Update

Cognitive rehabilitation following traumatic brain injury*

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The last decade has shown a rapid expansion in the provision of acute rehabilitation services for patients with traumatic brain injury (TBI) in South Africa, with a growing awareness of the problems associated with such injuries and recognition of the need for structured therapy programmes to maximise functional outcomes after injury. However, as a sub-specialty of the rehabilitation model, the field of cognitive rehabilitation is much less understood than the physical modalities. Despite the fact that cognitive disorders derived from TBI are extremely common and can affect all aspects of an individual's functioning, few patients are referred to specialised rehabilitation units with cognitive remediation as the primary goal.

This article identifies the common cognitive impairments in TBI and discusses therapeutic treatment options for these conditions. It also touches on the devastating impact such impairments can have on family members and caregivers, who are in as much need of psychological support as the victim during the rehabilitative process.

Changes in cognition, behaviour and personality after TBI are considered by clinicians to be among the most difficult disabilities to assess and manage effectively.[1] A loss of physical function is easily identified and can usually be attributed to tangible impairments as a result of brain injury, such as weakness, spasticity, or loss of balance. It is therefore relatively easy to measure these impairments objectively at the start of a rehabilitation programme, subscribe a graded programme of therapy over a determined course of time, and evaluate the outcome at the end.

However, the evaluation and rehabilitation of cognitive deficits is a much less tangible practice. There is also the perception that the severity of a disability depends on the level of physical impairment – if a patient can walk and perform his activities of daily living independently following a TBI then he has made a good recovery. In practice, however, the exact opposite can be true. Often the most tragically disabled patients are the 'walking wounded' – those who are physically independent, but with such significant disorders of cognition that they are unable to perform the most basic tasks without supervision. Even patients who on the surface have good cognitive skills can present with subtle nuances of change in behaviour and personality that make them strangers to their families and misfits in their previous social circles. A comprehensive cognitive rehabilitation programme aims not only to address the cognitive deficits and psychological needs of the patient, but also to educate, counsel and support the family, caregivers and employers in dealing with the ramifications of the injury.

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Research from major TBI centres strongly suggests that the bulk of neurological recovery from acute brain injury occurs within the first 6 months post injury. The maximal duration of the recovery period is more controversial. Some researchers affirm that neurological recovery is virtually complete by 1 year, whereas others assert that recovery can extend 2 years or more after injury. It is clear, however, that certain areas of dysfunction recover more quickly than others. For example, recovery of physical abilities and functional skills such as mobility occurs rapidly, often within 3 months after injury. However, recovery of cognitive abilities (particularly higher level cognitive and communicative skills) tends to take much longer.[2] There is probably no final endpoint to the recovery process; rather, the pace of recovery slows and its scope narrows. Patients continue to learn new adaptive skills and compensatory strategies in order to solve functional problems. Thus, cognitive recovery merges imperceptibly into ongoing learning and adaptation.

Cognitive impairments after TBI

Due to the diffuse and multifocal nature of TBI, there is great variety in the cognitive impairments that can result. However, certain common patterns do exist, most likely related to the common areas of grey-matter injury (i.e. frontal and temporal poles) and white-matter damage (i.e. midbrain and corpus callosum).

Impairments of arousal and attention

Deficits in levels of arousal and attention are among the most widespread cognitive deficits following TBI. Arousal may be defined as the general state of responsiveness to external stimuli. Deficits in arousal could lead to low levels of responsiveness, slowed speed of information processing and an inability to cope with cognitively demanding situations. Attention may be considered to be the selective channelling of arousal. It includes the ability to focus attention on certain stimuli (focused or selective attention), to sustain attention over time, to filter out distracting influences, to switch attention between two or more task demands and to shift attention in line with changing goals and priorities. Disorders of attention may result in poor command following abilities, distractibility, poor task completion, poor error monitoring, and confused and disorganised thought processes. Other areas of common cognitive deficit such as memory, perseveration and impulsivity may also be related to disorders of attention.

