with END [2,5]. These findings

indicate that deterioration frequently reflects ongoing tissue compromise rather than abrupt new injury. In ICH, radiological predictors such as baseline hematoma volume, intraventricular extension, lobar location, and hematoma expansion are robustly associated with neurological decline and mortality [3], underscoring the dynamic nature of hemorrhagic injury in the early period.

Beyond acute radiographic features, SBIs represent a distinct and under-appreciated imaging marker of cerebrovascular vulnerability. These MRI-visible infarcts occur without corresponding acute neurological symptoms but reflect cumulative vascular injury and impaired cerebral resilience [11]. SBI is independently associated with an approximately two-fold increased risk of future symptomatic stroke and has been linked to poorer outcomes, including an elevated risk of hemorrhagic transformation following thrombolysis [12,16]. Their presence therefore identifies patients in whom compensatory capacity is already diminished, rendering them more susceptible to both early deterioration and delayed complications.

Failure Modes: Atypical Presentation, Cognitive Bias, and System Overload

Diagnostic failure in the ED rarely arises from a single lapse and more commonly reflects the convergence of atypical clinical presentation, cognitive bias, and system-level pressures. These factors interact to obscure evolving neurological injury, particularly when early decline does not conform to canonical stroke patterns.

Atypical presentations represent one of the strongest drivers of missed cerebrovascular events. Patients whose strokes are misdiagnosed are disproportionately likely to present with non-specific symptoms such as headache or dizziness rather than classic focal deficits. Non-traditional presentations are associated with a marked increase in the odds of misdiagnosis, whereas strokes presenting with clear hemiplegia or aphasia are seldom overlooked. Mild, transient, or poorly localised symptoms, including isolated headache, dizziness, or temporary sensory disturbance, are particularly vulnerable to diagnostic dismissal. Reduced level of consciousness further compounds this risk, with adjusted odds ratios for missed acute ischemic stroke exceeding three in some cohorts. Posterior circulation strokes are also consistently overrepresented among missed diagnoses, reflecting the limited sensitivity of standard neurological screening tools to these presentations [10,13].

Cognitive bias amplifies the diagnostic vulnerability created by atypical symptomatology. Patients later found to have missed ischemic stroke are frequently assigned broad, non-specific alternative diagnoses, most commonly “altered mental status,” which accounted for over one-third of missed cases in a large observational study. Such diagnostic anchoring may prematurely curtail further evaluation, particularly in complex presentations where neurological findings are subtle, fluctuating, or difficult to localise. Once an alternative explanatory framework is adopted, subsequent clinical data may be unconsciously interpreted in support of that initial label rather than prompting reassessment [13].

System-level factors further shape diagnostic performance. Missed strokes occur more frequently in non-teaching and low-volume hospitals and are disproportionately observed among younger patients and women [10]. Interestingly, higher misdiagnosis rates have been reported on days with lower ED admission volumes, suggesting that variability in clinician risk tolerance and decision thresholds, rather than simple crowding alone, may influence diagnostic behaviour [10]. Together, these findings highlight that SND often emerges not from a lack of information, but from the interaction between human cognition and organisational context.

Common Pitfalls Leading to Missed Neurological Deterioration

Failure to recognise and respond to clinical deterioration during emergency care remains a major contributor to preventable adverse events [6]. Up to one in seven ED patients may experience unrecognised deterioration, and between 36% and 71% of ED adverse events are considered preventable. In the context of acute neurological disease, these failures are particularly consequential [6].

Stroke remains among the most frequently missed (and most dangerous) ED diagnoses. Diagnostic error-related deaths occur more than thirty times more often in stroke than in myocardial infarction. Large observational studies indicate that approximately 14% of patients with confirmed acute ischemic stroke presenting to, or occurring within, the ED are not initially recognised as such. These delays systematically exclude patients from time-sensitive interventions, including reperfusion therapy, and defer the initiation of secondary prevention strategies, thereby increasing the likelihood of subsequent neurological decline and long-term disability [10,13].

At a population level, the cumulative impact of these diagnostic failures is substantial. An estimated 15,000-165,000 cerebrovascular events are misdiagnosed annually in the United States of America. EDs alone, most commonly when presentations are dominated by non-specific symptoms such as headache, dizziness, or altered sensorium [10]. Collectively, these patterns illustrate that missed neurological deterioration in the ED is less a consequence of inadequate testing and more a failure of detection frameworks to capture early, dynamic, and systemically mediated neurological risk.

