Mild Traumatic Brain Injury.

Review of the literature and

A look at the WCB of BC data

By

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A. Mild Traumatic Brain Injury: Review of the Literature

I. Epidemiology and Background Information

The direct association between head trauma and brain injury has long been established. Ambroise Pare used the term commotio cerebri in the 16th century[1]. Pare defined commotio cerebri as a kind of short-lasting paralysis of cerebral function due to head and brain movement. In the 18th century, Alexis Littre performed a famous post-mortem which provided evidence that concussion can occur without obvious anatomical damage to the brain. He performed an autopsy on one particular patient that had been rendered unconscious and died soon after his head hit a wall. Littre detected no cerebral injury which was consistent with Pare's notion that the symptoms of concussion reflected a functional disturbance rather than structural damage such as contusion, hemorrhage or laceration of the brain[1].

At present, traumatic brain injury (TBI) is a major public health concern and a leading cause of disability worldwide[2-8]. In Canada, the incidence of people who acquire traumatic brain injury is estimated from 100/100,000 to 200/100,000, annually[84]. Based on these statistics, it is estimated that there are 6000 new traumatic brain injury cases in British Columbia annually. Twenty percent of these traumatic brain injury patients are predicted to die on the way to the hospital. Thus every year, there are 4800 survivors of traumatic brain injury in British Columbia (BC). 3840 of these 4800 survivors are estimated to be mild traumatic brain injury cases[84]. In the United States, it has been estimated that between 1.5 - 8[3-5] million people per year suffer from traumatic brain injury ranging from mild to severe. More than 1 million of these patients were treated at the emergency department[3]. It is estimated that $US 56 billion is spent annually in direct and indirect cost due to TBI. Of the 1.5 - 8 million TBI patients, between 75% - 90%[3-5] are classified as mild. In European countries, the incidence of TBI is estimated between 100/100000[8] - 1967/100000[2] person/year. In Europe, it is estimated that 80%[8] - 95%[2] of TBI patients are classified as mild with moderate and severe accounting for about 5% - 20% of all cases.

There have been numerous studies of Mild Traumatic Brain Injury (MTBI). In its effort to conduct a systematic review on various aspects of MTBI, a recent World Health Organization (WHO) Collaborating Centre Task Force on MTBI[9] had identified over 38,000 abstracts on MTBI published from 1980 to March 2002. However, only about 735 studies were judged to be eligible for review according to their criteria. Of the 735 eligible studies, only 117 were judged to be scientifically admissible for review. There were 62 guidelines on the management of MTBI (including those used in sports) but only 2 guidelines were judged to be evidence-based. The WHO researchers also found that there is a distinct lack of uniformity in diagnostic criteria and management of MTBI. Such is the state of literature on MTBI at present.

The purpose of this paper is:

- to obtain published systematic reviews on mild traumatic brain injury (diagnostic criteria, assessment and treatment of MTBI)
- in the absence of any practical high quality systematic reviews, to obtain the highest quality or grades of evidence review of MTBI.
- to form the medical portion of a more comprehensive MTBI claims management document

This paper will review, in separate sections, many different aspects of MTBI, including:

- diagnostic criteria and how this may relate to various outcomes
- natural history (including known and presumed pathophysiology and epidemiology of return to work and activities of daily life)
- diagnostic tools with “rules”, where available, suggesting when/when not to use
- post concussive syndrome including a discussion of potential “red flags” for prolonged recovery.
- medical treatment(s) with a discussion on the best available evidence
- neuropsychology and its potential role in MTBI case
- rehabilitation

In essence, this paper attempts to put practical suggestions forth, based on sound academic science where available, in an effort to deal with the multitude of medical and claims issues present when dealing with such patients/claimants in a compensation setting.

All evidence that is reviewed and discussed will have its 'level' identified where appropriate.

II. Methods

A literature review (up to June 2003) was conducted on multiple databases including PubMed, Cochrane Library Database, DARE, NICE, AHRQ, US-CDC, INAHTA and member countries for any systematic reviews on mild traumatic brain injury. The search was limited to English language literature (or the availability of English language abstract) and was done by employing keywords: mild brain injury or mild head injury or mild traumatic brain injury or mild traumatic head injury or closed head injury or closed mild head injury. The word mild was then substituted by the word minor in conjunction with the above in an attempt to broaden the scope of the search.

A non-systematic literature review was done on PubMed (up to March 2003) in order to identify any published guidelines, non-systematic review articles or primary research papers on MTBI. The search was focussed on the diagnostic criteria of MTBI, assessment and early management, objective diagnostic tools, outcome and prognostic factors, post-concussive syndrome and the role of neuropsychological assessment. Various combinations of keywords for MTBI, as mentioned above, were employed. The search was limited to English language literatures (or the availability of English language abstract).

Searches were also done on workers' compensation board websites in Canada, and Washington and Colorado State workers' compensation board in the United States. The purpose of the search was to identify medical practice guideline in managing MTBI in their respective jurisdictions.

III. Definitions of Mild Traumatic Brain Injury

Head injury (specifically, closed head injury) and traumatic brain injury (TBI) are often used interchangeably within the literature. Both are the result of direct contact or acceleration-deceleration forces applied to the head and neck area. However, it is acknowledged that only TBI is associated with loss of consciousness, retro- or antero-grade amnesia and or focal neurological signs[11].

There is a wide range in the incidence of TBI. In the US alone the estimated number of new TBI cases for the whole country varies from 1.5 million - 8 million annually[3-5]. The wide range of annual incidence estimate is probably due to the fact that an unknown proportion of MTBI victims do not seek any medical attention[3]. This wide range of incidence may also be due to the fact that there is inconsistency among researchers and organizations in defining MTBI[9-11].

MTBI has many synonyms including brain concussion, head injury, closed head injury or even simply mild head injury (as it is called in some European countries)[5,11]. Brain concussion implies a transient disturbance of neuronal function secondary to mechanical forces[5]. Table 1 summarizes some criteria that have been used in diagnosing MTBI. Our review on the literature suggests that there is no universally accepted criteria for the diagnosis of MTBI. However, despite variations in criteria between different organizations and experts, there are multiple points of agreement. Areas of agreement include a Glasgow Coma Scale (GCS) score at admission of 13-15, brief loss of consciousness (LOC), brief post-traumatic amnesia (PTA), and negative neuroimaging scans (usually CT scan) at presentation.

Initially, the GCS was formally developed to assess the depth of coma but in the case of TBI it is also used to assess the severity of head injury[18]. GCS contains crude scoring categories on which 2 patients with the same score may not function at the same level. Recent studies show that there are differences between GCS score 13, 14 and 15 in term of morbidity and mortality[19,20,26]. In a consecutive series of 3370 patients admitted to hospital with the diagnosis of mild head injury, Culota et al[19] found that 1.08%, 3.01% and 3.41% of patients with GCS score 15, 14 and 13, respectively, died. Further, they found that 4%, 16% and 28% of these patients with GCS score 15, 14 and 13, respectively, had abnormal head CT scan results varying from contusions to subarachnoid hemorrhage. 5.7%, 13.1% and 18% of these patients with GCS score 15, 14 and 13, respectively, needed rehabilitation post hospital admission. In a separate study, Gomez et al[20] found that among 2484 consecutive cases, patients with GCS score 13-14 had a significantly higher incidence of initial LOC, of skull fracture, abnormal CT findings, need for hospital admission, delayed neurological deterioration and need for operations than patient with GCS score 15. In a multivariate analysis, Gomez et al[20] found that, adjusted for age, focal signs and skull fracture, patients with higher GCS score were less likely to have abnormal CT findings. In a consecutive series of 66 patients undergoing head CT, Tellier et al[26] reported that patients with GCS score 13 had a higher percentage of abnormal scans than those with GCS score of 14 or 15. (Studies by Culota et al[19], Gomez et al[20] and Tellier et al[26] provide level 4 evidence). The Society of British Neurological Surgeons in their guidelines for the initial management of head injury[35] estimated the risk of having an operable intracranial haematoma post head injury is 1 in 3615, skull fracture 1 in 81, PTA 1 in 6700, skull fracture and PTA 1 in 29, without any risk (i.e. without evidence of skull fracture and or PTA) 1 in 31300 among patients with GCS score 15 (Level 4 evidence).

PTA is defined as the length of interval after trauma during which the patient is unable to store current events at the time of injury until the return of continuous memory. Included in this time of PTA is the period of unconsciousness, confusion and disorientation[7]. The validity of PTA as a predictor of outcome across a wide spectrum of TBI is supported by its positive relationship to acute neurological abnormalities and to the extent of brain damage[21] (Level 4 evidence). Despite its demonstrated predictive utility in research settings, data on PTA must be used with great care. In a hospital setting, PTA is unable to be assessed prospectively. Gronwall and Wrightson[22] reported that the duration of PTA may be underestimated by some patients. However, in others it may well be overestimated due to the inclusion of periods of sleep or impaired consciousness due to alcohol, drugs or medications. Despite this, in a series of mild to moderate TBI patients, van der Naalt et al[23] showed that a prospective assessment of PTA was a reliable predictor of outcome (Level 4 evidence).

Many authors have shown that loss of consciousness (LOC) is correlated with risk of cranial fracture and intracranial complications[1,20,24] (Level 4 evidence). However, there is no agreement among experts with regard to duration of LOC to define MTBI (see Table 1). In children, 100% positive outcome was observed when the duration of LOC was < 15 minutes[1]. Jennet[25] concluded that a LOC ≤ 30 min could be considered MTBI (Level 4 evidence).

Given the pros and cons on GCS, PTA and LOC, currently, there is no biologically objective measure that quantifies the severity of the neuropathology more accurately than the combination of GCS score, PTA and LOC[15] (Level 4 evidence). Interestingly, the National Institute for Clinical Excellence of England and Wales, on its currently published evidence-based guidelines for head injury, did not classify head injured patients according to level of severity (mild, moderate, severe) in its attempt to provide a standardize triage, assessment, investigation and early management of head injury in infants, children, adolescence and adults[6].

<u>In summary</u>, MTBI is a common but usually not serious injury. The majority of MTBI cases do not need any specific medical treatment. Currently, there are no standard criteria for the diagnosis of MTBI. The criteria available include an extraordinarily broad range of injury severity (i.e. the American Congress of Rehabilitation Medicine criteria[10]). Most of these criteria do not include important distinctions between subtypes of MTBI. Level 4 evidence suggests that a GCS score 13-15, brief LOC, brief PTA and probably negative head CT scan findings are probably acceptable criteria for making the actual diagnosis of MTBI.

IV. Pathophysiology, Natural History and Return to Work

IV.1. Pathophysiology

The impact of traumatic force in MTBI has been one of many subjects of debate among experts[5,11,13,15,27,28,29,30] (Level of evidence 4). Currently, the dominant theory behind the neuropathology of MTBI is that of Diffuse Axonal Injury (DAI). The clinical diagnosis of DAI is achieved by a process of diagnosis of exclusion i.e. its presence is inferred in post-traumatic LOC (coma) patients without detectable intracranial lesions or cerebral contusion on their initial neuroimaging scan.