Behavioural programmes to retrain arousal and attention include taking frequent rests or naps, frequent task changes, graded training to increase speed of performance, and practising activities to such a degree that they no longer place much demand on disordered attentional processes. Compensatory strategies include engaging in tasks when most alert, giving alerting

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cues, making use of a non-distracting environment, breaking complex tasks down into simpler steps, providing supervision and allowing adequate time for responding.[3]

Impairments of learning and memory

Memory impairments are among the most common, most persistent and most handicapping of the cognitive complaints after TBI. All patients with moderate to severe injuries, and most with mild injuries, experience amnesia with regard to the events immediately preceding (retrograde amnesia) and following the injury (post-traumatic amnesia or PTA). As patients recover from the acute confusional state post injury, these amnesias may resolve to a greater or lesser extent; the final interval unaccounted for ranges from minutes to weeks or months, depending on the severity of the injury. PTA is nearly always longer than retrograde amnesia and its duration is one of the best measures of injury severity.

Other forms of memory commonly affected include deficits in anterograde memory (difficulty in storing and retrieving new information), prospective memory (ability to remember to do something in the future) and the elements of memory involved in explicit, effortful learning (e.g. memorising a list of words). In contrast, implicit, automatic learning (e.g. remembering how to tie a shoelace) is often spared after a TBI. Even patients who have forgotten how to perform basic procedural tasks such as dressing are able to improve with practise of these skills, based on implicit memory abilities.

Therapeutic remediations of memory deficits focus largely on the 'exercise' of memory – practising tasks and repeating material until they become familiar and over-learnt. Of particular benefit in this area is the concept of 'errorless learning', in which patients are prevented from making errors during the learning process, with the level of assistance required gradually tapered as the patient begins to initiate more of the actions correctly. One of the most commonly used remedial approaches is that of using compensatory strategies. Patients are taught to use written strategies (commonly date books, diaries, organisers, memo notes, etc.) to record events and provide memory cues for themselves. The success of these approaches, however, depends on an individual's awareness of memory deficits and acceptance of the need to use prosthetic devices (and indeed, often the physical ability to use these devices).[4,5]

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Impairments of frontal executive function

Executive functions refer to the cognitive abilities that allow us to adapt to change, solve unexpected problems, anticipate outcomes and generally cope with situations that fall outside of our normal routines. This requires complex cognitive skills such as cognitive flexibility, reasoning, self-monitoring, self-adjustment of performance, modulation of responses to people and situations, planning, consideration of multiple alternatives and their consequences, and the execution of a plan of action based on all these modalities. There is considerable evidence that executive functions are highly dependent on the integrity of the prefrontal cortex, an area that is often injured in TBI. Examples of executive dysfunction include disorganised speech and action, aimless behaviour, stimulus-boundedness (inability to detach from irrelevant stimuli), aberrant or inappropriate interpersonal or sexual behaviour, and impulsive and/or perseverative thought and action. Lack of insight into these and other deficits and poor ability to profit from feedback are usually part of the clinical picture.

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Assessment of these executive functions is complicated by the fact that they are least obvious in highly structured and routine environments such as hospitals. Because executive disorders relate to the ability to deal with novelty and change, they are difficult to see within structured tasks but must be looked for in challenging or open-ended situations. Patients may do fairly well on traditional cognitive tests yet show profound life disruption from executive dysfunction. The neuropsychological changes that a patient may undergo as a result of executive dysfunction are arguably the least understood and most difficult to deal with, particularly from the family's point of view. Physical disabilities and overt cognitive handicaps are glaringly obvious, but deficits in executive dysfunction may be subtle and only observable over time. There is therefore often little allowance made for executive impairments once the patient re-enters his 'normal' environment, when in fact these deficits can be catastrophic to independent living, employment and successful relationships.

