The Neurobehavioral Disturbances of Traumatic Brain Injury

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Abstract

Objective: Cognitive and behavioral disturbances after traumatic brain injury (TBI) are not uncommon in clinical practice. Although the neurobehavioral consequences following TBI are richly documented in the western literature, studies on this topic in T'aiwan are still limited in scope and number.

Method: The present article first reviews the cognitive problems after head trauma, including deficits in information processing, memory, and executive functions. Meanwhile, emotional and behavioral disturbances, such as the disinhibition and apathy syndromes that occur after trauma, are also discussed.

Results: There is a variety of neurobehavioral disturbances after TBI, which can be severely detrimental to the patient's social and occupational functioning.

Conclusion: There is a necessity to establish a comprehensive assessment of neuropsychological deficits following TBI in T'aiwan, with linkage to cognitive and/or behavioral remediation.

Keywords: TBI, Neurobehavioral Disturbances

Introduction

In Taiwan, traumatic brain injury (TBI), a significant public health problem with an incidence of 160,000 to 200,000 people, produces more than 8,000 deaths annually (Department of Health, 2005). The western studies also evidenced a remarkable annual incidence of 1.5 to 2 million people who suffered from TBI in the United States (National Institute of Health, 1998). There is a bimodal distribution of TBI subjects, with the highest incidence among young people aged 15 to 24, and a second group of people aged greater than 75 years (Kraus & McArthur, 1996). Kraus & McArthur (1996) also found that nearly 25% of the TBI patients require hospitalization, and have a wide variety of emotional problems and cognitive impairments in addition to physical disabilities. These deficits, in turn, impair the functional outcomes of patients suffering from TBI, such as resuming work and social relationships. Several studies (Booze et al., 2005; Fife, 1987, Max et al., 1991) have also shown that losing productive work time after TBI may be the largest component of the economic costs to the government.

Definition of TBI severity

Several kinds of classifications of head injury are available for the clinician, for example, classifications by level of consciousness, mental status following head injury, level of trauma severity, or location of body injury (Stein, 1996). Among them, the Glasgow Coma Scale (GCS), developed by Teasdale & Jennett (1976), is the most widely used scoring procedure for mental and neurological status after head trauma, which is scored on the sum of three components: eye opening, verbal response, and best motor response. Its scores range from 3 to 15, with higher scores indicating better mental status. According to GCS, the severity of head trauma could be further subdivided into mild injury (GCS=13-15), moderate injury (GCS=9-12), and severe injury (GCS=3-8). However, the definition of mild TBI (mTBI) is not universally accepted (American Congress of Rehabilitation Medicine, 1998; Allexa et al., 1995; Granacher, 2003; Ingebrightsen et al., 2000). The main differences in defining mTBI lay in the GCS score, the duration of LOC, and the presence of intracranial lesions. Many researchers have tried to resolve this confusion by dividing mTBI patients into subgroups. For instance, in an early study, Williams et al. (1990) subdivided mTBI patients into the complicated group (with radiological evidences of focal lesions and depressed skull fracture) and the uncomplicated group (with normal CT scan findings). They found that the uncomplicated group had a better functional outcome than the complicated one over a period of 6 months after head trauma. A recent study by Borghero et al. (2003) supported this result and provided further evidence that patients in the complicated mTBI group had more emotional and cognitive deficits than those in the uncomplicated one. Ingebrightsen et al. (2000) even excluded patients with intracranial lesions who usually scored less than 13 on the GCS from the mTBI group. In terms of clinical management (e.g., discharge from emergency room, or admission for observation), they thought that mTBI patients should be divided into the minimal group (GCS=15 without any LOC) and the mild group (GCS=14-15 with a LOC of less than 5 minutes, and no neurological signs). Hence, in order to avoid the controversial definition of mTBI in future studies, researchers have to recognize the heterogeneity of the mTBI group and clearly describe patients' clinical features in the studies.

