Improved characterization/classification of TBI
The role of Clinical assessment of on days 1 through 14

The Clinical Assessment Work Group

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Purpose of acute clinical assessment:
While the justification for recording variables is based on prognostic import, the primary role of clinical assessment is to drive triage, establish a diagnosis oftraumatic brain injury (TBI), optimise resuscitation to minimise secondary injury, trigger diagnostic interventions (biomarkers, CT, MRI), allocate patients to clinical pathways (discharge from the Emergency Department [ED], or admission to a Hospital Ward or Intensive Care Unit (ICU), and determine follow up.

This document therefore starts with assuming that a diagnosis of TBI has been made based on any standard definition – e.g., the National Institute of Neurological Diseases and Stroke (NINDS) definition: TBI is defined as an alteration in brain function, or other evidence of brain pathology, caused by an external force.[1] Even though some of the tools for characterization (clinical features, in this document) are used to show "alteration in brain function, or other evidence of brain pathology" – the purpose of the discussion in this document is not to address the **diagnostic** aspects, but to better characterize an established TBI.

We should recognise, however, that, following the clinical characterization recommended here, some patients may still only have a diagnosis of "suspected TBI" after head injury, which requires further confirmation by other means – such as biomarkers and/or neuroimaging.[2] These are traditionally the less severely injured cohort who present with a Glasgow Coma Score (GCS)[3] of 15 and have not been deemed to need computed tomography (CT) scanning based on accepted decision rules,[4] or are in in the "CT negative" category after CT imaging.

Such diagnostic challenges often arise when it is difficult to confirm whether loss of consciousness and/or amnesia had actually occurred at the time of injury (classically in the following cohorts: patients with pre-injury cognitive impairment, young children), or whether any amnesia that is documented is not conclusively attributable to TBI (such as in patients with pre-injury intoxication). Our document does not address these **diagnostic** challenges, but diagnosis in such patients may be clarified by further neuroimaging or by the results of blood biomarker measurement – both of which are discussed in other parts of the Reclassification symposium

Recommendations

These recommendations specifically apply to patients who present to hospital with a TBI. The Working Group recommends that, as part of a **CBI (clinical, biomarker, imaging)** characterization of TBI, the clinical characterization of these patients is best considered in the following categories:

[i] Summary:

The following must be noted as a summary description of injury severity:

- Full Glasgow Coma Score (GCS; separately specifying motor, verbal, and eye components)
  - For optimal prognostic impact and consistency, GCS is ideally be recorded at Emergency Room arrival.
  - Confounds for GCS assessment (e.g., alcohol, sedation, intubation) should be explicitly noted.
  - If a GCS component is untestable, specify this - do not score this as "1" (consider imputation in research settings).
- In all patients, but particularly in patients with a GCS $\leq$12, pupillary responses must be recorded:
  - This is best done independently of the GCS.
  - There is insufficient evidence to recommend an integrated GCS-pupillary (GCS-P) score (scored as 1-15)
  - Use automated pupillometry whenever possible.
- In all patients with a GCS verbal score $\geq$ 4 in the Emergency Room
  - Assess Post Traumatic Amnesia (PTA) using a validated tool.
  - Acute symptoms should be documented, ideally using standardized rating scales.

[ii] Expanded:

These items are important variables in TBI characterization, and should be included in the medical notes

Injury factors: A more complete assessment of patients should include text entries about:

- Injury mechanism and impact velocity, and any impact mitigation (seat belts, airbags, helmets, etc).
- In patients with a GCS verbal score $\geq$ 4, record if there is history of Loss of Consciousness (LoC).
- Extracranial injuries (specifying injuries that would, in isolation, mandate admission even without a TBI).
- Early physiological insults, using consensus thresholds (e.g. from TQIP) till definitive evidence is available.
- Post-traumatic amnesia duration, ideally determined by prospective serial assessment with a validated tool – with a record of the assessment point (e.g., ED arrival, ICU discharge) and time post-injury