Structured and Validated Detection Methods:

Because most serious in-hospital adverse events are preceded by abnormalities in physiological parameters, structured detection systems are central to identifying patients at risk of deterioration in the ED [6]. Current approaches broadly fall into two categories.

Aggregate scoring systems integrate multiple physiological variables to stratify risk and guide escalation of care. Tools such as MEWS, VIEWS, REMS, and NEWS/NEWS2 have been applied across ED settings to identify patients at increased risk of adverse outcomes [6]. While these systems improve

standardisation, they primarily capture global physiological instability and are less sensitive to early, organ-specific decline. Single-parameter trigger systems, by contrast, activate predefined responses when individual thresholds are crossed. Although simple and rapid to apply, these systems lack the capacity to detect gradual, multifactorial deterioration and may fail to register evolving neurological risk until late in the clinical course [6].

Beyond formal scoring tools, several simple bedside and laboratory markers have demonstrated utility in predicting early neurological deterioration. Measures such as the Blood Urea Nitrogen/creatinine ratio and urine specific gravity illustrate how routinely available data can signal heightened vulnerability even in the absence of overt neurological worsening [4]. Despite this, such markers are inconsistently integrated into neurological risk assessment pathways.

Collectively, existing ED detection strategies remain poorly aligned with the clinical reality of silent or evolving neurological decline. Reduced level of consciousness, one of the strongest predictors of missed diagnosis, continues to challenge prevailing assessment frameworks [6,10,13]. This persistent gap suggests that current systems prioritise overt instability over latent neurological vulnerability, leaving a critical window for early intervention unaddressed.

Clinical Detection in the ED

The ED is a high-acuity, assessment-intensive environment in which timely recognition of neurological deterioration depends on the continuous interpretation of dynamic clinical signals, particularly neurological status and physiological parameters [6]. In practice, however, acute stroke assessment in the ED has become increasingly technocentric, with disproportionate reliance on neuroimaging and laboratory investigations. While these modalities are indispensable for diagnosis and triage, there is limited evidence that escalating dependence on advanced imaging alone improves the accuracy or timeliness of stroke diagnosis in real-world ED settings [10]. As a result, evolving neurological injury may remain undetected despite apparently adequate diagnostic workup.

Missed or delayed diagnosis remains a persistent feature of emergency stroke care. Approximately 14% of acute ischemic strokes are initially misdiagnosed in the ED, contributing to preventable mortality at a scale that exceeds that associated with myocardial infarction by more than thirty-fold [10,13]. These failures often reflect the limitations of standard neurological assessment, which is optimised to identify fixed focal deficits rather than subtle, evolving decline. When deterioration is driven by systemic, metabolic, or physiological instability rather than overt focal change, early warning signs may fall outside conventional neurological frameworks. For instance, systemic markers such as an elevated blood urea nitrogen/creatinine ratio (>15) have been independently associated with a markedly increased risk of early neurological deterioration, yet are rarely incorporated into routine neurological risk stratification pathways [4].

Compounding this challenge, most ED deterioration recognition systems are designed to predict downstream high-mortality outcomes, such as intensive care admission or death, rather than to identify early, potentially reversible neurological decline in real time [6]. These systems typically prioritise global physiological collapse over organ-specific vulnerability and often trigger only after neurological injury has already progressed. Substantial variability in scoring systems and thresholds across institutions further limits their consistency and applicability in acute neurological presentations [6].

Neuromonitoring Strategies for Early or SND

The primary objective of neuromonitoring in early or silent neurological deterioration is the identification of evolving cerebral dysfunction before it becomes clinically manifest [1]. Growing evidence indicates that non-invasive neuromonitoring has value not only in patients with primary neurological injury, but also in critically ill individuals without overt brain pathology, in whom secondary neurological injury may evolve covertly [1]. This recognition has driven the development of multiple bedside neuromonitoring modalities, including Electroencephalography (EEG), Point-of-care EEG (pEEG), evoked potentials, transcranial Doppler ultrasonography, automated pupillometry, optic nerve sheath diameter measurement, and near-infrared spectroscopy [1].