Pathophysiology of TBI

The initial events of brain trauma involve mechanical distortion of the brain. The injury is typically classified as primary or secondary. The primary neuropathology of TBI is diffuse axonal injury (DAI) caused by shearing forces generated in the brain by sudden deceleration (Fovilishock et al., 1986). Furthermore, Gennari et al. (2002) reported that the distribution of DAI seemed to be parasagittal deep white matter spreading from cortex to brainstem. Secondary injuries, such as focal hemorrhages, brain edema, and increasing intracranial pressure (ICP), are the results of normal physiological responses to the primary one (Nolin, 2005).

The common focal hemorrhages after TBI are brain contusions, subdural hemorrhages (SDH), and subarachnoid hemorrhages (SAH). A contusion is caused mainly by contact between the surface of the brain and the bony protruberance of the skull base (coup injury), and a rebound effect where there is a compression of the brain tissue 180 degrees from the point of impact (contre-coup injury) (Graham et al., 2002; McCance & Huether, 2002). Graham (1996) reported that the most common locations for cerebral contusions are the frontal and temporal poles, the orbital gyrus, the cortex above and below the Sylvian fissures, and the lateral and inferior aspects of the temporal lobes. These contusions can result in symptoms involving neurobehavioral functions, such as attention, executive functions, personality, and memory which, in turn, are very harmful to the TBI patients' social and occupational functions (Booze et al., 2005; Testa et al., 2006).

SDHs are hemorrhages that develop below the dura mater and are usually induced by the rupture of the bridging veins. Acute SDH often develop rapidly over the first 24 to 48 hours after injury, while chronic SDH, which may present weeks or months after what appeared originally to be a trivial head injury, are particularly common in elderly patients (Granacher, 2003).

In summary, TBI can cause both diffuse and focal brain injuries, which in turn compromise patients' cognitive and emotional functioning. Diffuse injuries
are usually detrimental to the "state" functions (Weintraub, 2000), such as information processing and sustained attention, while focal injuries often deteriorate the "channel" functions (Weintraub, 2000), including memory, executive functions, and personality

**Neurocognitive deficits following TBI**

As described in the previous section, TBI is associated with many types of pathophysiology that damage cerebral cortex and subcortical structures as well as white matter connections. These brain lesions usually disrupt a large neural network and lead to subsequent neurobehavioral changes. Additionally, there are many other complicating psychosocial and medical problems that may contribute to post-traumatic cognitive, behavioral and emotional impairments. Hence, any neurobehavioral deficits have to be carefully evaluated on an individual basis, as they might be influenced by premorbid conditions (e.g., prior head trauma history, psychiatric disturbances), comorbid conditions (e.g., post-traumatic seizure, side effects of medications) and environmental constraints (e.g., stresses of working and interpersonal relationships).

**Information processing and attention**

Information processing and attention problems after TBI are the most common cognitive deficits reported by patients, families and clinicians (Levin, 1988, Gronwall, 1981). For instance, Levin (1988) reported that 9% of severe TBI patients have impairments in vigilance (the maintenance of attention over time), while 77% of remaining patients showed increased distractibility within the context of normal vigilance. Following a mTBI performance on simple measures of attentional capacity, such as the Digit Span subtest of the Wechsler Adult Intelligence Scale (WAIS), Wechsler (1997), may recover to relatively normal levels. The attentional deficits may not be uncovered unless more sophisticated neuropsychological measures are used. In the early literature, Gronwall et al. (1977) used the Paced Auditory Serial Addition Task (PASAT) to explore mTBI patients' information processing speed and sustained attention, and they found that patients performed poorer than the controls did one week after head trauma but performed as well as healthy participants did 3 months post-injury. A recent study by Mathias et al. (2004) further confirmed that information processing speed in terms of reaction time was compromised after mTBI, particularly in the visual task difficulty increase.

