How Severe is a Brain Injury? A Guide for Lawyers

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Deciding on the severity of a brain injury can be critical in medicolegal claims, but can also be problematical. In this article, I provide a brief guide which may help those

Acquired brain injury forms a significant proportion of both personal injury and clinical negligence claims, with traumatic brain injury (TBI) making up much of the former and non-traumatic brain injury making up a large chunk of the latter. Non-traumatic brain injury could include conditions such as stroke, brain infections, hypoxia, brain tumours, etc. A key issue that often arises is, How severe is the brain injury? as this will often guide thinking about which expert opinions to gather, quantum calculations, rehabilitation needs, how long the case might last, etc. In this article, I provide a brief guide which may help those in the legal profession in understanding brain injury severity, and I refer to recent findings from brain injury research.

1. Comatose State

Loss of consciousness has typically been considered a key marker of severity of brain injury. The Glasgow Coma Scale was developed in the 1970s with this in mind, and it remains the most widely used measure of loss of consciousness. It measures eye opening, best verbal response and best motor response, with a maximum score of 15/15 (normal, no coma) and a minimum score of 3/15 (deep coma). Numerous studies have shown a relationship between duration of coma and outcome (Mattei and Teasdale, 2020). Correlations are high, but not perfect, which in turn highlights two important points. Firstly, there are a number of other early indicators of severity of brain injury which combine with depth and duration of coma to influence outcome. These early indicators include factors such as infection, hydrocephalus, seizures, post-traumatic confusional state, etc. Secondly, there are acute brain injury conditions which involve a major insult to the brain, but where there is no loss of consciousness. In the case of TBI, this includes some cases of penetrating missile injury to the brain and in non-traumatic brain injury this includes some cases of stroke.

2. Post-Traumatic Amnesia

Post-traumatic amnesia (PTA) is usually defined as the post-injury period for which the patient has no clear, continuous memory. It will invariably include the period for which the person was unconscious, and also the subsequent period when they were significantly confused. The duration of post-traumatic amnesia is often considered important as it will generally be a good indicator of the severity of injury and

the likely long-term outcome. The brain mechanisms underlying PTA have now been well documented (De Simoni et al., 2016). There are two broad methods of gauging duration of post-traumatic amnesia. One method is the 'concurrent' one which usually involves giving orientation and simple memory tests to the patient daily during their confused state. When they have obtained a pre-determined score (usually maximum score) for several successive days, they are then considered to be 'out of PTA'. The second method is the 'retrospective' one, which entails asking the patient some months/years after the injury what they recollect of events post-injury, such as visits of family or friends, tests carried out in hospital, discharge from hospital, etc. A good deal of research has been carried out both on which PTA scales are best to use, and the important issue of confounding factors which may affect estimates of PTA. These confounding factors include analgesia and anaesthesia that patients may receive during the post-injury period (Kemp et al., 2010), as well individual differences in recollecting events that occurred months or years previously (Friedland and Swash, 2016). Nevertheless, for both TBI and for other non-traumatic conditions such as stroke or encephalitis, the duration of post-ictal amnesia (memory loss for events after the acute episode) can be a useful indicator of the severity of a brain insult.

3. Brain Imaging Features

The field of brain imaging correlates of severity of brain injury is a complex and evolving one (Shah et al., 2020). Brain imaging may often pick up features which point to the presence of a significant brain injury. Such features may range from compound or linear skull fracture, to focal lesions such as brain haemorrhage or contusions. Hydrocephalus (enlargement of ventricles) is sometimes found as a consequence of TBI, and this is usually also a marker for a severe TBI. Advanced MR imaging procedures now mean that more abnormalities may be detected in TBI than in the past. Signatures of axonal injury (damage to white matter tracts in the brain) and microbleeds (tiny haemorrhages) are two such abnormalities which may provide a sensitive indicator of extent of brain injury. Abnormal accumulation of proteins in the brain ('tau pathology') can also now be seen in advanced brain imaging procedures (Gorgoraptis et al., 2019). It is however important to remember that some patients with a severe TBI will not only have a normal CT brain but may also in rare cases have a normal standard MRI scan (Bigler, 2016). While brain imaging has to date been the major biological variable that has been used to measure severity of brain injury, novel 'biomarkers' which are evident in the blood or cerebrospinal fluid are emerging as promising indicators of severity of brain injury (Mehta et al, 2020).

