Review article: Altered states of consciousness, theories of recovery, and assessment following a severe traumatic brain injury

By Diane Duff

Abstract

The causes and consequences of severe traumatic brain injury are reviewed, including mechanical injury and neurochemical changes following focal and diffuse injuries. A discussion of terminology seeks to clarify current nomenclature and descriptions of behaviours during emergence from coma.

Introduction

In this review of the scholarly literature, the causes, incidence, definitions, theories of recovery, and assessment techniques for altered states of consciousness following severe brain injury are discussed and critiqued. Future directions for nursing research and the role of the clinical neuroscience nurse are discussed.

Causes

Motor vehicle, motor vehicle-pedestrian, bicycle, and motorcycle collisions are the most frequent causes of severe traumatic brain injury. Less common causes include blunt trauma to the brain caused by falls, violence, and suicide attempts (Andrews, 1996; Grossman & Hagel, 1996; Kalsbeek, McLaurin & Harris, 1980; Snow, Macartney-Filgate, Schwartz, Klonoff & Ridgley, 1988; Statistics Canada, 1994; Wong, Dornan, Schentag, Ip, & Keating 1993). Approximately 50% of injuries occur to individuals between 15 and 40 years of age, with those between the ages of 15 and 24 at the highest risk. The majority of patients are single, and there are higher rates of unemployment and alcohol use than in the general population (Annegers, Grabow & Kurland, 1980; Klonoff, Costa & Snow, 1986; Vogenthaler, Smith, & Goldfader,1989; Wong et al).

Mechanical injury

The injuries that result from TBI are often classified as focal or diffuse brain injuries. However, individuals who experience a severe TBI frequently have elements of both kinds of injury. Focal injuries are large enough to be seen by the naked eye and are caused by lacerations, contusions, and intracranial hematomas. Falls and other blunt trauma generally cause focal injuries. Diffuse axonal injury (DAI) is the most severe form of diffuse brain injury. It results in widespread damage to the axons in the white matter of the brain. The predominant causes are high-speed motor vehicle collisions (MVC) or pedestrian motor vehicle collisions. There are two kinds of acceleration forces that result from the impact of a MVC. Translational forces caused by impact result in differential movement of the brain relative to the inner surface of the skull. However, it is the rotational acceleration that causes stretch and shearing of the connecting pathways between the white and grey matter due to density differences between the white and grey matter that is the most responsible for DAI (Adams, Graham, & Gennarelli, 1981; Adams, Gennarelli, & Graham, 1982; Gillis, Pierce, & McHenry, 1996; Grossman & Hagel, 1996; Hilton, 1995; McIntosh, Smith, Meaney, Kopatka, Gennarelli, & Graham, 1996; Meaney et al., 1995). Individuals who experience diffuse lesions are much more likely (75%) to experience coma than those with focal lesions (25%) and longer periods of post-traumatic amnesia (Wilson, Hadley, Wiedman & Teasdale, 1995). Diffuse lesions are usually located in the corpus callosum and the brain stem. Adams, Doyle, Ford, Gennarelli, Graham, and McClellan (1989) proposed a grading system for DAI. Grade I would indicate DAI throughout the white matter, Grade 2 would be the white matter plus the corpus callosum, and Grade 3 adds the brainstem.

Neurochemical changes

The initial injury caused by trauma is the primary injury. The initial injury triggers both local and distal disruption to the blood-brain barrier resulting in decreased cerebral blood flow, edema, increased metabolism, and microglial and macrophage proliferation. Considerable axonal injury also results from the complex cascade of biochemical events at the cellular level that unfold over a period of hours following the primary injury. Increases in acetylcholine are associated with neurological disturbances and damage. Catecholamines are markers of the extent of brain damage and they stimulate the sympathetic and adrenomedullary stems increasing circulating levels of epinephrine and norepinephrine. Cytokines, endogenous opioid peptides, and excitatory amino acids have been shown experimentally to increase the extent of the secondary injury, as part of this pathophysiological cascade (Duhaime, 1994; McIntosh et al., 1996). Ions such as calcium, magnesium, and potassium, all elements that are important to normal neuronal cellular metabolism and homeostasis, have been studied experimentally following brain injury, and metabolic abnormalities related to these ions were observed that contribute to neurotoxicity following injury. Neuronal damage is further extended by oxygen free radicals and lipid peroxidation that cause cerebral ischemia. Microscopic examination reveals transection of the axon from the cell body. The severed ends...
Definitions
Severe traumatic brain injury (TBI) has been variously defined as coma following a traumatic brain injury lasting more than six to 24 hours (Ezrachi, Ben-Yishay, & Kay, 1991; Wehman, Kreutzer, & West, 1990), a Glasgow Coma Scale of less than 7-8 (Bishara, Partridge, Godfrey, & Knight, 1992), post-traumatic amnesia longer than 24 hours (Boake, Freelands, Ringholz, Nance, & Edwards, 1995), or a combination of these criteria (Kater, 1989; Moore & Stambrook, 1995). The incidence of traumatic brain injury is 200-300 per 100,000 per year, with five to 25% of all brain injuries classified as severe. This results in an estimated 10-75 cases of severe TBI per 100,000. It is estimated that 10-14% of individuals with severe TBI will remain in a vegetative state (Grossman & Hagel, 1996; Jennett, Teasdale, Braakman, Minderhoud, Heldon, & Kurze, 1979; Levi, Linn, & Feinsod, 1989; Nordstrom, Messeter, & Sundberg, 1989; Tiret, Hausherr, & Thicoipe, 1990).