Although many of these technologies are now well established in perioperative and critical care environments, their penetration into ED practice remains limited. EEG-based monitoring, in particular, was initially introduced in the operating theatre to reduce intraoperative awareness but has since demonstrated broader relevance. Continuous EEG and pEEG are routinely employed for prognostication after cardiac arrest, and prolonged EEG suppression during cardiac surgery has been associated with postoperative delirium and long-term cognitive dysfunction [1]. Despite this expanding evidence base, neuromonitoring in the ED remains largely restricted to conventional clinical observation, with advanced modalities reserved for intensive care settings. This disparity persists even though many ED patients, such as those with sepsis, severe respiratory failure, polytrauma, acute stroke, or post–cardiac arrest syndrome, are at risk of silent neurological derangement despite a non-neurological primary diagnosis [1].

In contrast to neuromonitoring, ED deterioration detection continues to rely predominantly on recognition-and-response systems based on aggregate early warning scores or single-parameter physiological triggers [6]. While these tools provide a degree of standardisation, evidence supporting their effectiveness in improving real-time neurological safety in the ED is inconsistent [6]. A key limitation is that most systems are designed to predict downstream outcomes such as Intensive Care Unit admission or mortality, rather than to identify evolving neurological decline during the ED stay itself [6]. As a result, early cerebral dysfunction may progress undetected until it manifests as overt clinical deterioration.

Clinical prioritisation therefore remains critical. In acute ischemic stroke, patients with large artery atherosclerosis, anterior choroidal artery infarction, or high baseline NIHSS scores are consistently at

increased risk of early neurological deterioration and warrant particularly close observation in the initial days following presentation [2,5,7]. Similarly, patients with ICH require intensive surveillance, as neurological deterioration occurs in approximately one-third of cases, most commonly within the first 48 hours [3]. Emerging work suggests that AI-based analysis of clinical and imaging data may improve prediction of deterioration timing and risk in ICH, offering a potential avenue for earlier intervention [9].

Missed strokes in the ED further reinforce the need for improved neurological surveillance. Younger adults, women, and non-White patients, particularly those presenting with headache or dizziness, are disproportionately affected by diagnostic error [10]. In these populations, structured and vigilant neuromonitoring may help mitigate silent decline driven not by inevitable biological progression, but by delayed recognition and missed opportunities for intervention. Collectively, neuromonitoring for early or silent neurological deterioration represents a rapidly evolving field with clear potential, yet with substantial barriers to real-time implementation in the ED, where earlier detection is most likely to influence outcomes.

EEG-Based Detection and Silent Electrical Deterioration:

pEEG has expanded the scope of neurophysiological monitoring beyond traditional neurology units into EDs, intensive care units, and, increasingly, prehospital settings, enabling the detection of early or silent electrical deterioration [8]. These reduced-montage, rapid-access systems were designed to overcome the logistical and interpretive constraints of conventional EEG, allowing substantially faster deployment in time-critical environments [8]. Feasibility studies demonstrate median times to diagnostic interpretation of approximately 75 minutes in emergency contexts, with device setup achievable in as little as five minutes [8]. When combined with telemedicine support and AI-assisted interpretation, pEEG enables clinically actionable assessment even in the absence of on-site neurophysiology expertise [8].

One of the most consequential applications of EEG-based monitoring in acute care is the detection of non-convulsive status epilepticus, a common and potentially fatal cause of silent neurological decline in critically ill patients [8]. Although continuous EEG remains the reference standard for diagnosis, its resource-intensive nature limits widespread use in the ED [8]. pEEG addresses this gap by facilitating rapid seizure detection in environments where diagnostic delay may carry significant risk. While agreement between pEEG and full continuous EEG is imperfect, demonstrating only fair concordance in acute stroke populations, pEEG provides meaningful clinical value by identifying otherwise occult seizures in settings where conventional EEG is unavailable or delayed [8].

Beyond seizure detection, quantitative EEG (qEEG) markers are increasingly explored as tools for early stroke identification and triage. Because alterations in cerebral blood flow produce near-instantaneous changes in cortical electrical activity, qEEG metrics such as the brain symmetry index, phase synchronisation, and delta/alpha ratio have shown promise in detecting large vessel occlusion and

distinguishing acute ischemic stroke from normal states across both in-hospital and prehospital pathways [8]. However, EEG remains limited in its ability to reliably differentiate ischemic from hemorrhagic pathology or to distinguish acute from chronic abnormalities, constraining its use as a standalone diagnostic modality [8].