DAI is caused by shearing forces generated within brain parenchyma by sudden acceleration-deceleration. These forces disrupt fragile structures running in the long axis of the brain. It primarily affects the axons and associated small blood vessels. Axonal injury causes localized transport failures within the axon that causes swelling and often lysis of the axon with subsequent wallerian degeneration (fatty degeneration of nerve fibers). Small vessel injury can disrupt small vessels producing pethechial hemorrhages or local/focal edema. The earliest lesions can be detected 15 hours after injury. Some of these histopathological changes seen include microglial cell proliferation and pethechial hemmorhages[13]. The extent of axonal injury is thought to correlate with the GCS score, duration of LOC and duration of PTA. The primary distribution of injury seems to be the parasagittal deep white matter spreading from cortex to brainstem. It is hypothesized that this pattern of injury is responsible for the future predominance of attention and executive deficits in even the most mildly impaired patients[15,27,28].

Oppenheimer[15,27,28] was the first to demonstrate DAI in patients with mild TBI who had died from systemic injury. He reported the destruction of myelin, axonal retraction bulbs (bead-like structures at the proximal end of a ruptured axon) and aggregates of small reactive glial cells (indicating recent tissue injury) in a variety of brain regions in 5 patients with minor or trivial injuries. Experts have been able to duplicate Oppenheimer's observation in animals. Experiments on animals show a dose-response relationship between the magnitude of the deceleration force and the size of DAI damage. However, the relationship is not a simple linear one. Animal observations also show that there is regenerative activity (e.g. sprouting and enlarged axonal areas at the tip of growing axons) over a period of weeks to several months subsequent to the injury. This regenerative process is thought to mirror the recovery period in humans. Thus, based on the DAI model, it can be concluded that the causes of MTBI are identical to the causes of more severe TBI i.e. inertial force transmission by sudden acceleration-deceleration resulting in DAI. Most experts would agree that larger forces result in 'larger' injury to brain matter.

Niess et al[29] argued that TBI is not the only and may not be the main cause of DAI. The authors examined 450 consecutive human brains that were available on routine autopsy. Samples from pons and cerebrum were immunostained with β-amyloid-precursor-protein (β-APP) in order to assess axonal damage. β-APP has been shown to be a useful marker for axonal damage in human brain tissue samples from victims of fatal head injury. Of the 450 human brains, the authors found that axonal injury was detected in 12% of all cases on which only ? had a history of TBI. The majority of the positive cases were associated with drug intoxication (mainly opiates). β-APP staining was positive in both pons and cerebrum; more in pons for the

TBI cases. The authors concluded that in a non-preselected human population, mechanical injury (TBI) is not the major cause of DAI (level 4 evidence).

Other brain tissue pathological features that can be found in MTBI cases include brain contusion and intracranial hemorrhage[5,13,15]. Contusion refers to an area of focal cortical injury that results from direct external contact forces or from the brain being traumatized against intracranial surfaces via acceleration/deceleration forces. Signs of contusion may include focal weakness, numbness, incoordination, aphasia and difficulties with memory and cognition. Cortical contusions are associated with localized edema, mass effect and poorer outcome in MTBI[1,5,15] (Level 4 evidence). Epidural, subdural, subarachnoid or intracerebral hemorrhage may complicate MTBI[5,13,15]. In general, intracranial hemorrhage occurs less often in MTBI. However, MTBI victims who are on anticoagulation therapy or have coagulopathies have an increase risk of intracranial hemorrhages. Neurological deterioration in those with MTBI suggests a progressing intracranial hemorrhage. Epidural hemorrhage may be acute or subacute in its presentation, although more often than not, it is an acute event. Subdural hemorrhage may be acute, subacute or chronic in presentation. Chronic subdural hematoma can present clinically months or even years after the initial brain insult. Chronic subdural hematoma post MTBI occurs more frequently in the elderly (Level 4 evidence). Subarachnoid hemorrhage is more common in severe TBI.

In summary, the evidence that DAI is the main neuropathological process behind MTBI is weak at best or inconclusive given the current literature. Contusion and intracranial hemorrhage can also be found among those diagnosed with MTBI (also called 'complicated MTBI'). The presence of contusion or intracranial hemorrhage will likely influence the outcome of MTBI in a negative manner.

IV.2. Natural history

Shortly after the injury, many MTBI patients show typical signs and symptoms. In general, these symptoms can be grouped into 3 categories i.e. cognitive, physical and behavioral[2,13,31,32]. Physical symptoms can manifest as headache, dizziness, insomnia, fatigue, lethargia, uneven gait, nausea/vomiting, blurred vision or even seizures. Cognitive symptoms include attention difficulty, concentration problems, memory problems, orientation problems, self-appraisal, expression and speech or language problems. Behavioral changes include irritability, depression, anxiety, sleep disturbance, problems with emotional control, loss of initiative, blunted affect, somatic preoccupation, hyperactivity, disinhibition or problems related to employment, marriage, relationships, home and or school management. The prevalence of these symptoms among MTBI patients varies from 15% - 50%[7]. The most common reported signs/symptoms are headache, neck pain, nausea, dizziness, vomiting and amnesia[2,7,13,31,32].

Van der Naalt et al[23] followed 67 mild and moderate TBI patients for 1 year and recorded the prevalence of signs/symptoms at 1 month, 3 months, 6 months and 1 year. Similarly, Chambers et al[33] followed 940 MTBI cases with complete follow-up for 3 months and recorded the prevalence of symptoms and symptom combinations. Table 2 presents the prevalence of signs/symptoms from these 2 studies (Level 4 evidence). Thus, it can be concluded that symptoms following MTBI are prevalent. However, these symptoms are also common in the general population. Mickevicience et al[34] conducted a historical cohort study with minor head injured patients as cases and sex and age-matched minor non head injured patients as controls in Lithuania. The authors interviewed 200 cases between 22-35 months post injury, obtaining a 66% response rate. This study suggested that all the MTBI cases had acute

headaches after the injury, but that the headache disappeared in 96% of cases within 1 month. Further, the authors showed that the prevalence of headache, dizziness, memory problem and concentration problem was not significantly different than the controls (Level 4 evidence).

After 7-10 days, only more complex measures of higher brain function may be abnormal in some patients. MTBI patients perform less well on complicate tasks requiring prolonged attention and rapid response times when compared with controls. However, this deficit resolves in the majority of patients by 1 month post injury[13] (Level 2 evidence). Well motivated, young patients with the mildest concussion – 'ding' without LOC – recover in a few days. The most typical MTBI patients i.e. GCS 15 in the emergency room, brief LOC, and PTA < 1 hr, recovers in 6-12 weeks if there are no complicating factors. Patients with LOC > 10 minutes and PTA > 6 hrs may require months to years to recover and some may never completely recover. Patients older than 55 years may require much longer to recover or may not recover completely from some of these deficits. Patients with very demanding jobs or demanding personalities may always be aware of the deficits in their job performance in the majority of cases. By 3 months post injury, neurological recovery is substantial as measured by commonly used neuropsychological measurements. The number of patients still limited by symptoms beyond 3 months fall significantly. By 6 to 9 months, most will continue to improve and recover. By one year, only 10% - 15% may still be symptomatic[5,13,15,27] (Level 4 evidence). These 10% - 15% patients that are still symptomatic by one year may include patients with the persistence of one troubling symptom, varying clusters of symptoms or even worsening of their entire symptom complex. This condition is called 'Persistent Post Concussive Syndrome' (PPCS) [5,15] and is described in detail in section VI.

Well recovered patients may still be susceptible to periodic impairments under certain circumstances of physiologic or psychological stress. Patients may experience increased sensitivity to modest alcohol use, sleep deprivation, lengthy travel or increased work demands[5,15].

Various experts stress the importance of the first month post injury in the management of MTBI. During this period, patients and their families need to receive proper information, education and support[5,11,15,16].

In summary, the neuronal injury inherent in MTBI, manifested as memory and attention impairment improves with no lasting clinical sequelae in the vast majority of patients. The vast majority recover within days to weeks, with a smaller proportion taking many months. A small but significant minority may have ongoing 'problems' lasting more than one year.

IV.3. Return to work

In his study on MTBI and disability using Glasgow Outcome Scale (GOS) as the outcome measurement tool, Rimel et al[36] found that 3 months after injury 78% of MTBI patients had a good recovery. Moderate disability was assessed in 22% of the patients. Once again using GOS as the outcome tool, Williams et al[37] found that 97% of the patients had good recovery, while 3% of patients had moderate recovery at 6 months post injury. An increase in 'moderate' disability was found among those who had evidence of a focal brain lesion on neuroimaging studies (Level 4 evidence).

In general MTBI patients return to work quickly after the injury. In a summary of 2660 patients from 8 countries, Binder[21] estimated that 13.65% of MTBI patients suffered long term

disability (median follow-up < 12 months). Binder also estimated a mean weighted average of time off work at 3.56 weeks (5 studies, total n = 419) (Level 4 evidence).

Dickmen et al[38] concluded that the likelihood of returning to work is directly related to the acute severity of injury. Using GCS score as the measure of severity level, 63% of mild, 44% of moderate and 13% of severe TBI returned to work at 6 months post injury. After 12 months the figures were 80%, 56% and 26% for mild, moderate and severe TBI, respectively. The authors found that orthopaedic related injuries accounted much for the disability post injury. In fact in this study, only 87% of the orthopaedic controls return to work at 12 months (compare to 80% of the MTBI) (Level 4 evidence). This study demonstrates the importance of associated injuries and co-morbid conditions when assessing return to work or disability issues in patients/claimants with MTBI.

In a prospective study of young working men, Wrightson and Gronwall[39] found that the mean time off work was about 5 days, and almost half of the patients could not resume their pre-injury duties for almost 14 days. Three months post injury, 20% still had symptoms, generally concerning memory and concentration (Level 4 evidence).

In a more current study, van der Naalt et al[23] estimated that among MTBI patients 39%, 67%, 97% and 100% returned to work at 1, 3, 6 and 12 months post injury, respectively. Overall, the mean time of resumption of previous activities was about 2.7 weeks for the MTBI patients. In this series of 66 mild (43 patients) and moderate (23 patients) patients, none was disabled to such a degree that they could not resumed their previous activities (Level 4 evidence).

It seems that even though many of the MTBI patients had resumed previous activities or return to work (either partially or completely), working at full capacities was possible only several months later; approximately 6 months post injury[21,23,36,37,38] (Level 4 evidence). Complaints temporarily increased shortly after returning to work.