In terms of more severe TBI groups, attention problems are more easily observed (Butow et al., 1989; van Zomeren and Brouwer, 1990). Recently, Zoccolotti et al. (2004) recruited 106 patients who were initially examined in different domains of attention capacity, including alertness, sustained attention, selective attention, and divided attention. The patients had considerably much difficulty in the selective attention tasks requiring go-no-go responses or divided attention. These attentional deficits can have a remarkable impact on the ability to work independently, and the disruptive effect does not wear quickly (White et al., 2000). Wood (1993) has shown that when these deficits are disruptive enough to make TBI patients too distractible or too unable to maintain directed or focused attention, the patients cannot benefit from the cognitive restructuring. However, patients usually have to expend great mental and physical efforts in occupational tasks that they may have managed well before the head trauma. This, in turn, can lead to increased physical and psychological fatigue, especially in stressful environments.

In summary, attention difficulty and slowing of information processing manifest obviously regardless of the TBI severity. However, the prolonged reaction time of patients appear to be influenced by multiple factors, such as the type of task and the working memory load that it requires, usually represented by the number of competing stimuli and choices (Eisinger et al., 2007; Zahn & Minsky, 1999; Madigan et al., 2008). Therefore, we have to evaluate deficits of information processing after TBI through a more sophisticated task and a careful neuropsychological interview.

**Memory**

Patients often complain some difficulties to retrieve information after head trauma, even among those with mTBI and good medical recovery (Rimel et al., 1981). Our recent study (Yang et al., 2007) further revealed that subjective cognitive complaints, mainly manifested as memory and attention problems, have adversely affected mTBI patients' social functioning and family relationship at 2 months post-injury. In fact, some of these subjective memory impairments could also be substantiated by objective memory testing results, and the effects of these post-traumatic memory problems can be disabling and significantly limit the patient's capability to live independently, maintain a job, and get along with others appropriately (Drake et al., 2000).

When the severity of head trauma increases, the PTA, usually refers to the immediate and dramatic amnesic effects of TBI is easily observed. Clinically, the inability to save information in the memory storage continuously causes the patients to be confused, agitated and disoriented in the PTA stage. Ruff et al. (1989) recognized that PTA is a subclass form of antegrade amnesia, which is one of the most distinctive markers of head injury. PTA can be assessed with standardized instruments such as the Galveston Orientation and Amnesia Test (GOAT, Levin et al., 1982; Chiu et al., 1993). In addition, several studies (Tate et al., 2001; Aldeno & Nowick, 2002) have shown that the duration of PTA can be reliably predictive of functional in dependent outcome.

Once the patient becomes oriented, some memory functions may still not return to normal level. Those memory problems are typically called antegrade memory deficits. In an early study, Levin (1979) has discovered that a severe memory deficit was found in 16% of those recovered from moderate TBI and 25% of those recovered from severe TBI. Even though Brooks et al. (1986) reported that short-term memory is less likely to be affected after head trauma, most clinical and scientific studies of TBI revealed a viably evidence for this disruption of memory processing. For instance, Watt et al. (1999) has indicated that TBI patients show greater deficits on explicit memory tasks than implicit ones. It was found that verbal or visual recall perfor- ance on the explicit memory tasks is often impaired with relatively intact recognition (Spikman et al., 1995; Milder, 1998; Milder et al., 1999), though Hannay et al. (1979) noted defective recognition memory.

In summary, memory function is usually the most severely affected cognitive function following TBI. Memory deficit, in turn, seriously deteriorates patient's daily and occupational functions. Moreover, the past studies evidenced that these memory impairments manifested a specific defective pattern after head trauma, that is, the problem in the acquisition of information more vulnerable than that in the retrieval of information (Hannay et al., 2004).