EEG also offers an objective means of identifying delirium, another form of SND that is frequently under-recognised due to reliance on subjective clinical screening tools [8]. Even single-channel, one-minute automated EEG systems have demonstrated operational potential, particularly when augmented by AI-driven analysis [8]. Distinct spectral shifts detectable on qEEG have been associated with delirium, enabling rapid bedside assessment in real time [8].

In perioperative and critical care environments, EEG and pEEG are already well integrated, with burst-suppression patterns linked to postoperative neurocognitive impairment and EEG features providing important prognostic information following cardiac arrest [1]. The limited uptake of EEG-based neuromonitoring in EDs therefore reflects not a lack of technological capability, but a translational gap between evidence and implementation. Closing this gap may be pivotal for the early detection of silent electrical deterioration and for improving neurological outcomes in high-risk ED populations [1,8].

Imaging-Based Detection of SND

The detection of subtle and clinically silent neurological injury relies heavily on advanced neuroimaging, with CT and MRI forming the foundation of contemporary stroke assessment and lesion characterisation [9]. Beyond identifying overt pathology, these modalities are increasingly central to recognising covert injury, early infarct evolution, and secondary processes that precede clinical deterioration.

Role of CT and MRI in Detecting Early Infarct Evolution, Silent Infarcts, and Micro-Injury:

Advances in imaging resolution and the widespread availability of MRI have substantially increased recognition of SBI, reframing it as a clinically meaningful marker of cerebrovascular disease rather than a benign incidental finding [11]. SBI is now firmly established as a subclinical predictor of future symptomatic stroke, conferring an approximately two-fold increased risk even after adjustment for conventional vascular risk factors [12].

On MRI, SBI is typically defined in research settings as a focal lesion ≥3 mm in diameter demonstrating hyperintensity on T2-weighted sequences [12]. Its clinical implications extend beyond stroke risk alone. Silent infarcts are strongly associated with cognitive impairment and accelerated cognitive decline, and in the absence of imaging-based detection, such impairment may be incorrectly attributed to normal aging [15]. In acute care pathways, SBI is frequently identified incidentally on baseline or

pre-thrombolysis imaging in patients presenting with an unrelated symptomatic ischemic stroke, further underscoring the continuum between silent and overt cerebrovascular disease [16].

Neuroimaging also plays a central role in identifying early infarct evolution. In acute ischemic stroke, imaging seeks to delineate the ischemic penumbra, the region of hypoperfused yet potentially salvageable tissue, whose fate ultimately determines clinical outcome [9]. CT perfusion-derived mean transit time maps are particularly useful in distinguishing benign oligemia from tissue at genuine risk of infarction [9]. Beyond macroscopic injury, machine learning approaches applied to MRI have demonstrated the capacity to detect subtle patterns of micro-injury, including diffuse changes associated with hypoxic-ischemic insult, reflecting the expanding sensitivity of imaging-based detection strategies [9].

Repeat Imaging Strategies:

Single time-point imaging may fail to capture the dynamic evolution of cerebral injury. Serial or repeat imaging therefore plays an essential role in monitoring infarct progression, cerebral edema, and secondary mass effect. Automated algorithms have been developed to quantify cerebrospinal fluid shifts on sequential CT scans, enabling objective assessment of evolving cerebral edema following large hemispheric infarction [9]. These approaches highlight the value of longitudinal imaging in detecting silent progression that may precede clinical deterioration.

AI and Computer-Aided Diagnostic Systems:

Rapid advances in computer-aided diagnosis, machine learning, and deep learning have transformed neuroimaging analysis in acute stroke care [9]. These systems aim to enhance sensitivity for ischemic lesion detection while reducing false positives and clinician workload. Deep learning architectures, including residual fully convolutional networks, have demonstrated increasing accuracy in segmenting ischemic core and penumbral regions on MRI [9].

Beyond lesion detection, AI-driven imaging tools offer workflow advantages by accelerating interpretation and prioritising critical findings, particularly in time-pressured emergency settings [9]. Their ability to identify subtle imaging changes that may be overlooked by the human eye positions them as valuable adjuncts for detecting early or SND.

Integration into ED Decision-Making:

Accurate and timely interpretation of neuroimaging is central to image-guided treatment decisions in modern stroke care [9]. The integration of AI-based imaging alerts into ED workflows represents a potential pathway for real-time risk stratification and triage. While still largely investigational,

prototype systems incorporating cloud-based and federated learning architectures suggest the feasibility of automated monitoring and alert generation for evolving injury patterns [9]. Such systems may support earlier escalation of care in patients with silent radiological progression, bridging the gap between imaging findings and clinical action.