Various factors have been identified that affect return to work rates[40-44] (Level 4 evidence). These include:

- the timing of data collection by researchers. Early after injury, physical limitation plays an important role in any attempt to resume major activities
- advise given by physicians not to return to work
- lack of information and encouragement given to patients surrounding return to work issues
- persisting symptoms and difficulties
- low pre-injury vocational status
- older age
- lower GCS score
- pre-injury physical and psychological difficulties, including neuropsychiatric history
- pre-injury alcohol abuse
- pre-injury lower level of motivation to work
- poor social support and coping strategies
- type of occupation. Those whose job require a high level of personal interaction or encompass frequent interruptions or need to work at several projects simultaneously are more likely to take a longer time to regain their pre-injury level of functioning

<u>In summary</u>, on average, MTBI patients require 3-4 weeks off work post-injury. Up to 97% of MTBI cases will return to work 6 months post injury. Several factors have been identified to affect duration of return to work, including pre-injury and social factors.

Only a small proportion of MTBI patients develop lingering symptoms (post concussive syndrome) and a very small percentage will develop prolonged, lingering symptoms (persistent post concussive syndrome; see section VI for an in-depth review of this topic).

V. Objective Diagnostic Tools for MTBI

Objective diagnostic tools in MTBI cases mainly involve neuroimaging modalities. At present, serum markers (i.e. serum S-100B, taken usually 6 hours post-injury) are being evaluated as a tool to objectively diagnose and categorize the severity level of TBI.

The role of neuroimaging in diagnosing MTBI continues to evolve and be debated in the literature. In general, structural imaging techniques play a role in acute diagnosis and management, while functional imaging techniques are being evaluated in an attempt to clarify the pathophysiology, symptom genesis and mechanism of recovery from MTBI[45]. Various neuroimaging modalities can be employed in helping to make the diagnosis of MTBI. Structural imaging modalities include Computed Tomography (CT) Scan, Magnetic Resonance Imaging, Volumetry, Fluid Attenuated Inversion Recovery (FLAIR), Magnetic Resonance Spectroscopy (MRS), Diffusion Weighted Imaging (DWI), Diffusion Tensor Imaging (DTI), Magnetization Transfer Imaging (MTI), Magnetic Source Imaging (MSI). Functional imaging modalities include Single Photon Emission Computed Tomography (SPECT), Positron Emission Tomography (PET) and functional MRI (fMRI) [45,46]. However, many of these modalities are still at the preliminary/research stage of development. Table 3 summarizes different imaging modalities that have been employed in the diagnosis of TBI and MTBI[45,46]. Currently, CT scan is the modality of choice as a diagnostic tool for acute MTBI[5,6,45,47,48]. Information on CT scan, MRI and SPECT are presented in the next subsection.

V.1. CT Scan

In general, the number and location of structural lesions seen on CT Scan varies as a function of injury severity. In milder injuries, abnormalities are generally limited to regions near cortical surfaces. Injury to progressively deeper structures may occur as the degree of severity progresses, particularly in relation to duration of unconsciousness[45].

A significant minority of MTBI patients have been found to have abnormalities on their CT scan and some of these patients may require surgical intervention. In her systematic review on mild head injury, SBU[53] reviewed 1028 studies on MTBI. Nine high quality studies and 22 studies of moderate quality identified a total of 25,222 patients with MTBI and normal physical findings when examined in a hospital setting. SBU found that approximately 9% of these patients had morbid changes identified by CT scan in the acute phase (Level 1 evidence). Surgery and other extensive treatments were required in 1% of the patients and mortality associated with MTBI was reported to be 0.1% (Level 1 evidence). Further, SBU concluded that it was rare to find an unexpected and negative course in patients where early CT results are normal. In 32 studies of 586 patients with complications and in a case series of 54,000 patients, SBU found only 2 confirmed and 9 possible cases of deterioration within 48 hours where early CT had been normal (Level 2 evidence). The frequency of cerebral hemorrhage from MTBI was somewhat higher among older patients and among those under the influence of alcohol. However, SBU failed to find evidence that children, the elderly or people under the influence of alcohol experienced greater benefits or were at greater risk with any given strategy (i.e. CT or not CT). The evidence did not show that their situations were more serious in relation to other patients if their CT scans were normal (Level 2 evidence). The information they obtained on how to proceed with CT scan among MTBI patients who were under anticoagulant therapy was suggested to be 'inconclusive'.

Falimisrki et al[50] on a consecutive series of 339 MTBI patients found that among 159 patients with a history of LOC and or PTA without any other identifiable symptoms related to MTBI, 11 (5.6%) were found to have acute injuries on their head CT scan (Level 4 evidence).

Stein and Spettell[24] in over 22,000 consecutive MTBI patients collected over 7 years from one institution found that 8.43% MTBI patients had complications evident on their head CT scan. Of those with complications, 1.2% deteriorated to coma and 5.9% were operated on for intracranial haematoma formation(Level 4 evidence).

In a consecutive series of 520 MTBI patients, Haydel et al[52] found that 6.92% had 'positive' head CT scans. These patients with positive CT scan were found to have short term memory deficits, were under drug or alcohol intoxication, had physical evidence of trauma above clavicle, were older than 60 years, had seizures, had headaches, vomited or were on anticoagulant therapy(Level 4 evidence).

A multicentre Canadian study on MTBI[51] found that among 3121 patients, 8.14% had an abnormal CT scan. Of those with abnormal CT scan, 47%, 35% and 18% had a GCS score of 13, 14 and 15, respectively (level 4 evidence).

Given the relatively small numbers of MTBI patient who had positive head CT scans after the injury, it is necessary, based on the clinical findings, if possible, to identify these patients who are at risk of further neurological sequalae. Based on its systematic review in June 2003, National Institute of Clinical Excellence of England and Wales (NICE) [7] recently issued guidelines on performing CT scans on head injured patients. The criteria being used by NICE are as follow (Level 1 evidence);

- GCS < 13 at any point since the injury
- GCS = 13-14 at 2 hours after the injury
- Suspected open or depressed cranial fracture
- Any sign of basal cranial fracture
- Post traumatic seizure
- Focal neurological deficit
- More than 1 episode of vomiting
- Amnesia > 30 minutes of events before impact
- Those with LOC or post injury amnesia, CT should be done immediately among:
  - Age ≥ 65 years
  - Coagulopathy
  - Dangerous mechanism of injury

It should be noted that in these management guidelines, NICE does not classify the management based on level of severity (mild, moderate, severe TBI).

Based on another systematic review, the American College of Emergency Physician concluded in 2002 that[47]:

- Cranial film radiographs were not recommended in the evaluation of MTBI. Even though the presence of a cranial fracture increased the likelihood of an intracranial lesion, its sensitivity was not sufficient to be a useful screening test (Level 2 evidence)
- Head CT scan was not indicated for MTBI patients who did not have headache, vomiting, age > 60 years, under drug or alcohol intoxication, had deficits in short term memory, had physical evidence of trauma above clavicle or had had a seizure [(the absence of these 7 criteria has been shown to have negative predictive value of 100% for intracranial lesions (Level 1 evidence)]

The Study Group on Head Injury the Italian Society for Neurosurgery[48] stated that the management of minor head injuries should centre on the risk of development of traumatic intracranial haema toma and the need to achieve early detection and evacuation of the blood clot. Based on Italian data, the Society estimated that the incidence of intracranial haematoma following minor head injury was about 1% - 3% of minor head injury patients admitted to hospital. In the absence of cranial fracture, clinical deterioration occurred in about 0.2% - 0.7% of these patients. When the minor head injury patients had cranial fractures, clinical deterioration was estimated to be about 3.2% - 10% in adults. The Society proposed that CT scans should be done among minor head injury patients with (Level 4 evidence):

- GCS 15 at admission who had LOC, amnesia, diffuse headache or vomiting with or without scalp contusion, pain in the impact area or dizziness
- GCS 14, patient confused with LOC, amnesia, diffuse headache or vomiting

Based on a systematic review, The Scandinavian Neurotrauma Committee of the Scandinavian Neurosurgical Society[6] recommended that (Level 1 evidence):

- CT scan was recommended among minor head injury patients with GCS 14-15 and or LOC ≤ 5 minutes without focal neurological deficit
- CT scan was mandatory for patients with GCS 9-13 or LOC > 5 minutes or with a focal neurological deficit (i.e. moderate head injury patient)

Based on the observation of 3121 consecutive patients, a multicentre Canadian study produced a document entitled 'Canadian CT Head Rule'[51]. Canadian CT Head Rule states that head CT scans are only required for patients with minor head injuries with any one of the following (Level 3 evidence):

- GCS score < 15 at 2 hours after injury
- Suspected open or depressed cranial fracture
- Any sign of basal cranial fracture
- Vomiting ≥ 2 episodes
- Age ≥ 65 years
- Amnesia before impact > 30 minutes
- Dangerous mechanism of injury (e.g. pedestrian struck by motor vehicle, occupant ejected from motor vehicle, fall from height > 3 feet or five stairs)

In its systematic review, Borg et al[54] from the World Health Organization Collaborating Centre Task Force on Mild Traumatic Brain Injury, concluded that there was evidence that clinical risk factors could be used to predict CT scan abnormalities, but weaker evidence that an early negative CT scan predicted clinical outcome. Thus, clinical risk factors can be used to select adult patients for CT scanning. Further the Task Force stated that cranial x-rays were not recommended for MTBI due to the poor diagnostic accuracy of cranial fractures in relation to intracranial lesions (Level 1 evidence).

Cushman et al[13], from the EAST Practice Management Guidelines Work Group, in their systematic review, concluded that CT scanning of the brain is the gold standard diagnostic imaging modality for MTBI patients. Further, the Work Group suggested that head CT scans should be performed on all patients sustaining transient disturbance of neurologic function secondary to trauma (Level 2 evidence).

In summary, < 10% of MTBI patients will have a positive CT scan of the head. In order to identify those MTBI patients who need CT scanning following injury, it is, perhaps, sensible to follow those guidelines of CT scan as set up by NICE[7] (Level 1 evidence) or the Canadian CT Head Rule[51] (Level 3 evidence).

V.2. MRI

To this date, it seems that MRI is the most sensitive imaging method for assessing MTBI. Various studies have demonstrated that MRI detects more lesions than CT in MTBI patients, especially if performed shortly after injury[45]. However, it should be noted that from a practical and logistical standpoint CT scanning is more accessible and readily utilized than is MR imaging. Levin et al[55] compared CT and MRI in 11 patients with MTBI (GCS 13-15). Overall, MRI showed 44 more intracranial lesions than CT scans. The estimate of lesion size were larger on MRI. Correlation was observed between lesion location, size and neuropsychological performance. More importantly, at 1 and 3 months follow-up on a sub-sample of these patients, it was suggested that a marked diminution of lesion size occurred in association with significant improvement in neuropsychological measures (Level 4 evidence).