A review of the literature reveals several overlapping terms used to describe the period of unawareness and unresponsiveness that often follows a coma associated with a severe TBI and that precedes intentional action. True coma rarely exceeds two to four weeks in duration (Plum & Posner, 1980). Clinically it is often observed that the patient is “lighter” as true coma is replaced by a state variously described as vegetative, post-comatose, minimally responsive, coma vigile, apallic, altered states of consciousness, or “slow-to-recover” (American Congress of Rehabilitation Medicine, 1995; Ansell, 1991; Bricolo, Turazzi, & Feriotti, 1980; Grossman & Hagel, 1996; Peters & Gerstenbrand, 1977; Plum & Posner, 1980; Szabolcs & Grosswasser, 1991). The International Working Committee, comprised of internationally renowned researchers in the field of severe TBI, met in 1995 with the intention of developing consensus on the diagnostic classifications of the clinical states following coma in order to facilitate assessment, progress, and treatment (Andrews, 1996). They noted the usual trajectory of recovery following a severe TBI is coma to a vegetative state to a post-vegetative state. They noted individuals following a severe TBI proceed through these states in a very individualistic way, depending on the type of injury. Plum and Posner (1980) noted that the limits of consciousness are hard to define satisfactorily and quantitatively we can only infer the self-awareness of others by their appearance and their acts (p. 3). Arousal and content are the two physiological components they believed are essential to consciousness. The nomenclature and definitions for the altered states of consciousness that follow severe TBI reflect both arousal, or the degree of wakefulness, alertness, and attention, and the content, or ability to perceive, process, and react to stimuli, of the individual.

Coma
No detectable signs of awareness or sleep/wake pattern. Painful stimuli may elicit reflex responses.

Vegetative presentations
Hypo-responsive state: No detectable signs of awareness. Sleep/wake pattern is present. Delayed or absent reflex response to strong environmental stimuli.

Reflexic responsive state. No detectable signs of awareness. Sleep/wake pattern is present. Mass extensor responses or startle to stimulation without habituation or flexor withdrawal. Roving eye movements and facial expressions may occur.

Localizing responsive state: Limited signs of awareness to touch, sound or visual stimuli, may turn to sound or touch. Sleep/wake pattern is present. Tracking eye movements without focusing.

Transitional state/borderline: More definite signs of awareness of visual, auditory, and tactile stimuli. Better able to localize. Able to track objects or people and may show emotional response (smile/cry) to family members.

Non-vegetative states
Inconsistent low awareness: Aware of stimuli, responds inconsistently to verbal commands. Sleep/wake pattern is present. However remains completely dependent with severe cognitive impairments.


Incidence
As a result of lack of clarity concerning definitions, it is difficult to determine the incidence of altered states of consciousness following a severe TBI. However, Szabolcs, Costoff, and Grosswasser (1992) conducted a retrospective chart audit over a seven-year period to calculate an incidence of post-comatose unawareness of 2.4 patients per 1,000 admitted to the hospital with a severe TBI or four cases per 100,000 per year for the general population. Bricolo, Turazzi, & Feriotti (1980) conducted a 10-year study of individuals who remained in coma, defined by more than two weeks of unconsciousness, and concluded it occurred in six individuals per 100,000 admitted with TBI. Andrews (1996) stated worldwide estimates of incidence for altered states of consciousness range between two to 10 per 100,000 per year.

Theories of recovery
There are many theories to explain recovery following a severe TBI. These include diaschisis, plasticity, compensation, and environmental quality.