System-Level Monitoring and Deterioration Response in the ED

System-level monitoring underpins patient safety in the ED, where recognition and management of clinical deterioration begins at triage and must continue throughout the patient's stay [6]. Although inpatient settings commonly employ formal rapid response systems, structured approaches specifically designed for post-triage monitoring and response within the ED have only recently emerged and remain less mature [6].

ED Early Warning Systems and Rapid Response Models:

Formal ED deterioration detection systems generally fall into two categories: aggregate scoring systems and single-parameter trigger systems [6]. Aggregate scores, such as the Modified Early Warning Score and the National Early Warning Score, quantify the number and severity of physiological abnormalities to generate a composite score that dictates escalation of care [6]. In contrast, single-parameter systems activate a response when any one predefined physiological threshold is breached [6].

The defining feature of any effective system is the presence of a clearly specified and timely response once deterioration criteria are met [6]. Documented responses include immediate senior medical review, mandatory bedside reassessment within defined timeframes, or department-wide alerts to mobilise clinical teams [6]. Compared with inpatient rapid response systems, ED–based response protocols are often less standardised, with greater variability in team composition, response timing, and accountability [6].

Workflow-Based Monitoring Strategies:

Ongoing, structured reassessment is critical for detecting neurological decline, yet current evidence reveals substantial gaps in ED monitoring practices [6]. Most studies assess patients only at arrival or at one or two discrete time points, rather than implementing continuous or interval-based reassessment throughout the ED stay [6]. Even when repeated physiological scores are recorded, many studies fail to describe a structured escalation pathway linked to abnormal findings [6].

As a result, many monitoring systems function primarily as predictive tools for post-ED outcomes, such as intensive care admission or mortality, rather than as real-time instruments to prevent deterioration during the ED encounter itself [6].

Challenges Unique to the ED:

The ED presents distinct barriers to effective monitoring. Accurate vital sign measurement is fundamental in this assessment-intensive environment, yet few studies specifically address optimisation of measurement processes within the ED [6]. There is also wide variability in systems used across institutions, limiting standardisation and comparability [6].

A further limitation lies in the response phase: few studies clearly define the actions taken once deterioration is identified, leaving uncertainty as to whether recognition translates into meaningful intervention [6]. Additionally, many scoring systems exclude parameters not immediately available, such as laboratory data, reflecting the time-critical nature of emergency decision-making but potentially limiting sensitivity for END [6].

Best Practices for Structured Recognition and Escalation:

Best practice recommendations emphasise that recognition systems must be paired with predefined, consistently executed response protocols [6]. The use of structured instability criteria combined with track-and-trigger charts and escalation pathways has been associated with reductions in unreported deterioration, although demonstrated outcome benefits remain modest and occasionally statistically non-significant [6].

Future system development should prioritise real-time interventional utility over retrospective risk prediction, shifting the focus toward preventing deterioration during the ED stay rather than forecasting downstream outcomes alone [6]. Further research is required to determine whether specific escalation strategies triggered by ED early warning systems translate into measurable improvements in neurological outcomes and patient safety [6].

Special Populations

In selected patient populations, neurological decline may remain clinically unapparent, leading to delayed recognition and adversely affecting prognosis and outcomes. Silent ischemic injury, multimorbidity, and demographic factors substantially influence both the development and detection of neurological deterioration [11,12,14,15]. Improving surveillance in acute and subacute stroke therefore requires an understanding of how these factors shape risk profiles and obscure clinical signals within distinct populations.

Elderly Populations:

Advanced age is one of the strongest predictors of SBI and covert neurological decline, alongside hypertension [11]. Prevalence increases sharply with age, rising from approximately 8% in individuals aged 60-64 years to 35% in those over 80 years [11]. SBI is also present in nearly one in five stroke-free older adults [12].

In elderly patients, baseline cognitive impairment, frailty, and atypical symptom presentation frequently obscure early neurological worsening [11,15]. Subtle deterioration may therefore progress unnoticed until overt clinical decline occurs, at which point therapeutic windows may already be lost.