**Executive functions**

Executive functions encompass the cognitive and emotional processes that underlie many aspects of human adaptation, adjustment and achievement (Eisinger et al., 2007) TBI-related damage to prefrontal neural circuits was linked to a variety of executive dysfunctions. Specifically, the dorsolateral prefrontal circuit is thought to mediate more highly cognitive aspects of behavior such as conceptual formation, planning, and working memory (Eisinger et al., 2000; Salmeron et al., 2001). Lezak et al. (2004) conceptualized executive functions as having four components: (1) volition; (2) planning; (3) purposeful actions; and (4) effective performance. Each involves a distinctive set of behaviors and are all necessary for appropriate, socially responsible, and effective self-regulation and performance.

In a recent study, Thomas et al. (2004) noted that volition refers to the capacity for intentional behavior and requires the ability to formulate a goal. People who lack volitional behaviors simply do not think of anything to do. They may be unable to initiate activities unless others instruct them to do so. Unfortunately, there are no formal tests for examining volitional capacity. The examiner has to rely on the observations of patients in daily life and/or reports by caregivers or families who see them regularly.

In order to plan a, person must be able to conceptualize changes from present circumstances and deal objectively with oneself in relation to the environment (Lezak et al., 2004). The planner has to identify and organize the steps and elements needed to carry out an intention or to achieve a goal which involves a number of capacities. Except for sequential and hierarchial aspects necessary for the ability of planning, good impulse control, preserved sustained attention, and intact memory functions are also required. Typically, working memory tasks are used to evaluate a patient's planning ability. For instance, Bobak et al. (2000) designed a working memory task needed to guide a sequence of actions and revealed that TBI patients per-
formed significantly poorer than did normal controls. In addition, the Tower of London test (TOL; Culbertson & Zillmer 2001) is also one of the most common tests to examine planning ability. In an early study of brain injured persons, patients with predominantly left anterior lesions performed least well, while those with posterior lesions did as well as normal subjects (Shallack and Bagge 1991). However, some recent studies (Levin et al., 1991; Cockburn, 1995) were not able to replicate the previous results; rather, found this test to be relatively insensitive to the cognitive impairments associated with TBI patients.

After planning, a person has to translate an intention into productive, self-serving activity. Doing so requires abilities to initiate, maintain, switch, and stop sequences of complex behavior in an orderly and integrated manner (Lezak et al., 2004). This programming function is usually necessary for the successful performance of nonroutine tasks but is not certain when the action sequence is routine (Shallice, 1982). Lezak et al. (2004) also observed that severe TBI patients, who had trouble in programming activities, could not associate their verbalized intentions or plans with the potential action. To evaluate this capacity, a construction test, the Tinkertoy Test (TTT, Lezak, 1982), made it possible for patients to initiate, plan, and structure a potentially complex activity and to carry it out independently (Garcia & DeLauro, 1990) further evidenced that the TTT was also a useful predictor of TBI patients' employability.

The ability of self-regulation can be assessed by two different components: productivity and flexibility (Lezak et al., 2004). Patients who have productivity problems can give the details of what needs to be done but cannot carry out what they verbally acknowledge or propose. Slowed response is probably the most common cause of low productivity in people with brain lesions. Clinical semantic fluency test, in which patients are asked to name as many exemplars as possible in a given semantic category, such as animals or fruits, is usually used to examine the capacity of productivity. In a recent study, Henry & Crawford (2004) found that the semantic fluency test was sensitive to frontal and temporal lesions caused by head trauma. In terms of flexibility, people have to shift their thoughts or action according to environmental demands. Inflexibility results in perseverative, stereotyped, nonadaptive behavior and difficulties in regulating motor acts. The Wisconsin Card Sorting Test (WCST, Nelson, 1976; Heaton, 1981) is recognized as the most useful test for revealing defects of mental shifting and conceptual formation. The WCST has been considered a good measure of frontal lobe dysfunctions since the 1960s (Milner, 1968), though this was questioned in recent studies (Griffin & Cooper, 1990; Michigan, 1995). In addition, many researchers (Robinson et al., 1988; Stuss et al., 1985) have confirmed that TBI patients made more perseverative errors on the WCST.