Biosychosocial-ecological vulnerabilities: These may modulate disease course and outcome:

- Physical and psychological comorbidities (either listed or using a suitable scale).
- Listing of relevant therapies (specifically mentioning treatments that affect haemostasis).
- Age and Frailty (using a clinical frailty assessment scale as a minimum).
- Socioeconomic status, educational attainment, and employment status *(not addressed by our WG).*

Dynamic: Published data suggest that dynamics of clinical change can refine prognostication:

- For hospitalised patients, record neuroworsening (based on GCS, pupillary reactivity, and neurological examination), and physiological monitoring and therapy intensity over the first 7-14 days.
- Ongoing assessment of symptom severity over 7-14 days post-injury, at least for patients who are not admitted to hospital.

[iii] Emerging:

There is emerging evidence for these assessments but additional evaluation may be needed:

- Detailed assessment of neurological deficits, including vestibulo-oculomotor dysfunction, primarily for less severe TBI.
- Assessment of cognition with standardized objective tests in the Emergency Department or soon after. There is insufficient evidence to recommend a specific platform/instrument for cognitive assessment.
- Assessment of mental health symptoms 7-14 days after injury, using validated scales
- The use of data-driven decision support tools that integrate admission, dynamic, and imputed data.

Justification for recommendations

[A] Neurological status: There are several tools for assessing neurological status, but the Glasgow Coma Score (GCS)[3] is most widely used across the TBI severity spectrum. The GCS has been traditionally trichotomized as mild (GCS 13-15), moderate (GCS 9-12), or severe (GCS ≤ 8) TBI.[5 6 7] However, current use of GCS (particularly trichotiomisation) has flaws. We need to use the GCS better and require additional assessment tools:

1. There is considerable variability in injury severity, treatment needs, and prognosis within each of these three crude bands – and such information is better communicated by stating the actual GCS, ideally specifying subcomponent contributions, since these have different clinical and prognostic relevance.

2. Patients with identical GCS score may have widely different severity, treatment needs, and prognosis. The GCS has significant floor/ceiling properties, and imprecision is worst at extremes (sum scores of 3 and 15). Further, the application of the GCS requires modification in preverbal children,[8] and can be affected by pre-injury cognitive deficits.

3. It is also critical to ensure that the procedures used to score the GCS follow standard approaches[3] (see also https://www.glasgowcomascale.org/ for details and instruction videos). Careful and consistent attention to detail is essential – for example, when the motor component is being scored, this is typically done in the upper extremities, with the response of the better arm recorded as the motor response.

4. Conventional clinical assessment works to identify the need for CT brain imaging, early neurosurgery or critical care interventions, and appropriate post-ED clinical pathway selection. This approach safely detects neurosurgical lesions or pathology requiring other immediate attention.[9] However, most patients present with normal or minimally impaired consciousness and a "normal CT". The prevailing wisdom is that this makes "clinically significant" TBI highly unlikely, and specific follow up and rehabilitation is unnecessary. We now know that this view is unduly optimistic: many of this "mild" group who present to Level 1 Trauma Centres and meet thresholds for CT imaging suffer ongoing disabling symptoms.[10]

5. Conversely, patients who present with a lower GCS may be subject to therapeutic nihilism and premature withdrawal of life-sustaining therapy (WLST),[11 12] despite the fact that GCS assessment at presentation (particularly in patients with a sum GCS of 3) may be falsely lowered by alcohol, recreational drugs, sedative medication or tracheal intubation; or confounded by a post-ictal state or systemic physiological derangements. Additional assessment tools are needed to address these confounds.