Multimorbidity, Renal and Cardiovascular Disease:

Systemic comorbidities both increase the risk of SBI and complicate neurological assessment. Chronic kidney disease shows a strong association with SBI, identifying renal failure as a particularly vulnerable subgroup [11]. Other associated conditions include hypertension, carotid stenosis, metabolic syndrome, heart failure, coronary artery disease, hyperhomocysteinemia, and moderate-to-severe obstructive sleep apnea [11].

Recent data highlight the interaction between renal and cardiovascular dysfunction. In patients with severe aortic stenosis, SBI prevalence reached 46% [14]. Renal impairment was independently associated with SBI, with e-glomerular filtration rate emerging as a key determinant [14]. Patients with both elevated CHA_2DS_2-VASc scores (≥4) and reduced e-glomerular filtration rate (<60 mL/min/1.73 m^2) demonstrated the highest risk [14]. In such multimorbid populations, neurological decline is often masked by systemic illness, increasing the likelihood of delayed recognition and intervention.

Socioeconomic Influences:

Sociodemographic factors further shape the burden and detection of covert neurological injury. In economically disadvantaged urban populations, the prevalence of at least one silent vascular-related lesion reached 22.8% among apparently healthy elderly individuals [15]. Educational attainment also modified risk, with SBI occurring more frequently in individuals with four or fewer years of education [15]. These findings suggest that social determinants influence both the incidence of silent injury and its recognition within clinical pathways.

Integrating a Multimodal Monitoring Model for the ED

Modern emergency care requires a shift from viewing neurological deterioration as an isolated cerebral event toward a multimodal monitoring framework integrating clinical examination, neuromonitoring, neuroimaging, and systemic physiological data [1]. In patients without primary brain injury, neurological decline frequently reflects secondary insult driven by systemic failure, most commonly hypoxia, hypotension, or sepsis. In this context, systemic instability functions as an early marker of cerebral vulnerability, as the brain is often the first organ to manifest dysfunction during physiological collapse [1].

To remain operationally feasible in a high-acuity, high-throughput environment, neurological surveillance should follow a tiered structure progressing from broad screening to targeted diagnostic escalation [4]. The first tier focuses on detection of neurological drift, particularly a change of ≥2 points on the NIHSS, which is more sensitive than GCS for identifying early deterioration in ischemic

cerebrovascular disease [4]. Any new focal deficit, fluctuation in level of consciousness, or unexplained behavioural change should prompt escalation rather than reassurance.

In patients with unexplained altered mental status, bedside examination alone is frequently insufficient to detect NCSE or other forms of silent electrical deterioration. Integration of pEEG enables rapid, objective assessment of cerebral electrical activity and effectively functions as a neurological vital sign within ED workflows. Contemporary systems can be deployed by non-specialist staff within 5-10 minutes, allowing timely exclusion or confirmation of subclinical seizure activity when diagnostic delay carries substantial risk [6].

When clinical drift or EEG abnormalities are identified, targeted neuroimaging becomes the diagnostic priority. While non-contrast CT remains first line for excluding intracranial hemorrhage, AI-driven coronary artery disease tools can accelerate detection of left ventricular obstruction and evolving ischemic penumbra [8]. AI-assisted interpretation reduces inter-reader variability and mitigates delays during peak demand or in resource-limited settings, supporting timely access to advanced decision-making [8].

Activation of the multimodal protocol should occur through predefined track-and-trigger systems. Escalation is initiated not only by neurological worsening but also by early systemic warning signs, including acute changes in respiratory rate, oxygenation, or systolic blood pressure. These abnormalities frequently precede overt neurological collapse and represent a critical window for intervention [1]. Once thresholds are met, a standardised response pathway should be activated, mirroring established emergency responses such as stroke alerts or code blue systems.

The overarching objective is the development of a reproducible and scalable ED framework. Automated surveillance of systemic physiology via the electronic heart record, combined with portable tools such as pEEG, allows neurological monitoring to be standardized across diverse clinical environments. This approach reduces reliance on continuous specialist availability and establishes a safety net capable of detecting silent or evolving deterioration across the broader ED population, rather than being limited to patients with overt neurological presentations or primary brain injury [1,6,9].

Clinical Consequences and Decision-Making

Early recognition of neurological decline in the ED is the primary trigger for life-saving intervention. In patients following thrombolysis or endovascular thrombectomy, sudden deterioration often reflects collateral failure or stroke recurrence and mandates immediate reassessment [7]. Hemorrhagic transformation requires prompt cessation of antithrombotic therapy to limit secondary injury [2]. In contrast, thrombus extension or migration may necessitate rescue thrombectomy or alternative reperfusion strategies [2].