Diaschisis
Some early functional improvement is related to the resolution of diaschisis which is the stage following brain injury in which healthy areas connected to damaged areas exhibit a temporary loss of function. It is caused by vascular and metabolic changes such as edema and neurotransmitter sensitivity that

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occur in response to the injury (Boyeson, Jones, & Harmon, 1994; Waxman, 1988). Early treatment interventions are aimed at controlling vascular and metabolic changes that can cause temporary and permanent secondary damage.

**Plasticity and compensation**

Almi and Finger (1992) proposed that neuronal tissues and processes have some limited capacity or plasticity to repair and recover function after injury. Additionally, they stated that undamaged areas are able to compensate for areas of damage and that new pathways can be developed over time to allow for improvement of function rather than true recovery. Redundancy theory proposes that pre-existing silent or accessory pathways exist that are unmasked following injury (Boyeson, Jones & Harmon; Sofroniew, 1993). Many brain injury rehabilitation strategies, such as sensory stimulation, are based on compensation theory.

**Environmental regulation**

Almi and Finger (1992) postulated that there are critical periods in which environmental quality will impede or facilitate recovery. LeWinn and Dimancescu (1978) noted that some animal studies using sensory deprivation and enrichment following brain injury supported the importance of sensory regulation to the reacquisition of pre-morbid abilities. However, it is an enormous leap to translate those findings to human cognition (Ansell, 1991). Still, Salisbury (1991) expressed concern that lack of a supportive environment, in which accurate assessment and institution of an individualized program of rehabilitation occurs, can lead to excess disability. In this instance, an individual fails to redevelop abilities despite the potential for improvement. Environmental regulation theory is the foundation for sensory regulation programs in which the individual receives directed stimuli of planned duration and intensity, periods of rest, and control of non-directed and environmental stimuli to reduce frightening or confusing environmental stimuli (Rader & Ellis, 1994; Talbot & Whitaker, 1994).

Retrospective patient reports in the literature have attempted to describe the process of regaining consciousness following an extended period of time spent in coma. The patients describe first becoming aware of strong feelings and sensations, as well as seemingly random bits of information that seem to surface. However, they cannot necessarily be recalled at will (Paul & Littlejohns, 1994). Collerton (1993) described this process as the joining together of little islands or fragments of memory. As these fragments coalesce into a more integrated whole and memory improves, the individual who has experienced a severe TBI is able to move beyond altered states of consciousness. However, the complexity of the human nervous system coupled with the uniqueness of the injury make it difficult to predict with accuracy the outcome of severe brain injury or trajectory of recovery following severe TBI.

**Assessment of consciousness**

Ideally for individuals in altered states of consciousness, a good assessment tool would have the capacity to accurately capture and track the early behaviours that are indicative first of increased arousal and then the behaviours that allow content to be inferred by increasingly sophisticated behaviours in response to stimuli. Additionally, the perfect tool would be easy to master and score, useful for both research and clinical applications, and have interdisciplinary appeal (Frank-Stromberg & Olsen, 1997).

While several tools have been described in the scholarly literature as being useful for measuring behavioural change with this population, none of them are widely used, and none approach perfection. Additionally, as consciousness must be inferred from behaviour, and the behaviours of individuals following severe TBI are often inconsistent and transient, intermittent assessments are often difficult to perform with reliable results.

The tool most uniformly used for assessment is the Glasgow Coma Scale (GCS) (Teasdale & Jennett, 1974). The GCS measures eye opening, motor movement, and speech. The range is from 3-15, with 3 indicating the eyes are closed and there is neither movement nor vocalization. Fifteen, at the other end of the scale, indicates that the eyes are spontaneously open and that the individual is able to respond appropriately to simple questions and commands using motor and verbal responses. The GCS was designed as a rough measure of coma and levels of consciousness for use in emergency settings, therefore it is not surprising that the GCS lacks the sensitivity to measure small, discrete changes in behaviour. As a result, most individuals, despite making some neurological gains beyond coma within two to four weeks post-injury, will continue to score in the 3-5 range for an extended period of time.

