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PERSPECTIVE

An initial Glasgow Coma Scale score of 8 or less does not define severe brain injury

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Abstract

The wide-spread use of an initial 'Glasgow Coma Scale (GCS) 8 or less' to define and dichotomise 'severe' from 'mild' or 'moderate' traumatic brain injury (TBI) is an out-dated research heuristic that has become an epidemiological convenience transfixing clinical care. Triaging based on GCS can delay the care of patients who have rapidly evolving injuries. Sole reliance on the initial GCS can therefore provide a false sense of security to caregivers and fail to provide timely care for patients presenting with GCS greater than 8. Nearly 50 years after the development of the GCS - and the resultant misplaced clinical and statistical definitions - TBI remains a heterogeneous entity, in which 'best practice' and 'prognoses' are poorly stratified by GCS alone. There is an urgent need for a paradigm shift towards more effective initial assessment of TBI.

Key words: acute brain injury, Glasgow Coma Scale, triage.

Introduction

Traumatic brain injury (TBI) is a leading cause of death and disability with resultant high economic and social costs. Health professionals globally have resolved to apply best evidence care to optimise outcomes. Unfortunately, TBI is a heterogeneous disease entity and as such, defining 'best practice' is challenging and predicting outcomes difficult.

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Since its introduction in the 1970s 'pre-CT' era, the Glasgow Coma Scale (GCS) has been used by clinicians and researchers as a means of stratifying brain injury to predict outcomes.¹ While well-intentioned, this stratification is contrary to the advice of both its inventors and modern guidelines, which caution against this practice. Concerningly, the adoption of GCS by researchers in the 1970s led others in the 1980s to further 'simplify' outcome measurement in TBI by espousing the now commonly held dogma that 'severe' TBI be defined as a GCS score of 8. Fossilised in the research of the 1980s, 1990s and 2000s, this research dogma is now firmly entrenched in global guidelines that continue to grapple with the very real issues of 'best practice' and 'prognostication' in TBI. Curiously, these same guidelines warn against the broad stratification of TBI into its modern groupings (mild, moderate and severe), while simultaneously using them to advise clinicians.

This wide-spread use of an initial 'GCS 8 or less' to dichotomise 'severe' from 'mild' or 'moderate' TBI is an out-dated research heuristic that has become an epidemiological convenience transfixing emergency care. Our contention is that it should be discarded, as this dichotomisation is associated with unacceptable rates of false negative and false positive cases of TBI that require urgent care for better outcomes.

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The GCS and its derived score were introduced in 1974 by neurosurgeons Teasdale and Jennett and was rapidly adopted and popularised as a means of recording the level of consciousness of brain-injured subjects, while reducing inter-observer variability.¹ This seminal contribution preceded the widespread availability of CT scanning, when sequential neurological examination demonstrating the development of lateralising neurological signs, was a key indication for neurosurgical intervention. In particular, the GCS provided a means of serially assessing and identifying subjects dying from progressive extradural and subdural haematomata, where early craniotomy may have been lifesaving.

In 1978, the value of the GCS and score was emphasised by Langfitt in an editorial recommending that it 'should be adopted by neurosurgical units throughout the world'.³ This coincided with the introduction of the American College of Surgeons Advanced Trauma Life Support programme, which facilitated the introduction of the GCS to an enthused, international medical audience.

The corresponding development of trauma systems and trauma centres required identification of those subjects most likely to benefit from transfer. Marshall et al. published a description of the pilot phase of the National Traumatic Coma Data Bank, a cooperative effort of six clinical head-injury centres in the United States.⁴ Data were collected on 581 hospitalised patients with severe non-penetrating traumatic head injury. Severe head injury was defined as a GCS of 8 or less following nonsurgical resuscitation, or deterioration to a GCS 8 or less within 48 h after head injury. Importantly, patients with a preliminary ED GCS of 8 or less, or a prehospital GCS of 8 or less, were not included.

A definition of 'severe brain injury' – incorrectly based on initial prehospital or ED GCS – developed. Adopting a GCS of 8 or less became an easily applied means of identifying the 'severely brain injured'. However, this overlooked both the intentions of Teasdale and Jennett, as well as Marshall *et al.*'s research constraints - that the GCS was specifically developed for subjects' brain-injured postresuscitation and following hospital admission. Furthermore, this dichotomizing of the GCS score contradicted the intent of Teasdale and Jennett, who had stated '...we have never recommended using the GCS alone, either as a means of monitoring coma, or to assess the severity of brain damage or predict outcome'.⁵ It has since been reemphasised that '...dichotomization is rarely defensible and often, will yield misleading results'.6