Lezak et al. (2004) considered that a performance is as effective as the performer's ability to monitor, self-correct, and regulate the qualitative aspects of behaviors. Defective self-monitoring can spell any kind of performance. Some patients simply cannot correct their mistakes because they do not perceive the errors, while others may perceive but do nothing to correct them. There are only few examination techniques have been developed to study self-monitoring or self-correcting behaviors. The examiner can only evaluate the patient's ability to self-monitor by observing the patient's responses qualitatively, such as checking the accuracy of their own behavior. According to some tests like the Arithmetic and Digit Symbol Coding Subtests of the WAIS (Wechsler, 1997), will readily expose poor self-monitoring.

In summary, executive dysfunction is also one of the most common cognitive sequela after TBI. In fact, both focal and diffuse frontal lesions caused by trauma can result in remarkable executive impairments which may represent a main component of a patient's dully living failure (Eisinger & Damasio, 1985; Fortin et al., 2003). Specifically, Acker (1990) has also found that the capacities (or planning self-regulation and self-monitoring) are critical for independent and adaptive functioning in the real world. However, a comprehensive and universally acceptable neuropsychological examination of executive functions is still lacking. Therefore, placing a priority on both assessment and linkage to cognitive remediation is urgently needed.

**Self-awareness**

**Disturbances of self-awareness**, a common neurobehavioral deficit following a variety of neurological disorders (Eisinger et al., 2007), are usually manifest by an inadequate understanding of one's own neurological or neuropsychological disturbances. Babinski (1914) first used the term “anosognosia” to describe a phenomenon in which a patient denied his/her hemiplegia. Frangitano (1991) further defined that self-awareness refers to a process to integrate the information from both the inner experiences and outer environment. Consequently, a deficit of self-awareness can be viewed as an inability to be aware of one's functional impairments.

Disturbances of self-awareness are also easily recognized in patients with TBI (Frangitano, 1991). In an early work of Oddy et al. (1985), the authors asked patients and their families to describe the behavioral problems that they have to face 7 years after TBI, and found that patients tended to under-estimate the frequency of problems compared to that reported by the families. Using a different methodology, Foy and Rochee (1986) developed a questionnaire to explore the TBI patients' awareness of their deficits. They also revealed that TBI patients typically rated themselves as much better at daily living and behavioral problems than did their clinician or family. In fact, many recent studies (Abreu et al., 2001; Powell et al., 2001; Wallace & Bogne 2000) confirmed the above findings and provided further evidence that, if present, self-awareness is one of the most disturbing problems for TBI patients' families and their care-providing professionals.

An inability to recognize their own functional impairments is a major barrier to rehabilitation in TBI patients (Frangitano, 1991). Patients with this deficit usually do not accept the rationale for participation in a rehabilitation program and may not put forward consistent efforts even when they have begun to take part in the treatment plan (Garcia & DeLauro, 1998; Frangitano & Wang, 1999). Unfortunately, there are few tools currently available for TBI patients with anosognosia: some researchers (Solberg et al., 1998; McKeown & Schindler, 1989; Langer & Padua, 1992) tried to develop therapeutic approaches, including contingency management and modeling techniques, to enhance the patient's self-awareness. Hence, further studies are needed to find out the most effective interventions for anosognosia after TBI.

**Neuro-psychiatric consequences following TBI**

Neuropsychiatric illness is a highly prevalent complication of TBI regardless of the severity of the trauma or the patient's age (Gendron et al., 2003; Jor ge, 2005). Fann et al. (2004) compared the frequency of psychiatric disorders among 499 TBI patients and 287 controls. They found that the prevalence of any psychiatric illness was 49% in the first year following moderate to severe TBI, 54% following mild TBI, and 18% in the control group. They concluded that TBI patients are at high risk of developing psychiatric problems compared to the control subjects. Another recent study (Aebrien et al., 2004) further examined TBI patients' psychiatric illness over a long-term follow-up. They discovered that the probability of having an axis I diagnosis of DSM IIIR decreases over time, while the prevalence of psychiatric disorders still continues to be significantly higher in TBI patients than the controls even many years after head trauma. Except for psychiatric disorders in the DSM IIIR, other motivational and behavioral disorders, such as apathy and the disinhibition syndrome, both were associated with lesions of frontal areas after TBI, are easily observed in any clinical setting.