6. Pupillary responses provide a strong clinical and prognostic biomarker,[13] suggesting brainstem compression due to a space occupying lesion, and while peripheral ocular or cranial nerve injury must be excluded, they have strong prognostic import. Recent discussions in this area cover the following issues:
   a. It can be difficult to robustly diagnose pupillary unreactivity, especially when pupils are small, and automated pupillometry may provide more consistent assessment.[14]

b. It has been suggested[15] that subtracting a point from the GCS for each unreactive pupil could generate an integrated "GCS-Pupils score (GCS-P)"
    i. This provides characterisation of patients below a conventional GCS floor of 3, where patients lose an additional point for each unreactive pupil. Thus, patients with one unreactive pupil would be scored as GCS-P = 2, and those with both unreactive pupils scored as GCS-P = 1.
    ii. While conceptually appealing, there are practical difficulties with this approach: At a GCS $\geq$3, a summary GCS-P score is necessarily ambiguous about whether points are lost for pupils or another GCS components.
    iii. This ambiguity could be resolved by specifying pupillary reactions as a GCS component (e.g., M2V1E1P1) or recording the pupillary reactivity separately.

7. Timing and confounding factors: These clinical assessments may vary significantly in the immediate post-injury period (either improvement after the initial ictus or neuroworsening) which makes them subject to substantial variability depending on when they are measured. Furthermore, early data may rely on first responders who are not experienced in assessing levels of consciousness in a reliable and reproducible way. Thus, reliable data are often missing from early on in the patient's course. The best initial GCS might reasonably be expected to most faithfully capture the severity of the initial injury. However, it may also be confounded by hypoxia or hypotension but the concept of 'best resuscitated' GCS is not relevant to modern prehospital practice where resuscitation and stabilisation may all occur concurrently and require sedation and endotracheal intubation. Therefore, whilst later assessments of GCS by more experienced practitioners might be expected to be more complete, for the more severely injured patients these might not be fully assessable as these patients would likely be intubated, sedated, paralysed and mechanically ventilated.
    a. We are therefore faced with how to best use GCS/pupillary responses assessed at a variety of timepoints, many of which might be missing. Imputation strategies have been examined using data from the CENTER-TBI dataset.[16] Where we need to choose a 'most predictive' neurological assessment from variably missing data, a substitution strategy is needed but there are a variety of possible choices as to which GCS/pupillary assessment to choose. Model performance (in terms of pseudo-R2 explained 'pseudovariance') varies somewhat with both substitution strategy and type of model (e.g., dichotomous vs ordinal regression) and so the choice is not an entirely trivial one.
    b. One approach to the confound of tracheal intubation has been to limit assessment to the motor subscore (which has strong prognostic import in patients with GCS 3-12).[17] However, broadly speaking, the GCS sum performs better than the motor score alone overall, so there is a case for exploring alternative strategies.
    c. Of a variety of choices, the strategy used by the International Mission for Prognosis and Analysis of Clinical Trials in TBI (IMPACT)[17] generally works well and is simple: the ED discharge assessment GCS / pupil assessment is used. If this is missing, this is substituted with the next available value looking back in time (i.e.: ED discharge —> study hospital ED arrival —> referring hospital ED arrival —> prehospital).[16]
    d. Where a GCS component is untestable, several options are available, but the amount of detail that can be obtained is always constrained by the burden of data recording:
        i. In the past un-assessed motor and verbal scores have been recorded as 1 – but this is not optimal.[16]
        ii. However, an alternative,[3] which we would recommend, is not to score it as 1, but to specify this, and amend notation to this effect (e.g., "V(t)" in an intubated patient). If a complex notation system is to be avoided, perhaps all untestable components could be specified

with a single notation (as either "T" or "U": e.g., V(T)), which in subsequent group level research analyses could identify scores for imputation (see iv, below).

iii. Less binary confounds (e.g., sedation, a recent seizure) may allow some scoring, and it is not clear what the best approach should be.

iv. In research settings, well justified imputation may be the best option.

v. It is unclear which of these strategies is optimal, and the best options may vary depending on whether the goal is individual TBI characterization in clinical practice, or analysis of a research dataset.