In another, older study (1992), by using serial MRI and cognitive testing, Levin et al[56] were able to demonstrate that the resolution of structural lesions was associated with the improvement of cognitive functioning. A study by Godersky et al[57] also suggested the same phenomenon (Level 4 evidence).

Voller et al[58] suggested it may be indicated to undertake MRI in the first 2 weeks following injury in order not to miss acute lesions.

The Workers' Compensation Board of Colorado has recommended that MRI is the diagnostic imaging of choice to detect late alteration in neurologic function. They felt that MRI should be done among MTBI patients who failed to recover within the expected time frame[12].

In summary, MRI is more sensitive than CT scan in demonstrating structural lesions and abnormal brain tissue in both acute or chronic cases. A variety of MRI parameters show promises in demonstrating different types of lesions. The type of lesions and the interval from injury to imaging will impact the utility of a given MRI technique. Despite this and recognizing CT scanning is much more widely available than MRI, CT scanning will likely continue to be the initial, acute neuroimaging modality of choice for the foreseeable future. Consideration should certainly be given to MR imaging in MTBI patients who have prolonged recovery.

V.3. SPECT

Many studies have explored the utility of SPECT in TBI, however these studies were done mainly among moderate, severe or mixed injury patient populations. Most studies concluded that abnormalities on cortical perfusion could be shown even in the absence of structural abnormalities and flow deficits observed with SPECT may more accurately reflect the size or extent of the damage tissue than CT scan did[45] (Level 4 evidence). On a series of 53 patients (20 with MTBI), Gray et al[59] showed that SPECT demonstrated more abnormalities than CT scan. 60% of MTBI patients had perfusion deficits, where CT scan only detected 25% of these same patients. However, the clinical significance of the perfusion deficits demonstrated on SPECT has not been consistently reproduced by others[60-62] (Level 4 evidence).

On a review of the use of SPECT as a diagnostic tool in MTBI, Davalos and Bennett[63] stated that SPECT was not a reliable tool for differentiating damage based on head trauma versus preexisting trauma or coexisting damage. The authors concluded that SPECT might be a useful tool in the detection of MTBI and treatment planning. However, due to the lack of consensus regarding SPECT's sensitivity further studies were needed to solve this issue (Level 2 evidence).

The Workers' Compensation Board of Colorado recommended[12] that SPECT might be useful in MTBI patients who were still symptomatic 6 months post-injury (Level 4 evidence).

In summary, SPECT seems to be promising as a diagnostic tool for MTBI. However, at present its value is still limited to that of a research tool.

VI. Post Concussive Syndrome and Persistent Post Concussive Syndrome

VI.1. Definition

Somatic, affective and cognitive symptoms (as described previously in the section on Natural History) may complicate the recovery period after MTBI. These symptoms are often brought to the attention of physicians days, weeks or event months after the injury. The most common symptoms appear to be headache and dizziness. Other common symptoms include sleep disturbance, neck pain and emotional or cognitive symptoms as mentioned previously. The post concussion syndrome (PCS) refers to the ongoing occurrence of several such symptoms that gradually taper in severity over time. PCS may be the result of direct brain injury or from trauma involving other head and neck structures. At present, there is no widely agreed upon diagnostic criteria for PCS. The presence of some of the symptoms listed previously after MTBI is presumed to be evidence of PCS[5,15,21,27]. Ten percent to 15% of MTBI patients may still be symptomatic 1 year after the injury. The symptoms may include one persistence troubling symptom, varying clusters of symptoms or even worsening of the entire symptom complex. This condition is called persistent post concussive syndrome (PPCS) [5,27]. Thus, PCS and PPCS is a continuum of symptoms across time that occurs in a minority of patients.

The 4th edition of the Diagnostic and Statistical Manual of Mental Disorders[64] states that the essential feature in PCS is an acquired impairment in cognitive functioning, accompanied by specific neurobehavioural symptoms, that occurs as a consequence of closed head injury of sufficient severity to produce significant cerebral concussion. In order to establish a uniform research criteria for the diagnosis of PCS, DSM-IV listed several criteria that have to be fulfilled. These criteria include;

a) A history of head trauma that has caused significant cerebral concussion
b) Evidence from neuropsychological testing or quantified cognitive assessment of difficulty in attention (concentrating, shifting focus of attention, performing simultaneous cognitive tasks) or memory (learning or recalling information)
c) ≥ 3 of the following occur shortly after the trauma and last at least 3 months:
    - becoming fatigued easily
    - disordered sleep
    - headache
    - vertigo or dizziness
    - irritability or aggression on little or no provocation
    - anxiety, depression or affective lability
    - changes in personality (e.g. social or sexual inappropriateness)
    - apathy or lack of spontaneity
d) the symptoms in b and c have their onset following head trauma or else represent a substantial worsening of preexisting symptoms
e) the disturbance causes significant impairment in social or occupational functioning and represents a significant decline from a previous level of functioning. In school age children, the impairment maybe manifested by a significant worsening in school or academic performance dating from the trauma
f) the symptoms do not meet criteria for dementia due to head trauma and are not better accounted for by another mental disorder (e.g. amnesic disorder due to head trauma, personality change due to head trauma)

VI.2. Risk factors for PCS and PPCS

Various factors have been identified in the literature as possible risk factors for the development of PCS or PPCS. In his review of 'older' literature, Binder[21] identified older age, premorbid psychological problems, occupational status, educational status, sex, history of previous head injury and fat embolism as the major risk factors for the development of lingering symptoms among MTBI patients. Some of these risk factors have been identified as well from the more recent studies. Advanced age is associated with poorer outcome after head injury. However, there is a possibility that the relationship between age and poorer outcome is confounded by the level of severity of injury itself. In 1961, Russel and Smith reported that older age was related to longer PTA which in turn was related to poorer outcome. There was noted a strong relationship between pre-morbid psychological problems and poor outcome in these MTBI patients. A study conducted in France in 1991 showed that MTBI patients with pre-morbid psychological conditions were 2.5 time more likely to be unemployed than MTBI patients without preexisting problems. Further, it has been shown that a pre-injury history of depression was associated with depression following TBI. Two separate studies[21] conducted in Virginia in 1981 and in Sweden in 1974 showed that there was a relationship between higher occupational status and better prognosis after MTBI. It has also been shown that patients with a lower level of education have poorer outcome neuropsychologically, symptomatically and occupationally after whiplash injury. Two studies[21] from Ireland and one from the Netherlands showed that females were more likely to have poorer outcomes. The relationship between previous head injury and poor outcome following MTBI has been shown in a large population study. Carlsson et al[21] showed that there was a significant difference on some cognitive measures between men with multiple injuries and those with one injury. However, the effect size was negligible.

In her study on factors influencing outcome after MTBI, Ponsford et al[65] found that there was a significant difference in the occurrence of headache, dizziness, fatigue, visual difficulty, and memory difficulty among MTBI patients and controls. There was no significant difference in terms of noise intolerance, irritability, concentration, judgement, anxiety and sleeping difficulties at 1 week post injury. At 3 months there was no significant difference in the occurrence of all these symptoms between MTBI cases and controls. Among MTBI patients who were still symptomatic after 3 months post injury, the authors found that this group of patients was more likely to have a history of previous head injury, pre-morbid neurological or psychiatric problems, to be students, females and to have been injured in a motor vehicle accident (Level 3 evidence).

Adjusted for age, sex and treatment (bed rest within 10 days after trauma), de Kruijk et al[66] found that patients who reported headache, dizziness or nausea in the ER were twice as likely to have PCS compared to those without. Further, those with increased serum S-100B levels or neuron specific enolase (taken at 6 hours post injury) were twice as likely to suffer from PCS compared to those with lower serum S-100B levels (Level 3 evidence).

In an emergency room population, Bazarian et al[16] found that females were almost 8 times more likely to have PCS at 1 month. The presence of both antero- and retro-grade amnesia in the same patient, higher digit span forward scores and Hopkins verbal learning A scores have been shown to have protective effects against the occurrence of PCS at 1 month. At 3 months, the presence of both antero- and retro-grade amnesia and high digit span forward score again were found to be protective. The authors failed to identify any risk factors associated with PCS at 6 months (Level 3 evidence).

Adjusted for age, sex, educational status and employment status, Savola and Hillborn[67] found that MTBI patients with cranial fractures were 8 times more likely to develop PCS one month after injury. Those who had dizziness and or headache were almost 3 times more likely to develop PCS at one month. Furthermore, those with serum S-100B (taken within 6 hours post injury) ≥ 0.50 µg/l were almost 6 times more likely to develop PCS at 1 months post injury (Level 3 evidence).

The WHO collaborating centre task force on mild traumatic brain injury[68] concluded that for adults, cognitive deficits and symptoms are common in acute stage but the majority of studies being reviewed reported recovery within 3 to 12 months. Where symptom persists, studies have consistently identified compensation/litigation to be a factor. The task force found little consistent evidence among other predictors (Level 1 evidence).

VI.3. <u>PCS, PPCS in chronic pain patients and healthy individuals.</u>

Symptoms such as headache, memory and concentration problems, dizziness, ringing in the ears and hypersensitivity to noise are not specific and cannot be used to diagnose PCS or mild traumatic brain dysfunction. Studies had shown that even normal control subjects also complain of these symptoms. Further, studies have shown that factors differentiating late PCS symptoms onset from early onset was greater frequency of depression and compensation claims in the late onset patients[15,21] (Level 4 evidence).

Smith-Seemiller et al[69] compared 63 patients with chronic pain and 32 with MTBI by using the Rivermead Post-Concussion Questionnaire (RPCQ). The authors stated that there was no significant difference found for total RPCQ scores. There were some differences in the proportion of patients endorsing specific symptoms such as noise sensitivity, sleep disturbance, memory problems, double vision or restlessness. However, most people with chronic pain endorsed symptoms consistent with PCS. The authors concluded that PCS symptoms were not unique to post MTBI. PCS symptoms might be seen in conditions such as chronic pain (Level 3 evidence). Similar conclusions were obtained by Iverson and McCracken[85] on their study on 170 patients with chronic pain.

In their recent study, Iverson and Lange[50] investigated the prevalence of post concussive like symptoms among healthy individuals. 104 healthy individuals completed the British Columbia Postconcussion Symptom Inventory Short Form (BC-PSI-Sf). Specific endorsement rates of postconcussive like symptoms ranged from 35.9% to 75.7% for any experience of the symptoms in the past 2 weeks and from 2.9% to 15.5% for the experience of more severe, 'clinically significant' symptoms. Symptoms reported on the BC-PSI-Sf also showed a moderately high correlation with self reported depressive symptoms ($r=0.76$, $p < 0.01$). The authors concluded that postconcussive-like symptoms were not unique to MTBI, were commonly found in healthy individuals and highly correlated with depressive symptoms.

<u>In summary</u>, PCS symptoms are not unique to MTBI. The symptoms occur frequently in day to day life among healthy individuals and also found often in persons with other conditions such as chronic pain or depression.

