# The Extended Glasgow Coma Scale and Mtbi

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## Introduction

In cases where a lawyer is attempting to prove that a plaintiff has suffered a mild traumatic brain injury (MTBI) following a motor vehicle accident, it is likely that the plaintiff has experienced a brief period of post traumatic amnesia (PTA) and/or an altered state of consciousness, yet has scored at the top of the Glasgow Coma Scale (GCS) as reported by the ambulance crew or by emergency staff at the hospital.
The defence invariably seizes on the GCS score as proof that the plaintiff did not experience a MTBI. The independent medical examiner, usually a neurologist or a neuropsychologist, may provide an opinion such as:
The plaintiff could not have sustained a MTBI as a result of the motor vehicle accident notwithstanding that he has no memory of the accident or the ride to the hospital in the ambulance because he had a normal Glasgow Coma Scale score of 15/15 as recorded by both the ambulance attendant at the scene and the emergency staff at the hospital.

This medical opinion is incorrect according to the most widely accepted medical definition of MTBI developed by the American Congress of Rehabilitation Medicine. This definition includes a GCS of 13-15:[1]

## Definition

A patient with mild traumatic brain injury is a person who has had a traumatically induced physiological disruption of brain function, as manifested by at least one of the following:

1. any period of loss of consciousness;
2. any loss of memory for events immediately before or after the accident;
3. any alteration in mental state at the time of the accident (eg. feeling dazed, disoriented, or confused); and
4. focal neurological deficit(s) that may or may not be transient;

but where the severity of the injury does not exceed the following:

- loss of consciousness of approximately 30 minutes or less;
- **after 30 minutes, an initial Glasgow Coma Scale (GCS) of 13-15;** and
- post-traumatic amnesia (PTA) not greater than 24 hours.

## Why the reluctance to diagnose MTBI?

This failure to recognize MTBI by the medical profession was highlighted by the comments of Dr. Bryan Jennett, Professor of Neurosurgery at the University of Glasgow and Dr. G. Teasdale, co-authors of the seminal text, Management of Head Injuries, published in 1981:
Our conclusion is that the damage done by, and the symptoms subsequently suffered after, mild head injuries are frequently underestimated. Several factors contribute to this. One is that many of the hospital doctors who deal with mildly injured patients are unfamiliar with recent work in this field, and in any event are not used to dealing with the largely subjective complaints that are the feature of these patients' persisting disability. On the other

hand, those who are accustomed to dealing with severe head injuries are apt to view the mildly concussed patient as fortunate to have escaped serious brain damage - a comparison of little significance to the patient. [2]

Dr. Michael Alexander, a neurologist and recognized authority in traumatic brain injury confirms in the authoritative journal Neurology that MTBI is one of the most common neurologic disorders and explains why it is often left undiagnosed by medical professionals, including other neurologists:
Postgraduate teaching in neurology does not mirror the high prevalence of this disorder - i.e., most [medical] residents probably do not get proportionate instruction in the diagnosis and management of mild TBI…

Mild TBI is characterized by the following: (1) Head trauma may be due to contact forces or to acceleration/deceleration trauma. (2) The duration of unconsciousness is brief, usually seconds to minutes, and in some cases there is no loss of consciousness (LOC) but simply a brief period of dazed consciousness. (3) When the patient is evaluated in the emergency room or at the scene, the Glasgow Coma Scale (GCS) must be 13 to 15, by common definition. As discussed below, only a score of 13 or 14 is due to confusion or disorientation and will be associated with a longer period of amnesia. [3]

## Limitations of the Glasgow Coma Scale in MTBI cases

The GCS was developed by Jennett and Teasdale and was first published in the journal *Lancet* in 1974.[4] The GCS has become the universally accepted measure of the level of impaired consciousness following a brain injury and is graded as follows:

## Glasgow Coma Scale[5]

| | |
|---|---|
| **Eye opening (E)** | |
| Spontaneously | 4 |
| To verbal stimuli | 3 |
| To pain | 2 |
| Never | I |
| **Best motor response (M)** | |
| Obeys commands | 6 |
| Localizes pain | 5 |
| Flexion withdrawal | 4 |
| Flexion abnormal | 3 |
| Extension abnormal | 2 |
| No response | 1 |
| **Best verbal response (V)** | |
| Orientated and converses | 5 |
| Disoriented and converses | 4 |
| Inappropriate words | 3 |
| Incomprehensible words | 2 |
| No response | 1 |

**Coma scare (E + M + V) = 3 to 15**

A GCS score of 13 or higher generally correlates with a mild brain injury, 9 to 12 is a moderate injury and 8 or less a severe brain injury. However, the originators of the GCS have recognized its limitations at the higher range of the scale. In 1989 Jennett confirmed that the GCS was never intended to be applied in the assessment of MTBI:

...it [GCS] was not intended as a means of distinguishing among different types of milder injury. Many of these patients are oriented by the time they are first assessed and therefore score at the top of the Glasgow scale. Yet some of these patients have had a period of altered consciousness, either witnessed or evidenced by their being amnesic for events immediately following injury. **Impairment of consciousness is indicative of diffuse brain damage, but there can also be marked local damage without either alteration in consciousness or amnesia**...[6] [emphasis added]

## Pathophysiological mechanism of MTBI

Jennett and Teasdale suggested that the pathophysiological mechanism responsible for an altered state of consciousness is the same for both a concussion (MTBI) or a more severe brain injury:
Symonds has argued, from a clinical standpoint, that the difference between patients who remain unconscious for days or weeks rather than for minutes or hours could be in the *quantity* of brain damage and not in the *kind* of lesion or its location. He proposed that mild and severe concussion should be recognized; the most obvious pathological counterpart for this would be varying degrees of shearing damage of the white matter. There is some pathological evidence to support this view. Oppenheimer reported microglial stars in patients who had recovered from 'concussion lasting only a few minutes,' but who then died from an unrelated condition. …Indeed, attention is now shifting away from the brain stem as the site of the lesion responsible for the brief alteration of consciousness implied by the term concussion. An alternative explanation would be shearing lesions of a degree that tear only a few axons, but cause a stretch of many, with subsequent temporary failure of conduction in these nerve fibers. This would provide an explanation for the cumulative effect of repeated mild concussion and would be compatible with the evidence hat even mild concussion is associated with structural damage, albeit slight, which leaves its permanent mark in the brain. [7]

This is now the generally accepted view of experts in the field of traumatic brain injury.[8]Oppenhiemer's findings were confirmed in later study published in 1994 in *Lancet*.[9] The authors were able to examine the brains of five persons who suffered a mild concussion (GCS 14 or 15) following motor vehicle accidents. All five then died within 2 to 99 days post injury from other causes. Diffuse axonal injury (microscopic brain damage) was found in all five cases. The authors reported:
All five cases showed involvement of the fornices, which are the major hippocampal projection pathways and are thought to be important in memory, which suggests that such involvement may underlie some of the persisting memory disturbances in patients after concussion. The axonal injury in the fornices is thought to reflect a more widespread functional disturbance of these axonal connecting pathways. Forniceal atrophy on magnetic resonance imaging, which is apparently not related to neuropsychological outcome, has been reported in traumatic brain injury. Similarly, a more widespread functional disturbance of the axonal projections of the reticular activating system may explain the transient loss of consciousness, the mild multifocal axonal injury visible with APP staining representing the most severely damaged axons.

## Post traumatic amnesia

Jennett and Teasdale confirmed that it was the duration of post traumatic amnesia (PTA) and not the GCS that was the best "yardstick for assessing severity of head injury."[10] PTA allows the degree of diffuse brain damage to be assessed without any information from witnesses, or from ambulance or hospital records because it depends solely on the recollection of the patient.