Alternative severity classification and diagnostic schemes (e.g., Veterans Association /Department of Defense,[18] Mayo Classification System for TBI Severity[19] FOUR Score,[20] or American Congress of Rehabilitation Medicine,[2] World Health Organisation,[21] Centre for Disease Control TBI surveillance criteria[22]) are not suitable approaches for characterizing TBI across the severity spectrum in the acute phase. Similarly, while the presence or absence of loss of consciousness (LoC) is often recorded, there is inconsistent evidence that this binary variable provides prognostic information, so we would not recommend it as essential.[23,24] The AVPU (Alert, responds to Voice, responds to Pain, Unresponsive) scale is commonly used in prehospital and emergency care and provides a more nuanced, but still unitary measure of consciousness. While the AVPU score does correlate with the GCS, very few studies have examined its use in early TBI assessment.[25]

8. The presence and duration of post-traumatic amnesia (PTA; primarily anterograde) are both prognostic when assessed prospectively with validated tools,[26] such as the Galveston Orientation and Amnesia Test, Westmead PTA Scale, or O-Log. However, this is not always feasible in in the emergency setting and may be confounded (e.g., by analgesics).[27] Retrospective assessment of PTA is relatively unreliable,[28] but is likely more useful than no information about PTA. While the combination of a GCS of 15 with persisting PTA is usually transient in milder TBI, a notation that specifies whether PTA is present at a particular timepoint (e.g., ICU or hospital discharge) is useful, alongside a clear record of the interval between injury and assessment (e.g., *ICU Discharge (Day 14 post-TBI): GCS 15*(*PTA+)*). Studies of PTA thresholds in predicting outcome use varied methodology, but PTA <24 hours is generally classed as mild; 1-7 days as moderate; 1-4 weeks as severe; and >4 weeks as very severe.[26] More objectively, PTA <24 hours (usually applied in the context of less severe injury) predicts return to employment, while PTA >56 days predicts <10% Good Outcome on the GOSE at one year post-injury.[26]

9. Detailed evaluation of neurological function (e.g., lateralising weakness, language deficits, ocular dysfunction, cerebellar signs) are recommended as part of a comprehensive clinical examination,[29] and their ongoing monitoring can inform diagnosis and management decisions (see "*neuroworsening*", later). However, these assessments are not systematically and consistently performed or documented in the acute care setting in the ED. It is very likely that at least some of these assessments are important in the early characterisation of TBI, but the systematic incorporation of these assessments into clinical care, and recommending the best format for recording their findings, will require further evaluation of their sensitivity, specificity, reliability, and robustness in the ED setting.

[B] Mechanisms of injury: Low energy transfer mechanisms (e.g. falls from a standing height or < 2 metres in adults) are conventionally expected to result in less severe injury than high velocity injury (e.g. road traffic collisions, falls from a greater height), and information on injury mechanisms should be routinely recorded.[30] However, even low energy transfer incidents (especially falls in infants and older patients) can cause significant injury that is under-estimated by conventional clinical assessment, results

in under-triage and inadequate or delayed investigation or treatment.[31] Assessment of the mechanism of injury is usefully supplemented by data regarding mitigating protection (seat belts, airbags, helmets).

[C] TBI symptoms: Traditional indicators of neurological status such as GCS exhibit minimal variability and ceiling effects. Prospective cohort studies in non-hospitalized TBI have considered a broader range of clinical variables available in the Emergency Department and found that most have limited prognostic value.[32,33,34,35] Emergency Department assessment of symptoms (e.g., headache, dizziness, sensitivity to noise) using validated scales (e.g., Rivermead Post Concussion Symptoms Questionnaire for adults and Health Behavior Inventory for children) may be one exception, with some evidence in both children[36,37] and adults.[35,38,39] There is more robust evidence for the prognostic utility of acute symptom assessment following sport-related concussion,[40] which could be reasonably extrapolated. The evidence that symptoms are prognostic primarily applies to patients with a GCS of 15 (or a verbal score of 5) – but it is reasonable to record symptoms in patients with a GCS verbal score of 4.