The Ranchos Los Amigos or Levels of Cognitive Functioning Assessment Scale (LCFS/LOCF/LOCFAS/Ranchos/RL/RLA) is another tool developed to assess individuals in altered states of consciousness following coma. One significant difficulty in reviewing the literature in relation to this tool is that it has a number of different abbreviations. However, at least 12 papers are available in a CINAHL search (1972-2001) using either the full term or the abbreviations listed here as a search term, that use the LCFS as instrumentation in a study or use it as a measure of comparison (Flannery, 1995; Flannery & Korchek, 1993; Flannery & Land, 2001; Hilton, 1994; Mackay, Morgan, & Bernstein, 1999a,b; Malec, & Thompson, 1994; Neumann, 1996; Wright, Bushnik, & O’Hare, 2000; Zafonte, Hammond, Mann, Wood, Black & Millis, 1996). However, still other papers that either use the tool or discuss the tool’s psychometric properties have been missed in indexing (Dowling, 1985; Flannery, 1993; Powell, & Wilson, 1994; Reimer, Conrad, Newcommon & Annear, 1990; Sandel & O’Dell, 1993). Originally developed by Hagen, Malkmus, and Durham (1972) at the Ranchos Los Amigos hospital in Downey, California, the scale provides a description of behaviours indicating level of arousal and response to stimuli linked to cognitive levels. The categories range from I (coma) to VIII (normal cognitive functioning). Key approaches are identified as stimulation for levels I-II, structure for Levels IV-VI, and community integration for Levels VII and VIII (Mitchell, 1989). However, like the GCS, it lacks the sensitivity needed to allow clinicians to track discrete changes for individuals in altered states of consciousness, and individuals will be classified at Level II and III for extended periods of time following emergence from coma. While the LCFS has been
widely used, there are conflicting reports in the literature concerning its reliability and validity. Reimer et al. noted both poor reliability and validity of the tool and Dowling (1985) evaluated only for acceptable levels of inter-rater reliability. However, Flannery (1998) concluded that validity was acceptable in relation to the Glasgow Coma Scale, the Disability Rating Scale (Rappaport, Hall, Hopkins, & Belleza, 1982), and the Stover-Zeiger Scale (Stover & Zeiger, 1976). Considering the popularity of LCFS in clinical settings, further work on evaluating and refining the psychometric properties of the tool is warranted.

The Disability Rating Scale (DRS) is an eight-item ordinal scale used to assess arousalability, awareness, responsivity, self-care, dependence on others, and psychosocial adaptability. The scores range from 0 (no gross disability) to 30 (death). The Coma/Near Coma Scale (CNCS) expands the DRS rating scale “to measure small clinical changes in patients with severe traumatic and non-traumatic brain injuries who function at very low levels characteristic of near vegetative and vegetative states” (Rappaport, Dougherty, & Kelting, 1992, p. 628). The scale has eight categories: auditory, command responsivity, visual, threat, olfactory, tactile, pain, and vocalization. The stimulus is specified in duration, intensity and number of trials. Rappaport and colleagues followed 20 patients for 16 weeks who had scored 21-29 on the DRS using the CNCS, administering the assessment for three consecutive days, then weekly for three weeks, then every two weeks until the 16-week timeframe was reached. Good correlation was established between the CNCS and the DRS and there was excellent inter-rater reliability. Rappaport and colleagues stated the tool was sensitive enough to identify positive changes in this group of patients and the results were predictive of which patients would make progress beyond this state.

The Western Neuro Sensory Stimulation Profile (WNSSP) was developed to measure, monitor, and predict improvement of the cognitive functioning of individuals with severe TBI who were assessed in LCFS Levels II-V (Ansell & Keenan, 1989). The categories developed are arousal, attention, expressive communication, and response to visual, auditory, tactile, and olfactory stimulation. There are 32 items and rating scales of 0-1 or 0-5 are used. The score range is 0-110. Reliability of the scales ranged from 0.59-0.95 with five of the seven scales exceeding the acceptable standard of 0.8. Ansell and Keenan calculated a validity of 0.73, significant at the p=0.001 level, between the WNSSP scores and the individual’s rating on the LCFS using a Kendal rank-order correlation. However, criterion-related validity should use a superior measure of the behaviour, not one in which psychometric parameters are not well established (Frank-Stromberg & Olsen, 1997).

The Freeman Questionnaire (FQ) was developed as both a research and a clinical assessment and treatment tool for measuring and comparing baseline behaviours and behaviours in response to stimuli. Freeman (1989) in his book for families, developed 200 “yes/no” response questions in six categories: medical prognosis, vigilance, emotion, drive, sensory, and motor abilities, which are scored to determine the individual’s level of functioning. The scale ranges from Levels 1-4, with Level 1 being a state of coma, Level 2 includes vegetative and transitional states, Level 3 is indicative of locked-in syndrome, and Level 4 scores indicate the individual is non-vegetative. Other than one small research study (n=7), there are no study results in the scholarly literature using the FQ (Talbot & Whitaker, 1994). While Talbot and Whitaker noted good concurrent and content validity with the GCS, the RLA/LCFS, the Coma Near Coma Scale, and the Disability Rating Scale, detailed statistical analyses were not provided to support this statement. They also noted that the tool was sensitive for the population and had good inter-rater reliability, though again, statistics were not provided.