VII. Management of MTBI

The National Institute for Clinical Excellence[7] stated that the primary patient outcome of concern in managing MTBI is 'clinically important brain injury'. However, little is known about the optimal treatment of MTBI and PCS[27]. Various guidelines and studies suggest it is important to document baseline neurologic examination findings including cognitive and emotional states. Patients and families need to be educated at the first visit (as early as possible) regarding rationale for treatments and expectations regarding outcomes[2,5,6,11,12,27,70,71,72,73].

In its systematic review, the WHO collaborating centre task force on mild traumatic brain injury[72] concluded that there was some evidence on the value of early educational intervention in an attempt to reduce long term complaints. Early physical activity and observation at home instead of in hospital resulted in shorter time off work. However, the task force also concluded that written instructions were frequently not followed (Level 1 evidence).

NICE[7] in her systematic review based guideline on the management of head injury recommended (Level 1 evidence):

- that no patients presenting with head injury should be transferred to the community until they have achieved GCS score 15
- all patients with any degree of head injury who were transfer to the community should receive verbal advice and a written head injury advice card (Appendix 1). The details of the card should be discussed with the patients and their care givers.
- Patients and care givers should be alerted to the possibility that some patients may make a quick recovery, but go on to experience delayed complications.

The US-CDC[2] stated that considerations of physical, emotional and or behavioural signs and symptoms should guide management plans (Level 4 evidence). Management plans might include:

- Evaluating and treating patients who present early for somatic complaints and documenting baseline neurological findings, including cognitive and emotional state
- Assessing the ability of patient to return to everyday activities such as sports, work or operating motor vehicle
- Educating patients and their family members about the treatment plan and expected outcomes
- Prescribing medication, as appropriate, for significant anxiety or depression
- Referring patients, as appropriate, to neurologists and or psychiatrists when emotional or cognitive symptoms interfere with normal routines and relationships
- Referring patients to specialized multidisciplinary cognitive therapy programs as appropriate. Such programs may include psychotherapy, occupational/vocational, or adaptive strategy training
- Providing copies of patient materials, 'Head Up: Preventing Brain Injury' (Appendix 2) and 'Facts about concussion and brain injury' (Appendix 3)

In their medical treatment guidelines for traumatic brain injury, the Colorado Workers Compensation Board[12] (Level 4 evidence) stated that education of the patient and family, as well as the employer, policy makers and community should be the primary emphasis in the treatment of TBI and any subsequent disability. Practitioners were encouraged to develop and implement effective strategies and skills to educate patients, employers, policy makers and the community as a whole. Further, the Board stated that an education-based program should always start with inexpensive communication providing reassuring information to the patient.

No treatment plan was felt to be complete without addressing any issues of the individual and or group providing patient education as a means of facilitating self management of symptoms and prevention of PCS. Interventions should emphasize patient responsibility. Interventions, such as therapeutic exercise and or functional treatments were generally emphasized over passive modalities. The Colorado Board recommended that treatment should be re-evaluated every 3-4 weeks. With regard to the issue of return to work, the Colorado Board stated that return to work was not necessarily contraindicated in most cases. Return to work may be therapeutic assuming the work was not likely to aggravate the basic problem or increased symptoms. With regard to PCS, the Colorado Board recommended that psychological evaluation, interdisciplinary treatment and vocational goal setting should be initiated for MTBI patients who failed to make expected progress 6-12 weeks post injury. The Board expected 3% - 10% of MTBI cases would not recover within this time limit.

In its guideline for management of mild and moderate head injury, the Scandinavian Neurosurgical Society[6] recommended that all patients received written instructions on head injury at the time of discharge (Appendix 4). These instructions contained information on signs and symptoms of acute intracranial complications, cause and natural course of PCS. Further, the Society stated that routine follow-up was not recommended. However, patients with persistent symptoms should see their family doctors (Level 4 evidence).

De Kruijk et al[11] concluded that patients with MTBI discharged from the emergency department should be given instructions for further management. The authors recommended the application of Oxford Head Injury Service (OHIS) education intervention material. The OHIS was developed under the premise that emotional factors played an important role in recovery from MTBI (Level 4 evidence). (Note: EBPG is currently contacting the authors to get a copy of the OHIS protocol)

Ponsford et al[65] conducted a controlled trial among MTBI patient in order to investigate the impact of early education on the occurrence of PCS at 3 months post injury. 79 MTBI patients were given a booklet at the time of discharge and were seen again at one week post injury while 123 were not given any information at all and were not seen at one week post injury. The booklet outlined the symptoms associated with mild head injury and suggested various coping strategies. At 3 months follow-up, patients who were given information booklet reported less symptoms and were reported as significantly less stressed (Level 3 evidence).

Mittenberg et al[74] conducted a randomized controlled trial among MTBI patients. The control group received standard hospital treatment and discharge information, while in addition the intervention group received a booklet entitled 'Recovering from Mild Head Injury: A guide for patients'. The booklet was intended to support the reattribution of symptoms to selective attention, normal responses to stress and anxiety arousing or depressive self statements. At 6 months follow-up, 28% of control group patients developed PCS whilst only 11% of the treatment group developed PCS (PCS was diagnosed according to ICD-10 criteria) (Level 2 evidence).

A randomized controlled trial in UK also reported similar outcomes[75]. In this particular study, patients were given an information booklet that described PCS and how to manage them, the likely prognosis and recovery times, stress reduction techniques, method for coping with memory and intellectual inefficiency and advice on graded return to normal levels of activity. Patients were also given advise on this issue by the care givers who provided reassurance that the injury was mild, that transient PCS was normal and that stress or anxiety could make the symptoms even worse (Level 2 evidence).

<u>In summary</u>, early intervention and education towards MTBI patients and their care givers is perhaps the best available treatment for acute MTBI and for preventing/reducing the development of PCS in recovering MTBI patients. Management of MTBI may involve various health care professionals including family doctors, behavioural psychologists, clinical psychologists, neuropsychologists, neurologists, psychiatrists, neuro-ophthalmologists, neurosurgeons, nurses, occupational therapists, physical therapists, physiatrists, ophthalmologists, optometrists, rehabilitation counsellors, social workers, speech therapists or recreational therapists.

VIII. The Role of Neuropsychology in the Management of MTBI

VIII.1. Neuropsychology testing

As a science, neuropsychology is defined as the study of brain - behaviour relationships. Clinical neuropsychology as a practice is the application of these brain - behaviour relationship principles to the individual patient for assessment, treatment and rehabilitation purposes[76]. Neuropsychological diagnosis involves systematic collection of human performance data to aid in drawing conclusions about brain function in patients suspected of having neurological or psychiatric disease. Much data used in neuropsychological diagnosis are derived from the patient's history, from observations of the patient in structured and naturalistic settings and from the results of standardized procedures and normed tests. Conclusions made by neuropsychologist are based on clinical case analysis and empirical research on brain and behaviour relationships[77].

MTBI can result in a variety of cognitive, behavioural and physical symptoms that can be extraordinarily difficult to assess and treat. Thus, it may be necessary to refer MTBI patients to psychologists or neuropsychologists, in particular, for various reasons including identifying persistent symptoms, to evaluate progress, to identify symptoms that require treatment or management and perhaps for making plans to maximize long term cognitive and overall functional outcomes. It may also be important to conduct neuropsychological assessment when MTBI patients fail to improve, when the degree of disability is disproportionate to the clinical history, where the nature of the patient's occupation necessitates more extensive testing prior to vocational re-entry or when there is a need to assess whether the patient's condition has plateaued[12,76,77] (Level 4 evidence). However, in a meta-analysis on the effect of neuropsychological symptoms on recovery from MTBI, Binder et al[78] reported that positive neuropsychological test results had low positive predictive value (range 0.11 - 0.32) and high negative predictive value (range 0.98 - 0.99) across different sensitivity (range 0.70 - 0.90) and specificity (range 0.70 - 0.90) values in diagnosing brain injury in cases with chronic disability post MTBI. Implications arising from this meta analysis include those that suggest clinicians are more likely to be correct when not diagnosing brain injury than when diagnosing a brain injury in cases with chronic disability after MTBI (Level 1 evidence). It is well known that many MTBI patients suffer from PCS with subsequent low scores on neuropsychological testing during the first week post injury. However, studies have shown that neuropsychological testing can not reliably detect long term cognitive deficits in MTBI patients. This evidence has been shown in studies among trauma patients, athletes, children and the elderly[30] (Level 4 evidence).

In its attempt to screen MTBI cases who may require further treatment, the Quebec Automobile Insurance Board implemented a routine screening program in acute care settings[78]. MTBI patients were screened by a nurse, within 7 days of injury, using a French version of the Rivermead Post Concussion Symptoms Questionnaire. The patient was considered 'positive' when the Rivermead questionnaire listed any symptoms with score ≥ 2. Positive cases were then evaluated by a neuropsychologist, within 2 weeks of screening, using a selected battery of tests chosen based on the patient's complaints. Patients with positive results from the second screening were then referred to specialized rehabilitation teams in an effort to treat persistent symptoms. A pilot program was implemented among 724 patients. 202 (27.9%) patients had a positive first screening. Of these, 144 (71.3%) underwent neuropsychological testing. Neuropsychological testing was positive in 120 (83.3%) patients. 93 (77.5%) patients who positive on the neuropsychological testing required further treatment compared to 10 (41.7%) of

patients with negative neuropsychological test. Thus, it seems that a simple early screening program delivered by a nurse can identify MTBI patients that are more likely to require further treatment (Level 3 evidence).

The Colorado Workers' Compensation Board's[12] policies on psychology or neuropsychology assessment/treatment are as follows (Level 4 evidence);

- Between 1-3 months post injury.
  Serial testing with specialized MTBI batteries would usually be appropriate and sufficient to investigate recovery progress. The administration of full neuropsychological test battery was recommended when the patient failed to improve, when the degree of disability was disproportionate to the clinical history, when it was necessary to provide more extensive testing as required by the patient's occupation
- ≥ 3 months post injury.
  Neuropsychological evaluation was indicated when patients' effort to cope with their symptoms failed, or when secondary psychological symptoms (e.g. intolerance to certain types of environmental stimuli or reactive depression) were problematic

The recommendation regarding the timing and purpose of neuropsychological intervention from the Colorado Board is considered to be level 4 evidence (expert opinion) at best. A recent systematic review conducted by the World Health Organization collaborating centre task force on mild traumatic brain injury concluded that there was no consensus on timing and utility of neuropsychological testing in the management of MTBI[9] (Level 1 evidence).

In summary, given the high negative predictive value of neuropsychological test results, it may be appropriate in given cases to conduct neuropsychological testing in the earlier post injury phase in order to evaluate ongoing symptomatology and how these may or may not relate to MTBI. Given the evidence that neuropsychological testing can not reliably detect long term cognitive deficits, it may not always be appropriate to conduct neuropsychological test in MTBI patients after 3 months post injury.