**The apathy syndrome**

Marin (1991) revealed that apathy is a specific neuropsychiatric syndrome marked by a significant reduction of goal-directed behaviors resulting from motivational deficits. Marin (1990) proposed that this syndrome is not associated with disturbances of consciousness, cognitive impairments or emotional problems. Clinically, many psychiatric disorders and diseases of the central nervous system (CNS) can cause the apathy syndrome (Andreasen, 1982; Bachman, 1985; Levy et al., 1998), which further make the patients lack the motivation to get appropriate medical treatment and rehabilitation (Knipp, 1997). The apathy syndrome was associated with the lesions of some specific neuromatonic structures in the literature. Some researchers (Cummings, 1993; Paradiso et al., 1999; McPherson et al., 2002) found that the lesions of the frontal lobe lead to the apathy syndrome. For instance, Cummings (1993) reported that the manifestation of apathy has been related to lesions of the medial frontal cortex. A study by Paradiso
et al (1999) further confirmed the relationship between the lesions of the frontal lobe and apathy by revealing that patients with frontal lobe injuries exhibited a remarkable apathy and slowing of responses. Others (Kuzs et al., 1999; Habib, 2000) provided evidence that apathy was found in patients with subcortical lesions. For example, Habib (2000) found that lesions of the caudate nucleus, globus pallidus and bilateral medial thalamus could cause a persistent apathy syndrome. Recently, some researchers (Lee and Cummings, 2001; Lees et al., 2000) integrated the previous findings and reported that apathy could result from lesions of one of the frontal-subcortical circuits, the anterior cingulated prefrontal circuit, and concluded that the presentation of human motivation could be associated with lesions disconnecting the limbic and cortical system.

Because TBI usually causes lesions of the frontal regions, the apathy syndrome can easily be observed in those patients. Mattson and Levin (1990) explored the relationship between frontal lobe dysfunction and closed head injury and found that lesions of frontal cortex can lead to patient apathy. Loss of spontaneity, and slow responses were observed because the patients had difficulty controlling their impulses. Following this, the anterior cingulated prefrontal cortex is the most vulnerable brain regions to trauma. In the literature, Silver & Yudofsky (1994) revealed a high prevalence rate, that 31.7% of severe TBI patients had the dis inhibition syndrome and of whom 11% reported apathy without any depressive mood. A recent study (Rao & Lyketsos, 2000) revealed a similar result, that 10% of TBI patients presented apathy without depression. In addition to high prevalence of apathy in the TBI patient group, other researchers (Anderson and Ervedoza, 2001) further found that the TBI patients' cognitive functions (e.g., memory, motor speed and executive functions) are negatively affected by their apathy syndrome.

As our limited review, the apathy syndrome can determine both the patient's cognitive functions and capacities to participate in the rehabilitation program. Hence, it is also necessary to identify this syndrome as early as possible after head trauma and to establish a standard protocol to deal with patients disturbances of motivation evident in the disease course.

The dis inhibition syndrome

Starkstein & Robinson (1997) have recognized the fact that the dis inhibition syndrome is common in patients with brain lesions. They further identified 5 subgroups of this syndrome based on clinical manifestations. Patients with motor dis inhibition usually act hyperactively, with pressured speech and decreased need for sleep. Instinctive dis inhibition is characterized by hypersexuality, hyperactivity and behavioral dyscontrol. Patients manifesting irritability and euphoria were recognized as having emotional dis inhibition. The intellectual and sensory dis inhibition, like psychotic episodes, usually present with grandiose delusions and visual hallucinations, respectively.