4. When a 'mild TBI' may turn out not to be so mild

Mild traumatic brain injuries can be amongst the most problematical to diagnose with certainty (Menon et al., 2020). There are two broad ways in which what appears to be a mild traumatic brain injury may have not so mild consequences. The first is the more straightforward one of brain complications, such as delayed development of a blood clot or raised intracranial pressure (Kim et al., 2013). This phenomenon was first described in a classic paper published in 1975 about TBI patients who 'talk and die' (Reilly et al., 2015). The other way in which a mild brain injury may have severe consequences is a more complex one, and that is where the person may have had pre-injury vulnerabilities which lead them to have a 'catastrophic' reaction to a traumatic event and to symptoms that may initially be quite distressing but which would usually recover. These symptoms may include dizziness, headache, memory / concentration lapses, fatigue, mood changes, PTSD, etc. Although the term 'concussion' and 'post-concussion syndrome' are often used to describe mild TBI cases who have these symptoms as a persistent feature, it is being increasingly recognized that such terminology hinders rather than helps understanding of the consequences of mild TBI (Sharp and Jenkins, 2015). There can be pre-injury vulnerabilities which may predispose to disabling symptoms after mild TBI; these usually include factors such as pre-injury mental health issues and pre-injury addiction issues (Iverson et al., 2017). Age can also be considered a vulnerability factor, and a mild head injury in someone in their 80's or 90's may sometimes be associated with subsequent significant decline, especially in cognitive function (Abdulle et al., 2018).

5. When a 'severe TBI' may not have so severe outcomes

While a mild TBI may sometimes not turn out to be so mild, the reverse may also occasionally happen – that is, a severe TBI may be followed either by a remarkably good recovery or indeed some improvement in the individual's functioning. Surprisingly good recoveries after a severe brain injury have been described in a number of case studies (Schutz, 2007; McDonald et al., 2019), and are generally associated with high premorbid cognitive functioning, enhanced levels of motivation, good family support and a period of intensive rehabilitation. Paradoxical improvements after brain injury are rare but have been well-documented, and these have been subsumed under the topic of 'post-traumatic growth' (Lyon et al., 2020). Post-traumatic growth is where there have either been cessation or reduction in pre-injury maladaptive habits/attributes (e.g. addictions, low mood) or improved functioning in the form of greater resilience or enhanced mental wellbeing whereby the individual accepts and appreciates his/her current circumstances more than before, and feels happier and more satisfied as a result.

6. Classification

Having decided that a brain injury is at a certain level of severity, the next issue is what classification system and terminology we should use to describe that level of severity. Here there are a range of views. The traditional view, based on World War I and World War II studies, was that brain injury severity is best described at four levels – mild, moderate, severe and very severe (Russell and Smith, 1961). Since then,

there have been a wide range of classification systems. A recent classification system which has gained some popularity is the Mayo classification system, which has three levels of severity of TBI – mild possible, mild probable, and moderate-severe (Malec et al., 2007). While the Mayo classification is to be welcomed by attempting to provide an evidence-based approach, significant limitations of this scheme include the lack of substantive empirical validational studies to support the classification, the lumping together of moderate and severe brain injury, and the reluctance to accept a category of very severe brain injury. It is possible that with advances in brain imaging and biomarkers associated with traumatic brain injury, better classification systems will emerge in the years to come.