## The Extended Glasgow Coma Scale (GCS-E)

It is now recognized that an altered state of consciousness can result in MTBI that is not detected by the traditional GCS due to its insensitivity to milder brain damage. The greater sensitivity of PTA as a more reliable measure of MTBI has lead to the creation of the Extended Glasgow Coma Scale (GCS-E). The originators of the GCS-E stated:
A severity index that is more sensitive to the nuances of mild TBI would help resolve the controversy with regard to the sometimes severe consequences to which mild and even very mild brain injuries may give rise. A person with a GCS of 15 on admission or soon thereafter, even if amnesic and hypoaroused [diminshed arousal], may be prematurely discharged. Symptoms such as irritability, unreliable memory, and greater fatigue that develop in the days and weeks after discharge are likely to be attributed to malingering or posttraumatic stress rather than to a concussion syndrome. In developing countries, where there is especially high reliance on the GCS, an admitting score of 14 or 15/15 will often result in a denial of compensation claims, even if the victim is unable to return to employment. [11]

The GCS-E was developed with the support of the World Health Organization Advisory Group on the Prevention and Treatment of Neurotrauma that has adopted the GCS-E as an optional diagnostic variable for the revision of the "Standards for the Surveillance of Neurotrauma." [12]
The GCS-E defines 8 levels of PTA and assigns a score that is added to the traditional GCS score. The levels of amnesia are set out in the "Amnesia Scale:

### Amnesia Scale

| Score | |
|---|---|
| 7 | No amnesia: client can remember impact, can remember falling and striking a solid surface, etc. |
| 6 | Amnesia for 30 minutes or less: client regained consciousness while still in vehicle, in street at scene of incident, in ambulance, or on arrival at hospital.[13] |
| 5 | Amnesia of 30 minutes to 3 hours: remembers arriving at emergency room, admission to ward, etc. |
| 4 | Amnesia of 3 to 24 hours: determine duration by content of the first memory, which will be for an event in the ward or other hospital procedure. |
| 3 | Amnesia of 1 to 7 days. |
| 2 | Amnesia of 8 to 30 days. |
| 1 | Amnesia of 31 to 90 days. |
| 0 | Amnesia greater than 3 months. |
| X | Cannot be scored, e.g., can speak but responses are inappropriate or unintelligible, cannot speak because unconscious, intubated, facial fractures, etc. |

In applying the GCS-E, the GCS is first taken in the usual manner. The "Amnesia Score" is then taken and entered after the GCS. For example if the GCS was 15 and the PTA was 30 minutes, the GCS-E score would be 15:5. The GCS-E recognizes that the duration of amnesia (PTA) is in itself an indicator that a person is not laying down permanent memory and accordingly has suffered an alteration in brain functioning. This information is important in more accurately assessing the degree of brain damage. As Jennett and Teasdale stated in their text:
Altered consciousness soon after injury is the clue to the brain damage already suffered. When first seen in the emergency department it is useful to record whether or not the patient is talking. If he is talking, is he orientated and rational? And if he is, can he remember everything about, and since, the accident? **Amnesia for even a few minutes after a blow to the head is evidence of diffuse brain damage.**[14][emphasis added]

The authors of the GCS-E conducted a number of field trials in four hospitals to assess the efficacy of the scale. The GCS-E identified a number of cases that corresponded to Grade I and II concussions as defined by of the American Academy of Neurology. The authors concluded:

Flagging these mild cases allows them to be held in the treatment loop until symptoms remit spontaneously, or appropriate early treatment and counseling are given, and reduces the 'cognitive dissonance' between victims of mild TBI and treating professionals. Finally, wide use of the GCS-E would ease the access to compensation for that minority of patients with mild brain injuries who do sustain lasting cognitive-behavioral deficits; this is especially important in developing countries, where the admitting GCS is the gold standard by which the presence or absence of a brain injury is determined. [15] [emphasis added]

## Conclusion

Since 1989 the limitations of the GCS in the assessment of MTBI have been noted and published by its creators, Jennett and Teasdale. Yet in the following 12 years the GCS has continually been applied to exclude diagnoses of MTBI. Clearly there may be legitimate differences of opinion between qualified experts as to whether a particular individual falls into the category of the 10 to 15 percent of victims who do not recover from a MTBI [16] (sometimes referred to as the "walking wounded or the "miserable minority").[17] However, with advances such as the GCS-E the medical community will be better equipped to accurately diagnose MTBI following a traumatic event. Hopefully this will lead to fewer false negatives in the diagnosis of MTBI, which can leave the victim of a MTBI without recourse to appropriate treatment or adequate compensation.