In ICH, early identification of silent decline is critical to enable blood pressure control and limit hematoma expansion. Intensive blood pressure reduction following INTERACT-2 principles mitigates

secondary injury [3]. Timely administration of reversal agents, including tranexamic acid as studied in TICH-2, aims to arrest hematoma growth before irreversible damage occurs [3].

END is a major determinant of long-term functional outcome as measured by modified Rankin score at 3 months [5]. Beyond prognostication, deterioration drives immediate changes in management. Cerebral edema necessitates escalation from standard monitoring to aggressive measures such as osmotic therapy or decompressive hemicraniectomy [2]. Failure to detect early decline eliminates critical intervention windows, worsening both functional and survival outcomes [2].

Timely intervention depends on standardised documentation and serial communication. Patients require frequent neurological reassessment to distinguish early from delayed deterioration [3]. Clinically significant decline is documented using validated thresholds, including a ≥2-point increase in NIHSS for ischemic stroke [2,5], and a ≥4-point NIHSS increase or ≥2-point decrease in GCS for ICH [3]. Consistent recording of these changes should trigger repeat diagnostic evaluation to identify secondary vascular, infectious, or metabolic causes [5].

Future Directions and Limitations

AI-Enhanced Neurological Monitoring:

AI has substantial potential to improve early detection, diagnostic precision, and workflow efficiency for SND in the ED. Applications include AI-assisted pEEG, AI in imaging, and Multimodal monitoring. AI-assisted pEEG in automated spectral analysis and processed EEG metrics (Delta/Alpha ratio, Brain Symmetry Index) enable rapid detection of subclinical conditions such as Non-convulsive status epilepticus, delirium, and acute ischemic stroke, reducing time-to-EEG and supporting non-specialist staff. AI in imaging consists of machine learning and deep learning applied to CT and MRI can identify subtle lesions, estimate ischemic penumbra, and predict outcomes like infarct volume or hemorrhagic transformation, complementing targeted neuroimaging and AI-augmented interpretation described earlier. Multimodal monitoring with integration of pEEG, pupillometry, and near-infrared spectrum expands surveillance to patients with systemic illness, where neurological decline may otherwise go unnoticed, aligning with the tiered monitoring model for the ED.

These AI-driven approaches reinforce and extend previously discussed concepts, including early recognition of END, multimodal monitoring, and the value of repeat or targeted imaging, offering more rapid and objective detection.

Limitations and Challenges

Heterogeneity of studies: Variability in definitions, scales, thresholds, and timing of assessments limits comparability and complicates the evaluation of END and SND.

Mixed endpoints: Differences in outcome measures, physiological change, ICU admission, mortality, affect interpretation of monitoring system performance.

AI evaluation variability: Performance metrics and imaging modalities are inconsistent, complicating synthesis and real-world applicability.

These limitations underscore the need for standardised monitoring frameworks, integrating clinical, neuromonitoring, and imaging data with predefined escalation pathways. Such standardisation would ensure reliable detection, prognostication, and timely intervention, bridging gaps identified in prior sections of ED neurological monitoring and system-level response.

Conclusion:

Silent neurological deterioration represents a significant and underrecognized challenge in emergency and acute care. Its detection is hindered by atypical presentations, cognitive biases, systemic overload, and limitations in current ED workflows. Patients with subtle or non-specific symptoms, multimorbidity, or SBIs are particularly vulnerable, with delayed recognition directly impacting prognosis, treatment opportunities, and long-term functional outcomes. Early identification remains the cornerstone of improving patient safety and optimizing acute interventions.

Integration of multimodal monitoring strategies, combining clinical assessment, EEG-based surveillance, advanced neuroimaging, and AI-assisted tools, offers a promising approach to bridge existing gaps. pEEG, rapid CT/MRI interpretation, and AI-driven alert systems enable timely recognition of silent electrical or radiological deterioration, even in resource-constrained or high-acuity ED environments. Tiered frameworks that escalate care based on both neurological drift and systemic warning signs may standardize detection, reduce reliance on continuous specialist availability, and provide an operational safety net across the broader patient population.

Future efforts should focus on standardizing definitions, thresholds, and monitoring protocols, while evaluating the real-world impact of AI-enhanced, multimodal systems on clinical outcomes. Addressing barriers such as workflow integration, interpretive variability, and resource limitations will be critical to translating technological advances into meaningful improvements in patient care. Ultimately, proactive surveillance of SND has the potential to reduce missed diagnoses, optimize therapeutic windows, and improve both survival and functional outcomes in vulnerable ED populations.