VIII.2. Malingering

Neuropsychological evaluation can yield sensitive data on outcomes related to TBI. However, neuropsychological test procedures evaluate cognitive functions that may be impaired due to brain injury or to non-neurological, psychological or motivational factors[79]. Neuropsychological assessment involves collecting information in the form of symptom reporting and test performance. Some authors suggest that this type of information can be controlled by patients who wish to appear less functional than they truly are. Further, poor effort from patients can also distort the true value of many such tests. As such, assessment of symptom validity may be a critical part of forensic neuropsychological evaluations as has been suggested by numerous authors[76,77,79,80].

Based on natural history data, MTBI patients would be expected to have less symptoms and to perform better on neuropsychological testing over time. When patients report worsening symptoms or perform worse on the neuropsychological tests over time, this phenomenon may not be due to the biological effects of the initial injury or insult. Other factors such as chronic pain, depression or involvement in litigation/workers' compensation may need to be considered

as playing a role[30]. Malingering (sometimes also called symptom invalidity) is defined as the intentional production of false or greatly exaggerated symptoms for the purpose of attaining some identifiable external reward[79]. Some areas of potential exaggeration include pain, stiffness, dizziness, depression, memory disturbance, poor concentration, personality changes, blindness or visual loss, numbness, mobility restriction or range of motion, amnesia[79,83].

In a meta-analysis undertaken to assess the impact of financial incentives on recovery after closed head injury, Binder and Rohling[81] found that financial incentives had a moderate overall effect size on symptom persistence (slower recovery). This moderate effect size was clinically significant and the effect was particularly strong for those with mild head trauma. The data showed more abnormalities and disability among patients with financial incentives despite less severe injuries (Level 1 evidence). Similar conclusions were reached by Rohling et al[82] on the subject of chronic pain. However, the effect size of chronic pain was about 30% more than closed head injury (Level 1 evidence). Currently, there is no similar evidence on the development of long-term neuropsychological problems following uncomplicated MTBI[83]. Studies of MTBI that included control groups found that neuropsychological deficits following MTBI usually resolves within 1-3 months post injury[73,83,].

There is no single neuropsychological measure that can reliably detect symptom invalidity (malingering). Experts suggested that multiple sources of information and methods need to be employed in any case analysis[80,83]. These include understanding the relationship between the severity of injury and typical courses of cognitive and psychological outcome, ruling out other medical conditions that may influence neuropsychological status, identifying test performance patterns associated with incomplete effort or feigned impairment and understanding the role of psychosocial factors in MTBI cases. Various neuropsychological tests have been developed in an effort to detect symptom invalidity.

<u>In summary</u>, there is strong evidence that financial incentives have a moderate effect in the persistent of symptoms among MTBI patients. As such it may be necessary to conduct symptom validity testing among MTBI patients who have lingering symptoms.

IX. Rehabilitation in MTBI

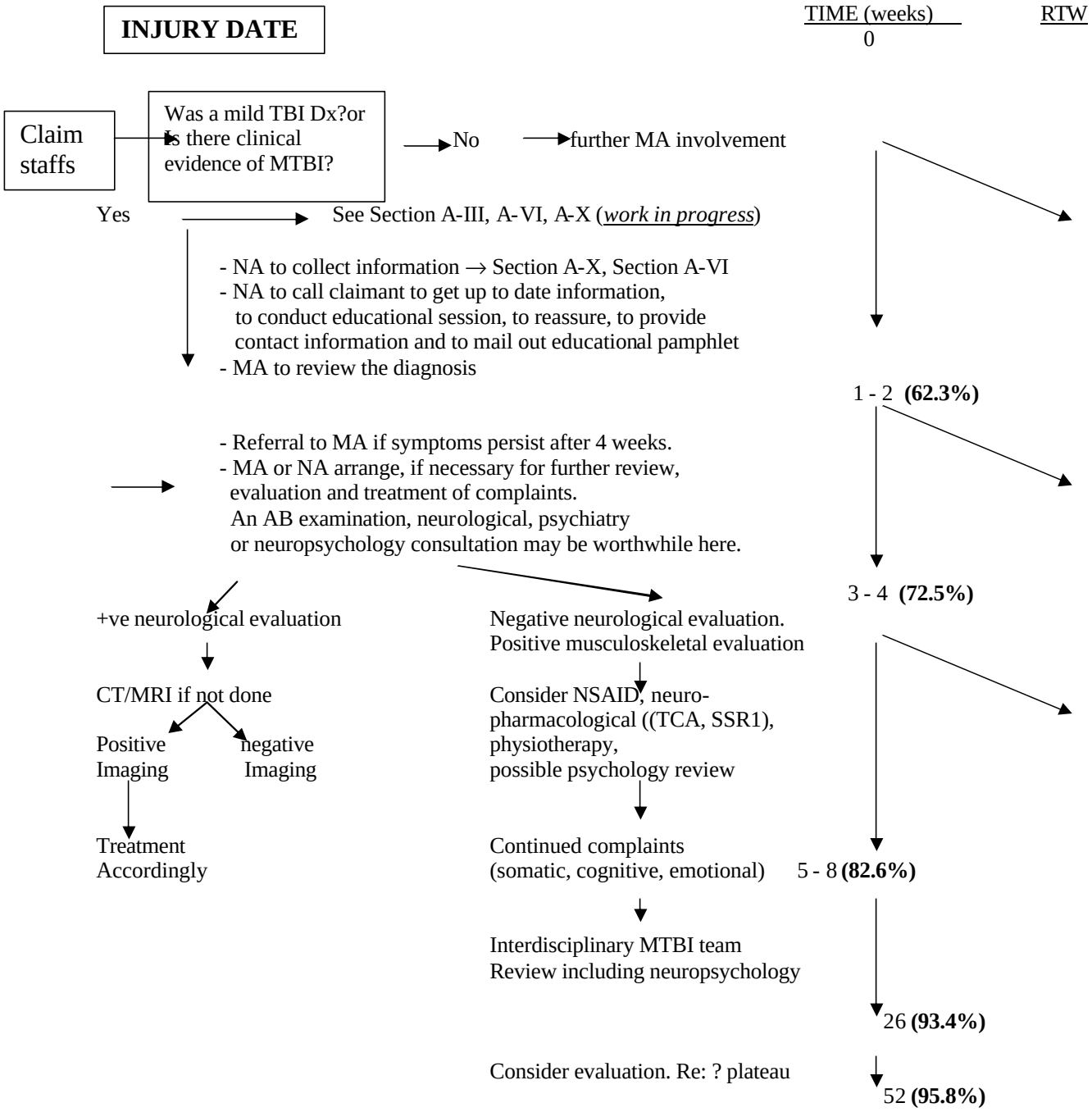
In general, TBI case management is a collaborative process that assesses, plans, implements, coordinates, monitors and evaluates the options and services required to meet an individual patient's health needs using communication and available resources to promote quality, cost-effective outcomes[(12)].

TBI case management operates with an underlying premise that when a patient reaches their optimum level of wellness and functional capability, everyone benefits including the patient and their family members, the health care system, the insurance carrier and society in general.

Thus, the primary functions of TBI case management are:

- to maximize patient and family understanding, compliance, and treatment outcomes through education and support
- to advocate for patient wellness and autonomy through advocacy, communication and identification of service resources
- to optimize patient access to appropriate health care services
- to integrate and coordinate service delivery by multiple providers and to prevent fragmentation of services
- to predict and avoid potential complications

X. Management of MTBI at the WCB of BC: Practical Issues



NB. MA = Medical Advisor NA = Nurse advisor
Bold percentage numbers on the right column show the percentage of MTBI related claims that had been off STD during that period. The goal of the new MTBI model is to meet or exceed this rate.

B. Mild Traumatic Brain Injury: WCB of BC Data

I. Introduction.

In the second part of this paper, the Evidence-based practice group presents the results of data analysis on MTBI claims submitted to the WCB of BC during the period of 1987 - 2001. The purpose of this analysis is to present the epidemiology of MTBI within the context of the WCB of BC. Results on incidence, injury distribution and outcomes including claim cost will be presented.

II. Material and Methods.

Data were extracted from the WCB of BC data warehouse (DSSEDW database) from tables 'CLAIM', 'ACCDT', 'ICDMD', 'INJRY', 'BDYPT' and 'SRIJT' in January 2003 by employing Crystal Report®™ software. The extraction was based on criteria using (ICD 9 code of '08500, 08501, 08502, 08503, 08504, 08505, 08509') and or (nature of injury 'concussion') and or (body part 'brain').

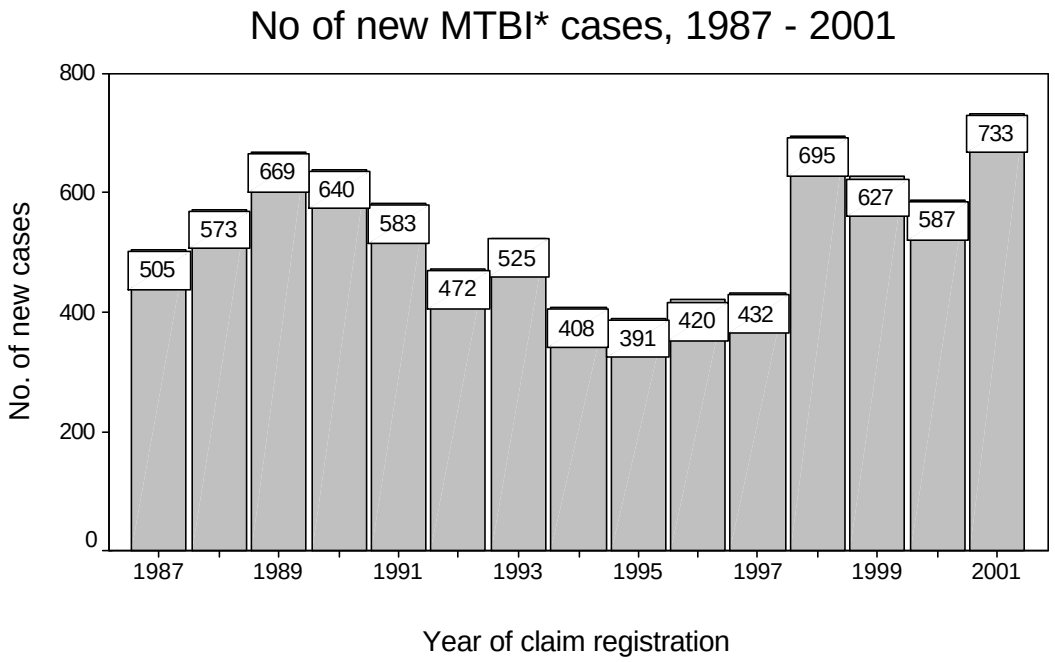
The data was originally extracted in text format, which was then translated into SPSS data format. All data was then analyzed by employing SPSS for Windows®™ ver. 11.0. For the purpose of this report, analysis was limited to data from the period of 1987 - 2001 (15 years). The year of 1987 was chosen as a starting point based on prior observations that data collected prior to this year was incomplete and less valid.