[1]Kay T. Definition of mild traumatic brain injury. *The Journal of Head Trauma Rehabilitation*, 1993, 8(3), 86-87.

[2] Jennett, B. and Teasdale, G. *Management of Head Injuries,* Contemporary Neurology Series, volume 20, F.A. Davis Company, 1981, 263.

[3]Alexander, M. Mild traumatic brain injury: pathophysiology, natural history and clinical management. *Neurology*, 1995, 45, 1253-1260 at 1253.

[4] Teasdale, G. and Jennett, B. Assessment of coma and impaired consciousness: A practical scale.*Lancet*, 1974, volume 2, 81-84.

[5] Beers, M. and Berkow, R. The Merck manual of diagnosis and therapy. 17th edition, Merck Research Laboratories, 1999, 1429.

[6] Jennett, B. Some international comparisons. In *Mild Head Injury*, edited by Levin, H.S., Eisenberg, H.M. and Benton, A.L., Oxford University Press, l989, 24.

[7] *Supra,* n. 2 at 91.

[8] *Supra*, n. 3 at 1254. In addition see: Povlishok, J.T. & Coburn, T.H. Morphological change associated with mild head injury. In *Mild Head Injury*, edited by Levin, H.S. Eisenberg, H.M. and Benton, A.L., Oxford University Press, 1989, 37-53; Ommaya, A.K., Faas, F. and Yarnell, P. Whiplash injury and brain damage: an experimental study. *The Journal of the American Medical Association*, l968, volume 204 (4), 285-289; Ommaya, A.K. and Gennarelli, T.A. Cerebral Concussion and traumatic unconsciousness. *Brain*, l974, volume 97, 633-654; Gennarelli, T.A., Thibault, J., Adams, J.H., Graham D.J., Thompson, C.J., Marcincin, R.P. Diffuse axonal injury and traumatic coma in the primate, *Annals of Neurology*, l982, volume l2 (6), 564-574; Jane, J.A., Steward, A., and Gennarelli. Axonal degeneration induced by experimental noninvasive minor head injury. *Journal of Neurosurgery*, l985, volume 62, 96-l00; Oppenheimer, D.R. Microscopic lesions in the brain following head injury. *Journal of Neurology_Neurosurgery and Psychiatry*, l968, volume 3l, 299-306; Dixon, C.E., Taft, W.C. and Hayes, R.L. Mechanisms of mild traumatic brain injury. *Journal of Head Trauma Rehabilitation*, 1993, volume 8(3), 1 - 12; Zasler, N.D. Mild Traumatic Brain Injury: Medical Assessment and Intervention. *Journal of Head Trauma Rehabilitation*, 1993, volume 8(3), 13-29 at 29; Gronwall, D. and Wrightson, P. *Mild Head Injury: A Guide to Management.* Oxford University Press, 1999, 20-28.

[9] Blumbergs, P.C., Scott, G., Manavis, J., Wainwright, D.A. Simpson, and McLean, A.J.. Staining of amyloid pecursor protein to sudy axonal damage in mild head injury. *Lancet*, 1994, volume 344, 1055-56.

[10]*Supra*, n 2 at 90.

[11]Nell, V., Yates, D.W., and Kruger, J. An extended Glasgow Coma Scale (GCD-E) with enhaned sensitivity to mild brain injury. *Arch Phys Med Rehabil*, 2000, volume 81, 614-617.

[12]*Ibid* at 617.

[13]*Ibid,* at 614. The event anchors for 5 and 6 have been amended to apply to the local response times in accordance with the authors' instructions.

[14]*Supra,* n. 2 at 96.

[15]*Supra,* n. 11 at 617.

[16]*Supra,* n. 3 at 1256.

[17]Ruff, R., Levin, H. and Marshall, L. Neurobehavioral methods of assessment and the study of outcome in minor head injury. *Journal of Head Trauma Rehabilitation*, 1986, volume 1, 43-52.