[D] Objective assessment of cognition, balance, and vestibulo-oculomotor dysfunction: There is emerging evidence that assessing cognition with standardized objective tests in the Emergency Department or soon after may further refine prognosis of non-hospitalized TBI.[41 42 43 44 45 46 47] Instruments to quantify vestibulo-oculomotor dysfunction include the VOMS [48] and EyeBOX®[49] are available, but the role of such assessments remains to be established outside of sport-related concussion.[50,51]

[E] Extracranial injury: Compared to isolated TBI, polytrauma is associated with a higher risk of moderate disability and severe disability/death, at both 3 and 6 months.[52 53] These worse outcomes may be due to the injury itself, a higher risk of early hypoxia and hypotension,[54] an aggravated detrimental host response,[55] and/or the effects of anaesthesia and surgery needed for extracranial injuries.[56] These considerations mandate a systems-based tertiary trauma assessment in all TBI patients. A range of trauma severity assessment tools have been used in this context, but the Abbreviated Injury Score (AIS)[57] is probably most widely used. Both the head AIS and the Injury Severity Score (the sum squares of AIS scores in the three most severely injured regions) may be of some prognostic value in TBI,[58] but an AIS $\geq$ 3 in any individual extracranial region also provides a convenient and pragmatic threshold for identifying extracranial injuries that are of relevance in the integrated characterization of multiple trauma that includes TBI, in registries and research studies.[52] If a formal assessment of AIS is thought to be less practicable for routine clinical evaluation, a useful approximation may be to record any injury that would, in isolation, have required hospital admission.[52] An integrated assessment of the severity of TBI and extracranial injury provides the best basis to plan extracranial surgery (balancing the risks and benefits of early definitive treatment against the risks of perioperative physiological compromise in a vulnerable brain). Such an assessment also allows for rational planning of follow up and rehabilitation.

[F] Early physiological Insults: Hypoxia, hypotension, hypothermia, and fever at presentation have all been associated with worse outcomes in TBI, and their presence should be recorded in any complete clinical characterisation of TBI. However, the most appropriate thresholds for identifying these insults are still not clear and the field is still evolving. For example, TCDB data suggested a systolic blood pressure (SBP) threshold of 90 mmHg,[59] but more recent publications suggest a higher thresholds,[60] or a U-shaped association with outcome, with SBP of 130-149 being optimal.[61] Similarly, while TCDB data focused on hypoxia,[59] there increasing exploration of hyperoxia as a risk factor.[62] We recommend relatively conservative consensus-based thresholds (such as those identified by the Amer8can College of Surgeons Trauma Quality Improvement Program (TQIP) Guidelines[63]) until definitive data emerge on this. While hypoglycaemia, hyperglycaemia, and hyponatremia represent additional important metabolic insults and are often available at the time of ED assessment in patients, they are not part of clinical assessment, and are hence not covered here.

[G] Age, comorbidities, and frailty: Age is among the strongest outcome predictors in TBI, with mortality and unfavourable outcome increasing continuously with age through adulthood.[64,65] This may be due to reduced physical or neurological reserve, and/or the presence of comorbid disease, which is often (though not exclusively) associated with ageing. The exception of these trends is in children, where infants have a higher mortality rate than older children,[66] and other outcomes have complex relationships with age.[67] While such knowledge should inform how we counsel patients and families about prognosis and the benefits of aggressive therapy, we need to avoid a nihilistic response to TBI management in all elderly patients, since such nihilism may (in itself) contribute to inconsistent WLST[10 11] and poor outcomes.[68] Indeed, even in an ICU setting, a significant proportion of such older patients may achieve a favourable recovery with appropriate therapy.[69] More refined approaches are needed to assess the impact of age and pre-existing disease:

1. Refine age-related vulnerability by recording **frailty**, a term used in both in adults and children, which quantifies loss of physiological and cognitive reserve, and may increase vulnerability to the stress of trauma.
2. Frailty scales may be based on the presence of comorbidities (such as: the Charlson Comorbidity Index [CCI], which can be reliably abstracted from electronic patient records;[70] the 70-item Canadian Study of Health and Aging [CHSA] Frailty Index;[71] the modified 5- and 11-item Frailty Index [mFI-5 and mFI-11],[72] and a five-item Paediatric Frailty Scale.[73]
3. The mFI-5 and mFI-11 are associated with worse outcome in TBI,[74,75] and a novel 30-item scale was also associated with worse TBI outcome in the CENTER-TBI and TRACK-TBI studies.[76]
4. While these scales clearly have research relevance, they may be difficult to implement in practice. Global clinical assessments, such as the Canadian Study of Health and Aging (CSHA) Clinical Frailty Scale (CFS),[77] associate with outcome, with thresholds scores of ≥ 4[78] or ≥ 3[79] on the 9 point CFS associated with a ~90-95% risk of death or severe disability. The CFS may provide a more pragmatic option for recording frailty in the context of clinical TBI management.
5. The discussion above primarily focuses on physical comorbidities and systemic physiological reserve, both of which have been shown to be important in modulating TBI outcome.[80] These scales also address pre-injury neurological status, but only in the context of established diagnoses. Current assessments do not address cognitive reserve or psychological health – both of which can be critical determinants of TBI outcome (and are covered by another Working Group). We need better means of quantifying the impact of these factors.
6. In young children, early recovery may be excellent, but children who sustain a TBI and appear to recover fully may be on a different developmental trajectory from their uninjured peers, and disabilities may only manifest years after the injury.[81] It is unclear whether initial assessment tools can identify children most at risk of such adverse late outcomes, but research in this area is needed.

[H] Concurrent therapy: It is critical that a full characterisation of acute TBI also records pre-injury therapies that are of relevance. While several drugs may be relevant in this context, anticoagulants and antiplatelet agents are best recognised.[82]

[I] Additional information over the first 2 weeks post injury: TBI pathophysiology evolves over time, and incorporating additional clinical information over the initial course provides improved selection of patients for acute therapy and follow up and refines late (months to years) prognostication. The ways in which such dynamic information is collected will depend on TBI severity and care path.

For non-hospitalized TBI: Assessing post-TBI symptom severity (using the Rivermead Post Concussion Symptoms questionnaire or comparable instruments) up to 14 days after injury has been repeatedly shown to refine prognosis,[83,84,85] likely above and beyond acute symptom severity. Several studies additionally measured mental health symptoms using validated self-report scales, designed to quantify

symptoms of depression (e.g., PHQ-9),[86] anxiety (e.g., GAD-7),[87] and/or post-traumatic stress (e.g., PCL-5),[88] and found that these scales explained unique variance in outcome from non-hospitalized TBI.[89 90 91] Symptom assessment could also inform the need for repeat biomarkers, further follow up, MRI, or inclusion in trials (Fig 1). For logistic reasons, attempts have been made to identify, at presentation, patients particularly high risk of persistent symptoms for such follow up,[84] but this remains an imperfect process.

For hospitalized TBI, ongoing assessment of neurological status, intracranial and systemic physiology, and therapy requirements provides important information for characterizing TBI and informing prognostication. Specific items include:

- Clinical neuroworsening (drop in GCS, progression of neurological deficit, development of a new neurological deficit, or new pupillary abnormality) is important for both prognosis and therapy.[92]
- Monitoring of systemic physiology, intracranial pressure, and brain oxygenation and metabolism, and charting of therapy intensity level and response.[93 94] It is likely that, in the future, these complex data can be usefully integrated and synthesised using novel data science approaches (including machine learning and artificial intelligence) to provide decision support tools that allow more individualized and precise management and prognostication. This will be covered by a briefing report on data Integration and interpretation, being developed separately by Ferguson et al, with a plan for submission as an ancillary publication.
- Daily assessment of post-traumatic amnesia, using the GOAT, WPTA Scale, or O-Log[26] in the period of emergence following hospitalised TBI can improve prognostication over and above the initial GCS score.[95 96 97]