Treatment programmes include the training of specific behaviours and task-specific skills, behaviour modification strategies and the teaching of compensatory skills for a variety of problems. Due to the nature of executive dysfunction and the fact that most of the deficits manifest once the patient has left the secure and structured environment of the hospital, careful analysis of the patient's premorbid social and vocational environments is of paramount importance. Therapists spend a large amount of time educating family and employers, both with regard to the condition, as well as to the adaptation of the home and work environments that will be necessary after discharge.[6]

Impairments of visuospatial perception and construction

Visuospatial disorders are observed less commonly than other disorders after TBI, perhaps because the posterior areas of the brain are less often damaged than the frontal and temporal regions. A brain-injured patient may also perform poorly on visual or spatial activities because of deficits in attention, problem solving, organisation or other motor impairments, rather than visuospatial deficits *per se*.

Examples of visuospatial perception disorders include visual scanning disturbances, alterations in body schema, and impaired perception of form, spatial relations, colour and figure-ground relationships. In general, patients with non-dominant hemisphere lesions will tend to display greater and more dramatic impairment in perceptual and constructional functions than those with dominant hemisphere lesions. These disorders have functional consequences in many areas of daily life such as dressing, grooming, eating, driving a vehicle, ambulating, writing and performing manual assembly tasks.

Treatment modalities aim at retraining perception by ongoing reinforcement (such as using a mirror to provide visual feedback while dressing or walking) or teaching compensatory strategies such as using verbal or written prompts instead of visual cues when working on functional activities.[7,8]

Social and behavioural disabilities as a result of TBI

Psychosocial problems are among the leading causes of prolonged disability in individuals and are a leading source of family stress, relationship failure and vocational handicap. Such disabilities usually result from a combination of cognitive deficits and behavioural impairments which interact in complex ways with each other; very seldom are behavioural problems present in the absence of cognitive deficits. A disturbed mind state as a result of cognitive impairment, in interaction with malfunctioning regulatory systems, can result in a myriad of behavioural disabilities which are often too complex to dissect into cause-and-effect relationships.

Agitated, aggressive and disinhibited behaviour

Agitation and aggression are common occurrences after a TBI and are typical of a generally agitated state as patients emerge from coma and PTA. Agitation is even thought to be a 'stage of recovery' for many patients as they emerge from PTA and often reduces as levels of orientation and cognition improve. The frontal regions so often damaged in TBI are believed to play an important role in the inhibition of impulse and inappropriate responses and many patients never fully recover control over volatile or disinhibited behaviours. In severe cases a tiny provocation can result in a dramatic outburst of verbal or physical aggression, while in milder cases this may manifest in chronic irritability or 'moodiness'. Unfortunately, insight into their behavioural disturbances and the consequences of such behaviour is often lacking, making remediation difficult.

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Therapeutic treatment programmes require the whole rehabilitation team to work together in analysing maladaptive behavioural triggers, providing positive reinforcement and modelling of appropriate behaviours. Specific treatment strategies include behavioural modification and redirection techniques and behavioural management training.[9]

When acceptable behaviour has been achieved in the controlled clinical environment, it should be generalised into home and community settings. Family members should be taught relevant interventions, including how to deal with behavioural regression when the patient is exposed to new people or environments. In fact, the role of family members and caregivers is critical in the effective management of these disorders.

Reduced initiation

Some brain-injured patients simply fail to act without extensive cuing or structure imposed from without. In extreme cases, patients may be able to describe a course of action verbally, and express a sincere intention to carry it out, but still do nothing. This type of deficit may accompany particular patterns of neurological damage that disrupt the linkage between limbic motivational inputs and the cognitive and motor components of action. The use of psychostimulants, activating tricyclics and dopaminergic agonists such as Sinemet and bromocriptine has been shown to be useful in improving initiation.

Commonly used rehabilitative approaches to impaired initiation include physical, verbal, written or pictorial cues for the steps involved in a task, with behavioural reinforcement for proceeding from one step to the next. Structured time frames for the completion of tasks, enforced by timers and buzzers, may also be of benefit.

Disorders of initiation can have profound effects on independent living and psychosocial function. Once again, family members require explicit education regarding these impairments, as otherwise they are prone to interpret such behaviour as laziness or poor motivation.[10]

Depression

Studies of patients involved in TBI rehabilitation have found that well over half are significantly depressed or have been at some point since the injury. In fact, the incidence of depression appears to increase from 1 to 5 years post injury. There is also a significantly high rate of concomitant depression and anxiety in patients' families (particularly spouses) after TBI. Depression after TBI probably results from neurological and psychosocial factors. The diffuse axonal injury of TBI induces acute disruption of neurotransmitter systems and it is theorised that neurotransmitter depletion, particularly in noradrenergic and serotonergic systems, could contribute to acute depressive symptoms.