III. Results.

III.1 Epidemiology of MTBI at the WCB of BC.

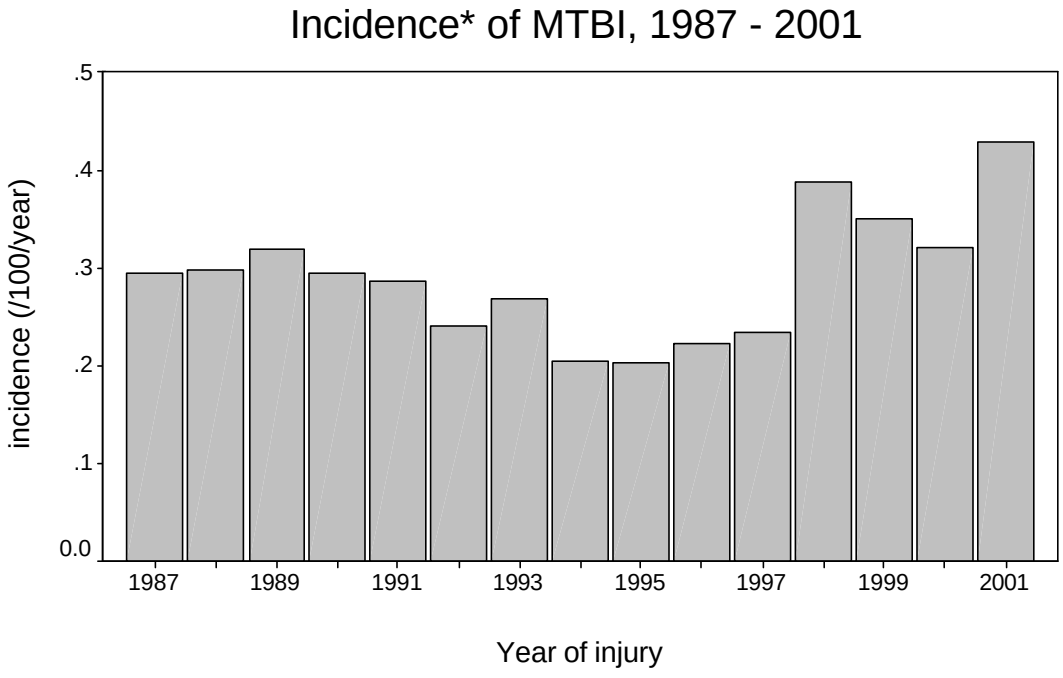
In the period of 1987 - 2001, 8260 claims were filed under MTBI. Of 8260, 27 (0.3%) patients/workers were classified as having sustained a brief loss of consciousness. The majority (98.6%) had 'concussion' coded (body part 'brain') as the nature of injury. Only 46 (0.6%) claimants were registered as having multiple injuries. During the 15 year period 1987 - 2001, on average, there were 551 new claims filed as MTBI (range 391 - 733) (Figure 1). The average incidence rate of MTBI annually was about 0.3% (range 0.20% - 0.43%) (Figure 2). However, the average cost of claims for each MTBI claim was at least double the average cost of all other claims in the same year (Figure 3A), while the median cost was at least 3 times the median cost of overall claims during the same year (Figure 3B). Overall, MTBI claims represent approximately 1% - 2% of the overall claim costs during any given year (Figure 4).

Figure 1. Number of new MTBI cases, 1987 - 2001.



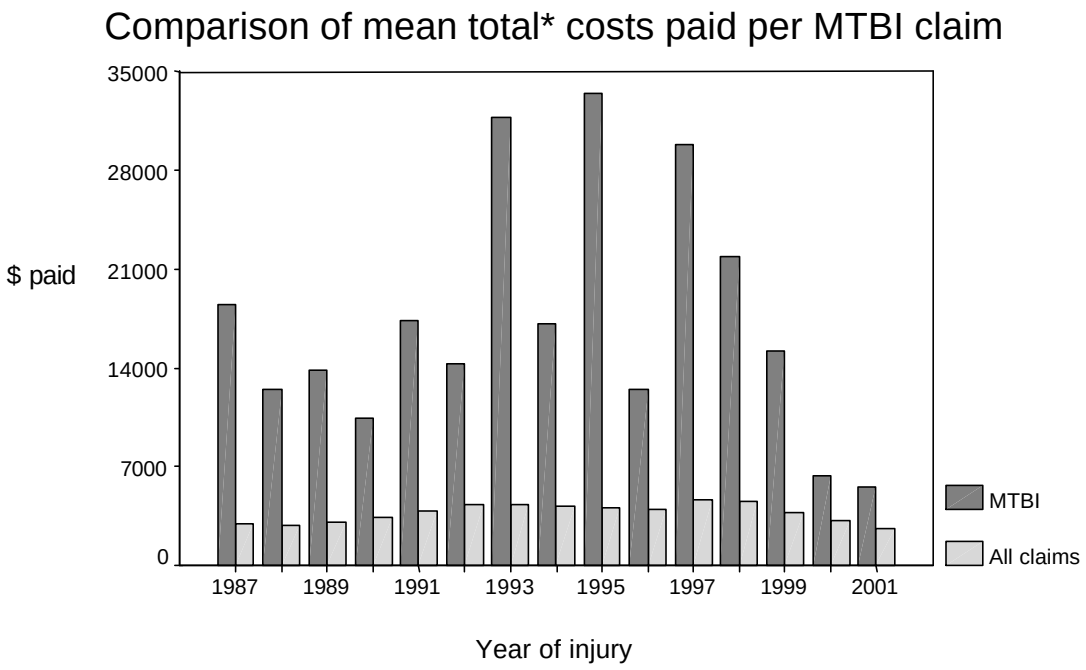
* MTBI is defined as ICD9 code 0850.0 or 0850.1

Figure 2. Incidence of MTBI, 1987 - 2001.



*incidence is defined as number of new MTBI/total claim on the same year

Figure 3-A Comparison of <u>mean</u> total costs paid per MTBI claim.



* Total refers to all health care, wage loss, rehabilitation and pension costs.

Figure 3-B Comparison of <u>median</u> total costs paid per MTBI claim.

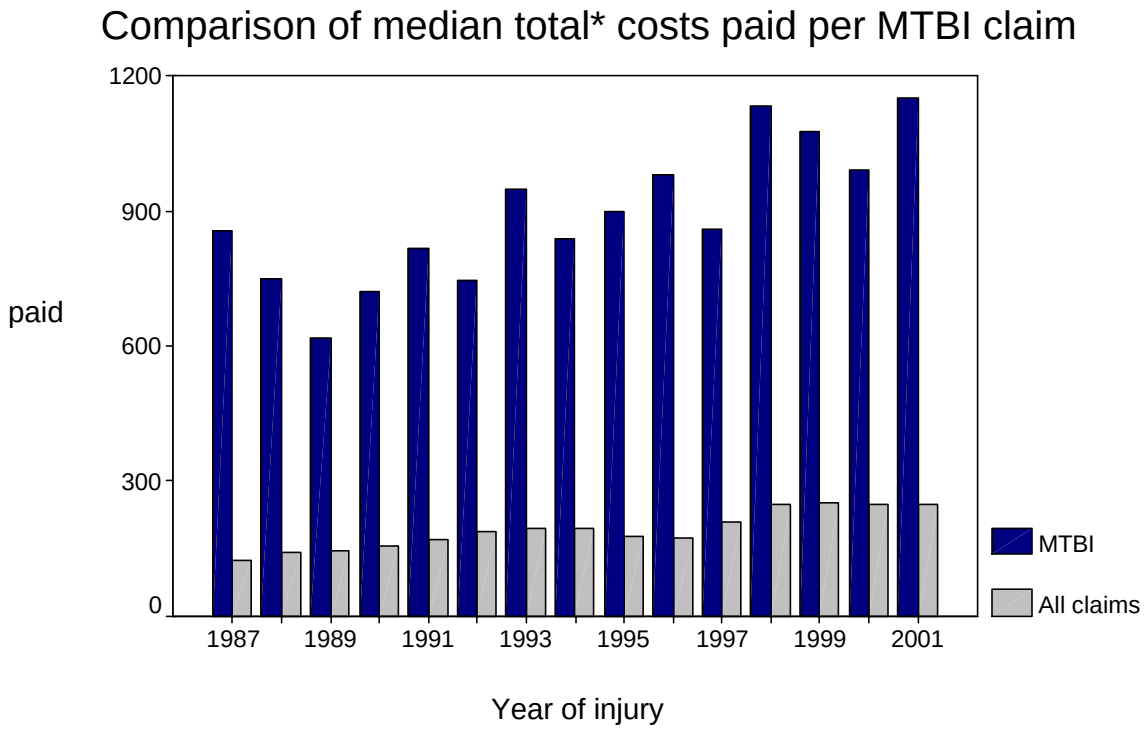
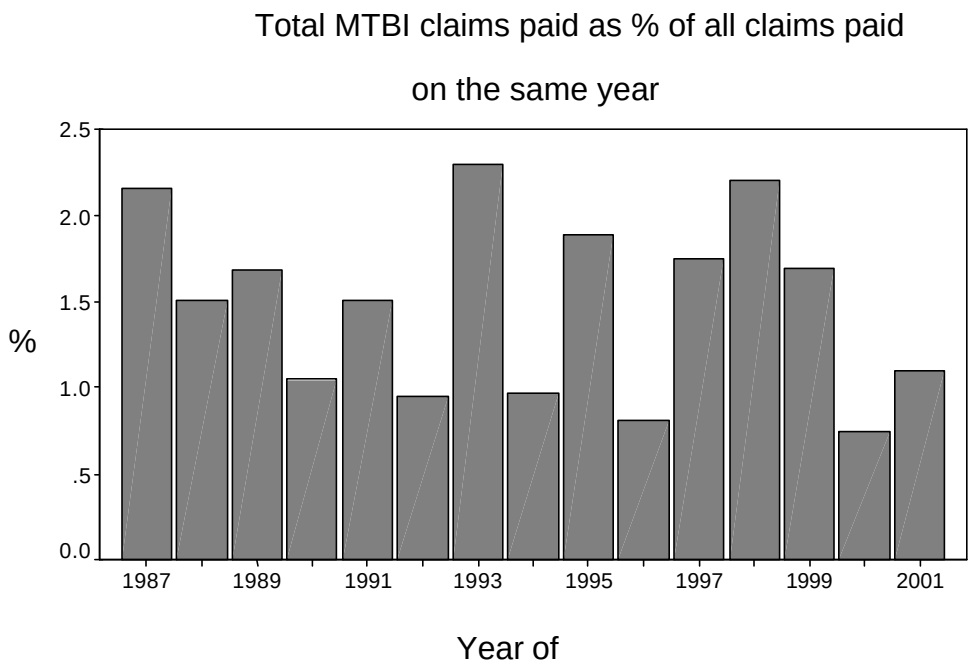
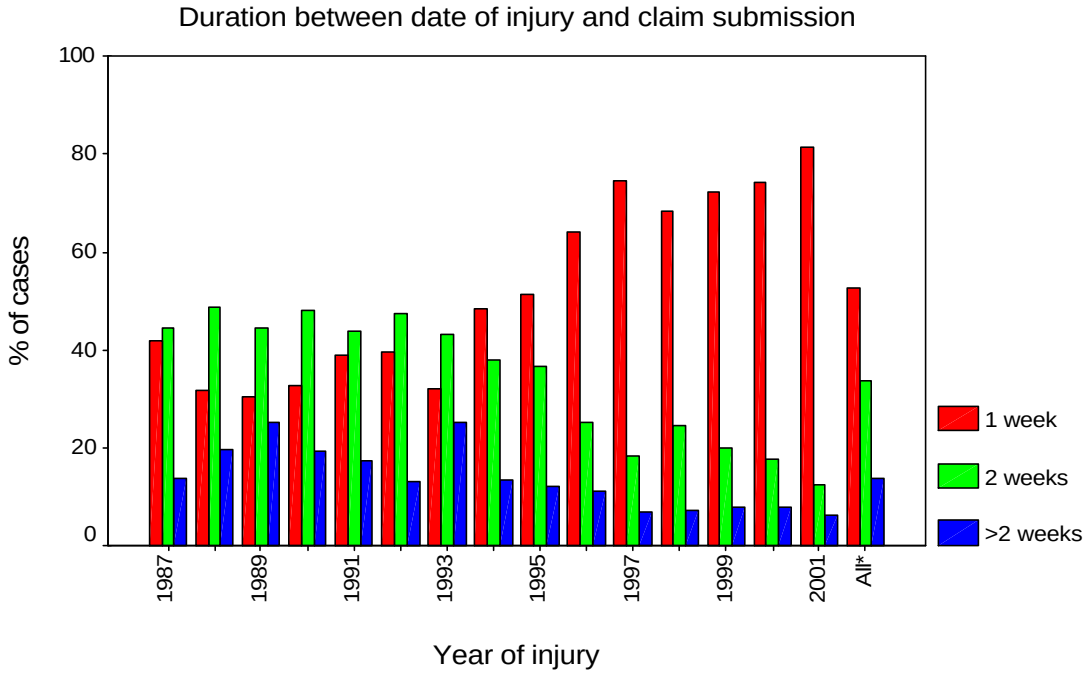