Risks of dichotomising the GCS

Advanced trauma systems now focus on timely care of all injuries with a focus on eliminating all errors. Triaging based on GCS can delay the care of patients who have rapidly evolving injuries. Sole reliance on the GCS can therefore provide a false sense of security to caregivers and fail to provide timely care for patients, as TBI patients presenting with, for example, a GCS of 15, often have gross CT abnormalities, and non-benign outcomes, including ICU admission, neurosurgical interventions and even in-hospital death.⁷ For example, patients with progressive extradural haematomata (EDH) a key patient group requiring urgent treatment that prompted the introduction of the GCS - may be initially assessed or triaged as a 'mild' TBI, despite a tendency to progress after injury, often in dramatic and rapid fashion.⁸ Chen *et al.* in 2012 described progressive EDH in 38 patients, of whom only 14 had been classified as 'severe' TBI (GCS 8 or less). Five of the subjects had an initial GCS of 13-15, with no relation between the initial GCS score the development of and progressive EDH.

Additionally, the current GCSrelated classification of TBI severity can be counterproductive for some patients who present with GCS >8, but have cognitive disability, with the severity of their injury not recognised or delayed. These patients commonly report difficulties in aspects of day-to-day functioning for at least 12 months post-injury, especially with TBI-related symptoms and interpersonal functioning.

Furthermore, the term 'mild TBI' misrepresents the immediate and long-term burden of TBI and other co-occurring factors experienced by this population. The effects of a concussion can be serious. In the short term, even in the absence of CT findings, mild TBI may cause temporary loss of brain function leading to cognitive, physical and emotional symptoms - such as confusion, vomiting, headache, nausea, depression, disturbed sleep, moodiness and amnesia. In combination with the high degree of heterogeneity in outcomes, these results appear to support the need to further refine emergency TBI classification systems beyond current approaches focused on the crude variables of admission GCS score.

Importantly, post hoc analyses link alterations of the prehospital and ED GCS to head injury. This is what most studies reasonably report - following the subsequent determination that the subjects included have sustained a CT demonstrable TBI. However, subjects with altered consciousness but without subsequently proven TBI proceed down a TBI treatment pathway (intubation, CT scanning, admission to ICU) with potential harm. This component of the total cohort of subjects without TBI undergoing emergency treatment triggered by a GCS <9 is usually not discussed in TBI research.

Unrealised perceived benefits

There are obvious perceived benefits in dichotomising the GCS. The definition enabled researchers to recruit and study a predefined population – subjects with a GCS of 8 or less prior to or on arrival at hospital – to determine the impacts of early interventions designed to reduce secondary injury. However, confounding factors such as intubation, drug and alcohol use, maxillofacial and ocular trauma, preexisting conditions (e.g. dementia, speech and hearing impairment) and physiological derangements (e.g. hypoxia, shock, hypothermia) are commonly present prehospital and in the ED. Also, the interobserver reliability of prehospital GCS assessment has been questioned.9 Unsurprisingly, including prehospital and ED subjects (who were not included in the initial GCS definition) and further dichotomising them into severe and non-severe has led to inconclusive results.

While there had been no observed improvement in mortality between 1930 and 1970 following severe closed TBI, from 1970 to 1990 there was a mortality decline at a rate of 9% per decade with adoption of the GCS, CT scanning, trauma systems development and intracranial pressure monitoring. However, there has been no progress evident in crude mortality rate reduction from 1990 to 2010.¹⁰

Accordingly, current TBI research methodology emphasises functional outcome and almost all large-scale TBI trials utilise some form of risk adjustment, using data beyond just the GCS including other key prognostic variables, such as age, and comorbidities.

Conclusion

Clearly, initial TBI assessment requires sophisticated observation. The GCS is a more nuanced scale than is often appreciated, with each of the three components carrying different weighting for various levels of injury severity.

The evolving nature of TBI in the prehospital and emergency phases of care must be respected. GCS remains an important means of monitoring conscious state – along with pupillary response and neuroimaging. Pupillary response is a key indicator of the severity of TBI, with a loss of pupil reactivity associated with an increase in mortality rate from 16.3% when both pupils reacted, to 38.3% when only one reacted, and to 58.7% when neither pupil reacted.¹¹ The developing area of biomarkers may eventually provide a 'troponin for the prediction of traumatic brain injury outcomes', which may then reliably stratify subjects into risk categories.

Nearly 50 years after the development of the GCS – and the resultant misplaced clinical and statistical definitions – TBI remains a diverse entity, in which 'best practice' and 'prognoses' are poorly stratified by GCS alone. There is an urgent need for a paradigm shift towards more effective initial assessment of TBI.

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Competing interests

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