Many studies (Levy et al., 1988; Kumar et al., 1990; Starkstein et al., 1994) found that lesions in a specific brain region can be responsible for the presentation of the dis inhibition syndrome. Both Levy et al. (1988) and Starkstein et al. (1994) evaluated patients with frontal dementia, and found that orbitofrontal and basotemporal dysfunctions could lead to dis inhibited behaviors such as hyperphagia, hypersexuality and irritability. In addition to the evidence from demented patients with prominent frontal lobe and an amnesia, TBI patients present those behaviors at a higher frequency because the caudate and orbitofrontal cortices are the most vulnerable brain regions to trauma. In the literature, Silver & Yudofsky (1994) revealed a high prevalence rate, that 31.7% of severe TBI patients may express some dis inhibited behaviors, while mild TBI patient also had a rate of 5-7%. Prigatano (1992) also reported that two-thirds of TBI patients developed significant personality changes that cause interpersonal difficulties, irrespective of severity of injury or age at injury. Furthermore, many researchers (Kim, 2002; Alderman, 2004; Ogle & Boll, 1995; Mallet & Taylor, 1997) found that the dis inhibition syndrome not only severely compromised the patient's recovery from acute neurological symptoms but also adversely affected the patient's family and social functioning in the chronic stage.

Unlike the apathy syndrome, dis inhibited behaviors are easily identified after TBI. However, those behavioral disturbances usually keep the potential family and social support away if not treated as soon as possible after head trauma. While discussing with the patient's family the negative influences of the dis inhibition syndrome on their functional outcome as early as possible after TBI is necessary, clinicians have to develop a comprehensive treatment plan including medication, behavioral modification, and occupational therapy within a rehabilitation program.

The post-concussion syndrome

Post-concussion syndrome (PCS), such as dizziness, headache, fatigue, memory impairments, and slowed responses, are common complaints following mild TBI. Rutherford (1989) reported that the most common acute complaints were physical problems (headache and dizziness) that occurred early, while the most common chronic ones were psychological difficulties (depression, memory impairment) that occurred late in the illness. Although most mTBI patients recover quickly, usually within 3 months post-injury, many studies (Dikmen et al., 1986; Bindel et al., 1997; Gasquoine, 1997; Warinner et al., 2003) identified a small proportion of patients who continued to have difficulties more than one year following head trauma. In fact the persistent cognitive and emotional disturbances are often considered to be potential risk factors to the development of post-concussion syndrome (Finn et al., 1998; Halkier, 2004). Our recent study (Yang et al., 2007) also confirmed the above results, and further showed that dizziness persistently compromises patients' functional outcomes for periods of one week to 2 months after mTBI.

However, whether PCS is specific to mTBI or simply a nonspecific symptom cluster is still a controversial issue. Some researchers (Iverson, 2003) examined the prevalence of post-concussion-like symptoms in a sample of healthy individuals. Participants completed the British Columbia Post-concussion Syndrome Inventory-Short Form (BCPSI-SF), a test designed to measure both the frequency and intensity of ICD-10 criteria for PCS, and the Beck Depression Inventory (2nd edition). Specific endorsement rates of post-concussion-like symptoms ranged from 35% to 75% for any experience of the symptoms in the past 2 weeks. Others (Yang et al., 2007) discovered that less than 15% of the healthy participants complained of PCS. Moreover, there was significantly more headache and dizziness in mTBI patients than in the healthy control group. Ryan & Warden (2003) tried to reconcile this controversy and reported that PCS is more likely to develop and continue from multiple pre-morbid, injury-related, and post-morbid neuropathological and psychological factors. Clinicians must assess those symptoms after TBI carefully to clarify possible etiologies of the PCS, such as physical injuries, poor family support, or brain lesions.