7. Conclusions

It is important for everyone concerned in brain injury cases, whether it be lawyers, doctors, allied health professionals, etc to come to an accurate understanding of the severity of a brain injury. There are situations where persistent symptoms are largely psychological in nature, perhaps in some cases consciously or unconsciously exaggerated, but where the patient is repeatedly told that he/she has suffered a severe brain injury (Baxendale et al., 2019). The term 'diagnosis threat' has been used to describe such a situation (Freeson et al., 2019). This may contribute to poor adjustment to the head injury in question and variable cooperation with rehabilitation services, and in some cases programmes of rehabilitation services are not required and may inadvertently reinforce the false impression that a severe brain injury has occurred. It is clear that brain injury severity is a complex issue, and further research from biological, medical and psychological perspectives may help to give a more solid basis to judgments and conclusions reached about the severity of a brain injury.

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References

Abdulle A. et al. (2018). Early predictors for long-term functional outcome after mild traumatic brain injury in frail elderly patients. Journal of Head Trauma and Rehabilitation, 33: E59-67.

Baxendale S. et al. (2019). Neuropsychological outcomes following traumatic brain injury. Practical Neurology, 19: 476-82.

Bigler E. (2016). Systems biology, neuroimaging, neuropsychology, neuroconnectivity and traumatic brain injury. Frontiers in Systems Neuroscience, 10: doi: 10.3389/fnsys.2016.00055

Di Simoni S et al. (2016). Disconnection between the default mode network and medial temporal lobe in post-traumatic amnesia. Brain, 139: 3137-3150.

Freeson M et al. (2019). Influence of diagnosis threat and illness cognitions on the cognitive performance of people with acquired brain injury. Neuropsychological Rehabilitation, 29: 1637-54.

Friedland D, Swash M. (2016). Post-traumatic amnesia and confusional state: hazards of retrospective assessment. Journal of Neurology, Neurosurgery and Psychiatry, 87: 1068-74.

Gorgoraptis N et al. (2019). In vivo detection of cerebral tau pathology in long-term survivors of traumatic brain injury. Science Translational Medicine, 11: doi: 10.1126/scitranslmed.aaw1993

Iverson G et al. (2017). Predictors of clinical recovery from concussion: a systematic review. British Journal of Sports Medicine, 51: 941-48.

Kemp S et al. (2010). Analgesia and other causes of amnesia that mimic post-traumatic amnesia (PTA): A cohort study. Journal of Neuropsychology, 4: 231-6.

Kim J et al. (2013). Injury patterns in patients who 'talk and die'. Journal of Clinical Neuroscience, 20: 1697-1701.

Lyon I, et al. (2020). 'Putting a new perspective on life': a qualitative grounded theory of posttraumatic growth following acquired brain injury. Disability and Rehabilitation,

doi: 10.1080/09638288.2020.1741699

Malec J et al. (2007). The Mayo classification system for traumatic brain injury severity. Journal of Neurotrauma, 24: 1417-1424.

Mattei T, Teasdale G. (2020). The story of the development and adoption of the Glasgow Coma Scale: Part 1, The early years. World Neurosurgery, 134: 311-322.

McDonald S. et al. (2019). Rebuilding life after brain injury. Hove: Psychology Press.

Mehta T. et al. (2020). Current trends in biomarkers for traumatic brain injury. Journal of Neurology and Neurosurgery, 12: 86-94.

Menon D et al. (2010). Position statement: Definition of traumatic brain injury. Archives of Physical Medicine and Rehabilitation, 91: 1637-40.

Reilly P et al. (1975). Patients with head injury who talk and die. Lancet, 2: 375-7.

Russell W, Smith A. (1961). Post-traumatic amnesia in closed head injury. Archives of Neurology, 5: 16-29.
Schutz L. (2007). Models of exceptional adaptation in recovery after traumatic brain injury: A case series. Journal of Head Trauma and Rehabilitation, 22: 48-55.

Shah S et al. (2020). Quantitative multimodal imaging in traumatic brain injuries producing impaired cognition. Current Opinion in Neurology, 33: doi: 10.1097/WCO.0000000000000872

Sharp D, Jenkins P. (2015). Concussion is confusing us all. Practical Neurology, 15: 172-186.