References:

1. Battaglini D, Pelosi P, Robba C. The importance of neuromonitoring in non-brain-injured critically ill patients: a narrative review. *Crit Care*. 2022;26:78. doi:10.1186/s13054-022-03914-4.
2. Geng HH, Wang Q, Li B, Cui BB, Jin YP, Fu RL, Zhang Q, Wang JJ, Wang PX. Early neurological deterioration during the acute phase as a predictor of long-term outcome after first-ever ischemic stroke. *Medicine (Baltimore)*. 2017;96(51):e9068. doi:10.1097/MD.0000000000009068.
3. Cuadrado-Godia E. Early neurological deterioration—easy methods to detect it. *Indian J Med Res*. 2015;141(3):266–268. doi:10.4103/0971-5916.159260.
4. Li J, Zhang W, Wang Y, Tang T, Wu D, Wei Z, Liu Y. Neurological deterioration in intracerebral hemorrhage. *Front Neurol*. 2022;13:937550. doi:10.3389/fneur.2022.937550.
5. Aasebø W, Ellekjær H, Alstad M, Vangberg T, Ihle-Hansen H, Salvesen Ø. Mechanisms, predictors and clinical impact of early neurological deterioration after acute ischemic stroke. *BMC Neurol*. 2014;14:201. doi:10.1186/s12883-014-0201-4.
6. Edwards MJ, Jacobs IG, Jelinek GA, et al. Systems for recognition and response to deteriorating patients in the emergency department: a scoping review. *Scand J Trauma Resusc Emerg Med*. 2021;29:157. doi:10.1186/s13049-021-00882-6.
7. Nunn E, Glynn L, McKevitt C, et al. Early neurological deterioration following acute stroke. *Front Stroke*. 2025;2:1518685. doi:10.3389/fstro.2025.1518685.
8. Smith L, Nguyen DT, Jones M, et al. Point-of-care electroencephalography (POC-EEG) in acute neurological care—operational feasibility and evidence. *Brain Sci*. 2023;17(4):48. doi:10.3390/brainsci17040048.
9. Yang W, Li J, Xiong B, et al. A review on computer-aided diagnosis of acute brain imaging (CT/MRI) in emergency settings. *Sensors (Basel)*. 2021;21(24):8507. doi:10.3390/s21248507.
10. Tarnutzer AA, Lee SH, Robinson KA, Wang Z, Edlow JA, Newman-Toker DE. Missed diagnosis of stroke in the emergency department: a systematic review and meta-analysis. *BMJ Open*. 2017;7:e016626. doi:10.1136/bmjopen-2017-016626.
11. Vermeer SE, Longstreth WT Jr, Koudstaal PJ. The epidemiology of silent brain infarction: a systematic review. *Lancet Neurol*. 2007;6(7):611–619. doi:10.1016/S1474-4422(07)70170-9.
12. Howard VJ, Kleindorfer DO, Judd SE, et al. Silent brain infarction and risk of future stroke: a systematic review and meta-analysis. *Neurology*. 2015;84(5):495–501. doi:10.1212/WNL.0000000000001212.
13. Kleindorfer D, Lindsell CJ, Broderick J, et al. Potentially missed diagnosis of ischemic stroke in the emergency department in the Greater Cincinnati/Northern Kentucky Stroke Study. *Stroke*. 2008;39(6):1891–1896. doi:10.1161/STROKEAHA.107.509885.
14. Lee KB, Kim BJ, Choi JY, et al. Prevalence and risk factors of silent brain infarction in patients with non-cardioembolic ischemic stroke: an MRI study. *Acta Neurol Scand*. 2020;141(4):279–286. doi:10.1111/ane.13219.
15. Yamada Y, Yamauchi H, Tobita Y, et al. High frequency of silent brain infarcts associated with cognitive impairment: an MRI study. *Geriatr Gerontol Int*. 2017;17(8):1280–1287. doi:10.1111/ggi.12844.
16. Xie X, Zhang J, Liu J, et al. Effects of silent brain infarction on hemorrhagic transformation and outcomes after thrombolysis. *Front Neurol*. 2023;14:10212719. doi:10.3389/fneur.2023.10212719.