Conclusion

In conclusion, western studies showed that neuropsychiatric behaviors, such as memory impairment, executive functions, and apathy, and dis inhibition syndrome, are not uncommon following TBI. In addition, these deficits severely detract from patients' social and occupational functioning. According to a recent study, the neuropsychiatric profile of TBI patients in Taiwan, and a rehabilitation program subsequently must be organized to improve patients' social and occupational capacities.

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References


頭部創傷後之神經行為異常

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摘要

目的：臨床上頭部創傷患者之認知缺損與情緒異常顯而易見。雖然國外已有許多研究分析該群患者之神經行為異常，國內卻未見相關議題之探討。

方法：有鑑於此，本文首先回顧過去文獻中頭部創傷病人之認知功能障礙，包括訊息處理、注意力、記憶力與執行功能等之缺損；其次則整理患者情緒與行為異常之文獻，例如：抑制困難與冷漠症候群等。

結果：對於頭部創傷患者之神經行為功能，國內尚無一套較為詳細之臨床評估模式，以利後續之復健與治療。

結論：頭部創傷、神經行為異常

不同憂鬱傾向、年齡之糖尿病患者其風險覺知與情緒狀態的特徵

林耀忠、林英雄

摘要

目的：本文探討不同憂鬱傾向和年齡屬性糖尿病患者的風險覺知和情緒狀態因素結構差異比較，嘗試釐清風險覺知與情緒狀態的正負向表徵，進而建立憂鬱傾向作為患者「生理－心理」互動狀態情緒衡鑑指標的可能性，以提供臨床心理介入健康照顧方案之參考。

方法：以北部某家醫院家庭醫學部糖尿病門診病患為樣本，進行相關量表測量，共抽取 145 名有效樣本。其中男性 75 名 (佔 52%), 女性 70 名 (52.4% 55.7%)。受測樣本的平均年齡為 61.12 歲 (標準差為 15.07), 罹患糖尿病的平均持續年數為 14.48 年 7.379, 受測樣本中有 30% (44人) 患有其他慢性病。

結果：本研究顯示，在「風險覺知」因素結構方面呈現多元性，即「知行不一」、「慣常踐行」、「不切實際」和「順其自然」。經控制「罹患糖尿病年數」與「是否尚有其他慢性病」變項後，相對低憂鬱組患者傾向「不切實際」風險覺知，雖可緩和憂鬱情緒，具功能性後果，但也可能是一種虛幻式控制。至於相對高年齡組，傾向「順其自然」風險覺知，可能基於因應策略的策略。情緒狀態的因素結構，分成「鬱煩無助」和「達觀福份」。「高憂鬱組」傾向持有「鬱煩無助」情緒，「低憂鬱組」則傾向保有「達觀福份」情緒。

結論：(1) 相對低憂鬱組患者傾向不切實際風險覺知，包含功能性和虛幻性後果。 (2) 跟其自然的風險覺知，較為符合高風險患者的因應效率。 (3) 對於慢性病患者之身心照護服務，若能從憂鬱傾向的風險覺知作為初級資料的建立，篩選出高憂鬱傾向族群，強化其自我效能和改變動機，當可逐步落實「生物心理社會」健康照護模式的臨床實踐觀點。

關鍵詞：風險覺知、憂鬱傾向、情緒狀態、年齡

前言

近年來，糖尿病為國人十大死因之一，已是常態現象 (行政院衛生署, 2006)。而且罹病年齡層逐漸降低，它不再是屬於中老年人的疾病，糖尿病成為任何生活週期的風險因子。如同於一九五五年首度創刊發行的「慢性病期刊」(Journal of chronic disease) 的創刊詞宣示，指出慢性病「不再是無望感的事件，也不是老人病」，這意味著慢性病在任何地方、任何年齡都可能發生，但只要進入醫療體系，這是可以進一步治療的疾病。然而，慢性病治療除了生理指標外，心理社會回應歷程亦十分重要。