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Briefing on sport-related concussion grading systems for the NIH TBI Classification Workshop
Mikolic, A., Silverberg, N.D., & Giza, C., on behalf of The Clinical Assessment Work Group
December 20, 2023

Several classification systems have been developed to grade the severity of sport-related concussions (SRC). The most widely cited include the (now retired) American Academy of Neurology (AAN) guidelines[1], the Cantu grading system for concussion[2]/ Evidence-Based modified Cantu Grading System for Concussion[3], and the Colorado Medical Society Grading System for Concussion[4]. These grading systems are summarized in Table 1. A number of other similar SRC severity grading systems have also been proposed[2 5-9]. None were universally accepted,[10] with specific concerns about insufficient empirically support.[11 12] For instance, loss of consciousness and post-traumatic amnesia duration did not strongly predict recovery time in SRC samples (where these predictors have skewed and restricted distributions).[13-15] In addition, SRC severity graded systems were not widely adopted for concussions occurring outside of sport, as their components (e.g., post-traumatic amnesia) were typically not prospectively assessed and documented.[16]

Table 1. Examples of sport-related concussion severity grading systems.

Concussion severity	**AAN[1] (1997)**	**Modified Cantu[17] (2001)**	**Colorado[4] (1991)**
Grade 1	Transient confusion without LOC, symptoms or change in mental status <15 mins	No LOC, PTA < 30 min	Transient confusion ≤ 15 min, without PTA, no LOC
Grade 2	Transient confusion, no LOC, symptoms or change in mental status > 15 mins	Brief LOC ≤1 min; PTA (retrograde or anterograde) or post concussion signs or symptoms > 30 min but < 24 h	Transient confusion and PTA > 15 min, no LOC, any symptoms > one hour requires medical observation
Grade 3	Any LOC, brief (seconds) or prolonged (minutes)	LOC > 1 min or PTA (retrograde or anterograde) > 24 h; post concussion signs or symptoms > 7 days	Evidence of LOC which is brief (seconds) or prolonged (minutes)

The 1st International Conference on Concussion in Sport[18] in 2001 "recognized the strengths and weaknesses" of existing SRC severity grading systems but decided not to endorse any of them. Instead, the group recommended characterizing injury severity and prognosis with a sideline evaluation of signs and symptoms as well as with acute neuropsychological testing.[18] This protocol was based on the Canadian Academy of Sport Medicine guideline.[19] At the 2nd International Conference on Concussion in Sport held in 2004,[20] a new severity classification into "simple" and "complex" was introduced. A simple concussion was defined as an injury that resolves without complication over 7-10 days, whereas a complex concussion was defined by prolonged LOC, cognitive impairment, or symptoms. [16] This classification was later criticized due to its retrospective nature and inability to accurately predict the course of recovery at the time of injury[21]. At the 3rd International Conference on Concussion in Sport

held in 2008,[22] the expert panel voted unanimously to abandon the simple-complex classification because "the terminology itself did not fully describe the entities." Instead, the panel advocated for an acute multimodal assessment of signs, symptoms, behavior, balance, and cognition using the standardized Sport Concussion Assessment Tool (SCAT2). The 2013 American Academy of Neurology practice parameter on SRC and 2014 National Athletic Trainers' Association position statement on SRC[23] similarly advocated for a multimodal evaluation for individualized injury characterization rather than an ordinal classification of overall SRC severity. The most recent consensus statement from the Concussion in Sport Group meeting in 2022[24] continued this trend and provided updated standardized assessment tools.

In summary, no SRC severity graded system is currently in use, likely because their prognostic utility has not been demonstrated. They were reviewed here to provide historical context for the NIH TBI Classification and Nomenclature Workshop. We infer that classifying SRC severity on the basis of loss of consciousness and post-traumatic amnesia duration is unhelpful. Expert consensus now favors individualized characterization of SRC-related symptoms and impairments. Certain acute symptoms and examination findings (e.g., balance test score) have been incorporated into recent multivariable prognostic models[25 26], which may be more useful for identifying athletes at risk of prolonged recovery.

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