Depression in reaction to physical, cognitive and psychosocial consequences of TBI is most common and may manifest weeks, months or even years after the initial neurological insult. To an extent, depression is an indication of a patient's increased awareness following a TBI and may be regarded, to a point, as a desirable 'normal' reaction to a catastrophic event. However, persisting and worsening levels of depression can completely derail a patient's recovery following a TBI and are a significant cause for concern. Depression may also be the breaking factor in a familial relationship struggling to survive the fallout following TBI.[11]

Therapeutic modalities include counselling, psychotherapy, support groups and community re-entry programmes. Family members and caregivers may also benefit hugely from interventative programmes.

Awareness deficits

Brain-injured patients frequently seem to lack insight into obvious deficits and their implications. They may be aware of some deficits and not others; patients with TBI are usually less aware of cognitive or behavioural limitation than they are of physical deficits. Some patients may acknowledge a deficit, but be sublimely unaware of the severity of the problem, or its consequences. Unawareness of a deficit is not the same as psychological denial, which is the conscious or unconscious refusal to admit to a problem of which one is (at some level) aware. Unawareness of deficits after TBI creates a significant obstacle to rehabilitation efforts and is often a predictor of poor performance and poor outcome following rehabilitation. Such patients may not be motivated to practise therapy tasks and unwilling to consider changes in employment or educational plans, and may even see no need for the entire rehabilitation effort. This lack of awareness is sometimes compounded by a lack of insight from family members and caregivers in the severity of the condition, particularly in patients who have otherwise made good physical and functional recoveries. This may result in the precipitate withdrawal of the patient from the rehabilitation programme, at a time when intervention is most crucial.[12]

Conclusion

Increased interest has been focused on TBI in recent years as more patients survive severe injury and as the long-term sequelae of all grades of injury become clearer. This focused interest has resulted in a number of important advances in knowledge and treatment of patients with TBI, and the field of rehabilitation is increasingly recognised as a vital step in the recovery of patients after injury. As awareness grows, so increasing numbers of patients are referred to specialist rehabilitation units throughout the country and many funders have in recent years included specific rehabilitation allowances to their schedules of benefits. However, the perception prevails that patients with physical impairments following TBI are the most suitable candidates for rehabilitation, and that there is little value in the remediation of cognitive deficits. Yet the neurobehavioural deficits of many brain-injured patients represent their most significant obstacles to community and vocational reintegration. Much research is in progress to clarify the extent to which cognitive and behavioural impairments are remediable, and to identify the most effective remediation strategies.

What is evident is that significant cognitive improvement is possible following a TBI and that, as in the recovery of physical abilities, this process can be optimised utilising appropriate interdisciplinary assessment, treatment and evaluation programmes. It is also evident that a comprehensive programme of family and caregiver education and supportive counselling during the rehabilitation process is of vital importance in order to achieve maximal outcomes and successful reintegration into social and community settings.

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In a nutshell

- The field of cognitive rehabilitation is much less understood than the physical modalities.
- Cognitive disorders following TBI are extremely common.
- Neurobehavioural deficits of many brain-injured patients represent their most significant obstacles to community and vocational re-integration.
- Changes in cognition, behaviour and personality after TBI are considered by clinicians to be among the most difficult disabilities to manage effectively.
- Patients with little or no physical fallout from a TBI may have significant cognitive impairments.
- Research strongly suggests that the bulk of neurological recovery from acute brain injury occurs within the first 6 months after injury.
- Memory impairments are among the most common, most persistent and most handicapping of the cognitive deficits after TBI.
- Patients with cognitive impairments may be completely unaware of their deficits and limitations.
- The role of family members and caregivers is critical in the effective management of patients with cognitive impairment.