Freeman (1996) has also published an abridged version of the FQ, the Coma Exit Chart that uses the categories of the GCS to measure exit from coma and vegetative states. However, there are no reports in the research literature concerning use of this tool in research or clinical applications.

The Sensory Modality Assessment Rehabilitation Technique (SMART) was designed to assess and monitor cognitive progress in individuals in vegetative states in a rehabilitation setting. The tool focuses on assessment of awareness and response to stimuli and is said by the originators to be more sensitive than WNSSP in a single study of 30 individuals diagnosed as being vegetative (Gill-Thwaites, 1997). According to Wilson and Gill-Thwaites (2000), those who emerged from an altered state of consciousness could be mathematically predicted using their SMART scores. However, only these two reports by the originators appear in the scholarly literature.

While several tools have been developed to assess and monitor individuals who are in altered states of consciousness following severe TBI: LCFS, CNCS, WNSSP, FQ, and SMART, there are few research or clinical reports in the scholarly literature to evaluate their utility in measuring the small, incremental changes that occur as individuals begin to emerge from coma and progress to states of awareness. A Cumulative Index of Nursing and Allied Health Literature (CINAHL) and MEDLINE search revealed only nine documents in which WNSSP was reported to have been used or discussed. Two were by the researchers who developed the tool, the others were independent studies (Anderson, 1993; Corrigan, Bogner, Mysiw, Clinchot, & Fugate, 1997; Gill-Thwaites, 1997; Hall, MacDonald & Young, 1992; Lippert-Gruner, Quester, & Terhaag, 1997; Talbot & Joanette, 1998; Wilson, & Gill-Thwaites, 2000). A similar search revealed only two studies that used the CNCS after the original work of Rappaport, Dougherty, & Kelting (1992): Pilon and Sullivan (1996) and Wolfe and Gleckman (1995). Only two papers used or discussed the FQ (Duff & Wells, 1997; Talbot & Whitaker, 1994). Even clinically the tools developed for monitoring altered states of consciousness following coma are not widely used by nurses. Neumann (1995-1996) surveyed 214 certified neuroscience nurses to investigate their use of neuroassessment tools and found that while 55.6% used the GCS and 18.2% used a neuroflow sheet designed by their institutions, none reported using the other tools discussed in this paper (pp. 5-6).

**Conclusion**

Severe traumatic brain injury has devastating consequences for the individual. The trajectory of recovery following such
an injury is uncertain, especially for those persons who remain in coma and altered states of consciousness for prolonged periods of time. While some work has been done to clarify definitions of altered states of consciousness and progress has been made in developing valid, reliable, and sensitive tools for measuring clinical progress, efforts remain fragmented with few reports in the scholarly literature and little consensus to date for use of classifications or use of assessment tools in practice or research. Given limited local populations, it is perhaps unsurprising that this has occurred. However, if real progress is to be made in evaluating and improving the effectiveness of treatment modalities, it is first necessary to utilize an internationally recognized classification system to standardize data collection. Then universally accepted assessment and monitoring tools for altered states of consciousness are required. The international and interdisciplinary acceptance and use of the Glasgow Coma Scale improved the ability to compare findings across a variety of studies. Adopting a standard assessment measure for altered states of consciousness would be similarly beneficial for facilitating sharing of findings.

Global communication via the Internet, e-mail, and electronic access to abstracts and full text papers has the potential to facilitate this process.

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References


Neuroscience nurses who are at the bedside 24 hours a day seven days a week from emergency admission to community or long-term care have a unique responsibility and opportunity to improve assessment of clinical progress following coma. Nurses need to be involved in expanding knowledge in this area. Nurses can be involved in developing, refining, and evaluating definitions and tools for use in research and clinical settings. Replication studies, clinical use and careful evaluation of assessment tools, studies evaluating the psychometric properties of existing tools for sensitivity, validity and reliability, refinement of categories and scales of existing tools, meta-analysis of existing studies, and detailed, descriptive case reports describing emergence from coma and interventions related to environment regulation are all required to improve knowledge on altered states of consciousness in order to improve patient outcomes for this population of patients.