Figure 4 Total MTBI claims paid as % of all claims paid on the same year.



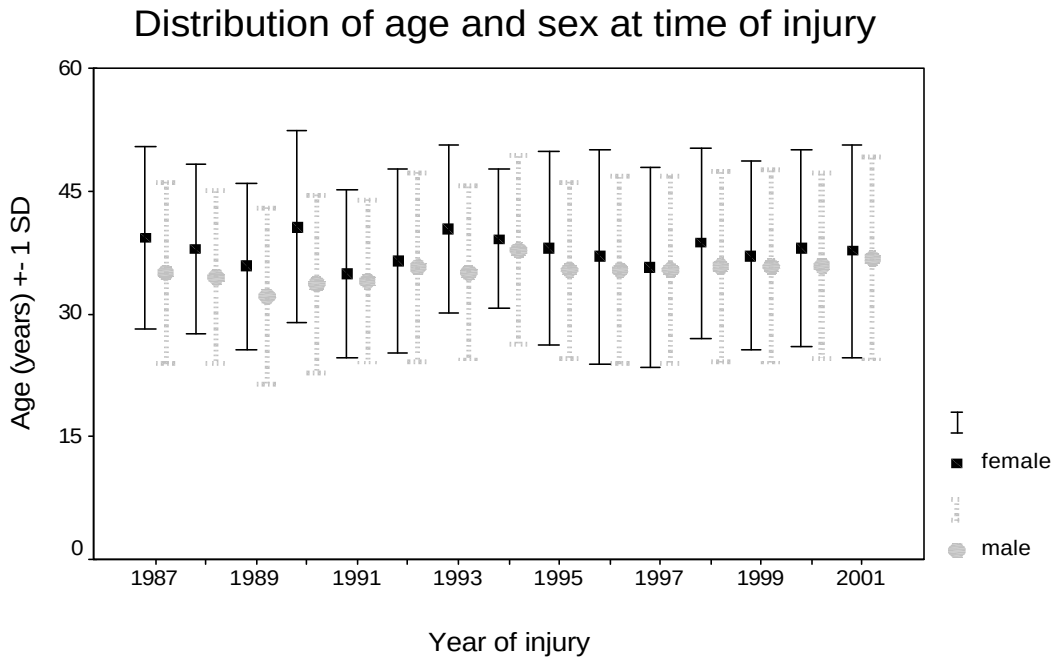
In the 15 years period, the average duration between injury and claim submission was 9.85 days (standard deviation 22.3 days) and median of 7 days. 52.6% of claimants submitted their claims by the first week, 33.7% by the second week and by the third week 95.5% of MTBI related claims had been submitted to the WCB of BC. There appears to be a significant trend across time that this duration is narrowing such that claims are being submitted 'quicker' (Figure 5).

Figure 5. Duration between date of injury and claim submission.



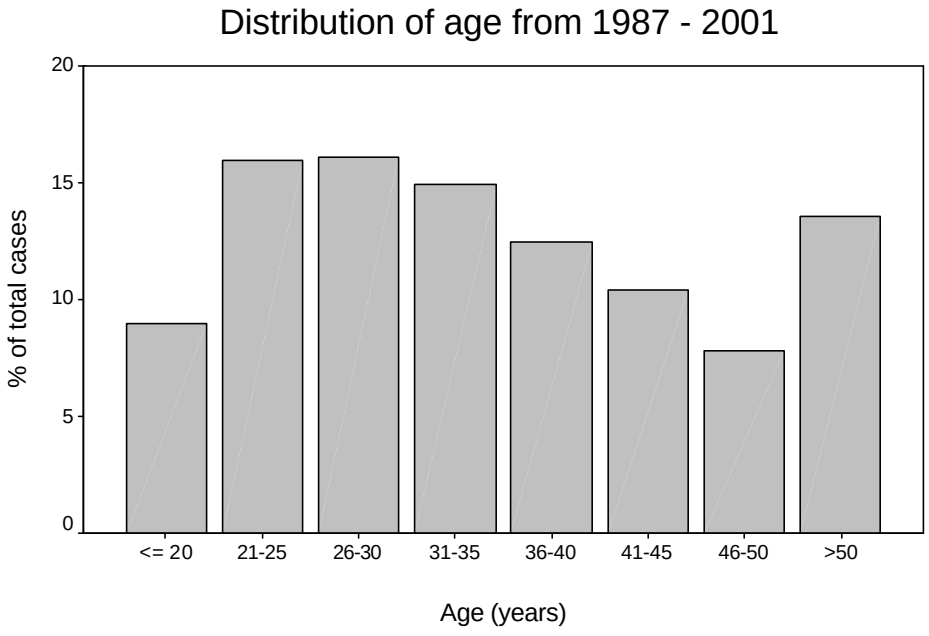
In the period 1987 - 2001, the average age of claimants was relatively stable at 30 years (range from 34 - 37 years old) (Figure 6).

Figure 6. Sex and age distribution at the time of injury.



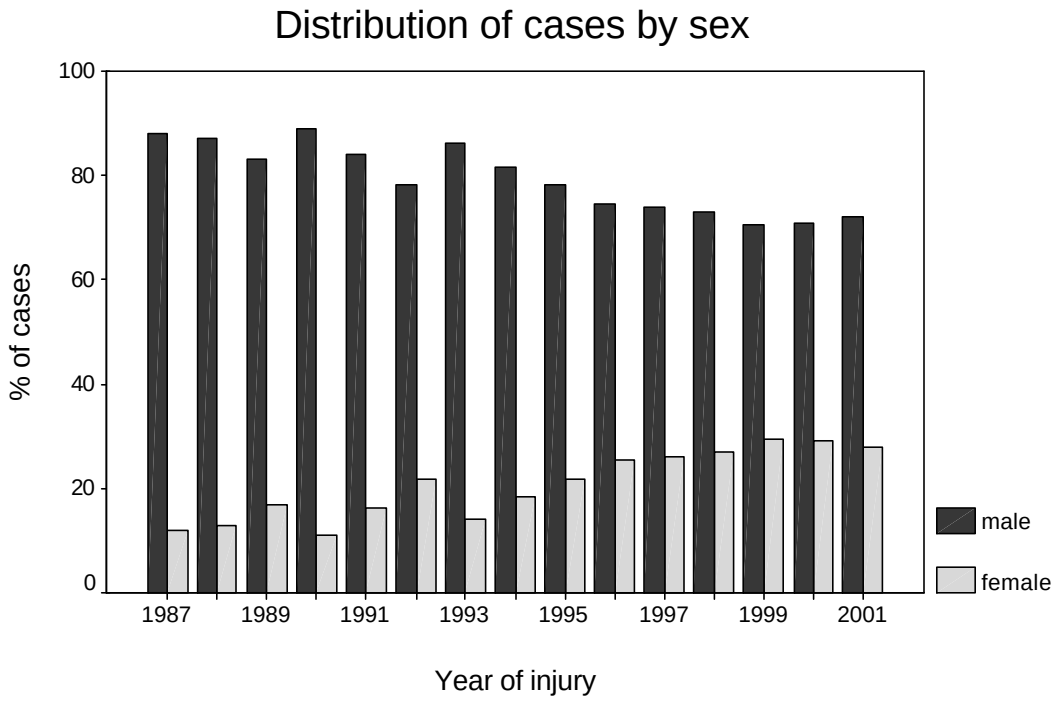
Almost half of the claimants were age 21 - 35 years old (Figure 7A). Interestingly, there were a large number of claimants older than 50 years. This is significant in that, as our data attests (and this is consistent with the world literature) 'older' MTBI claimants/patients have longer, more expensive disability (section IV.3 and section VI.2 on this document)

Figure 7. Distribution of age of MTBI claimants from 1987 - 2001.



In general, males were 3 - 4 times more likely to have an MTBI claim compared to females. There was a significant increase of female MTBI claimants across time (Figure 8). Female MTBI claimants also tended to be older than their male counterparts (Figure 6).

Figure 8. Distribution of MTBI claimants by sex



III.2 Costs and outcome of MTBI claims.

III.2.1. STD

The overall median days of MTBI claims on STD in the period 1987 - 2001 was 8 days with average of 63.7 days (standard deviation 204.9 days). The median days on STD among MTBI claimants did not vary much in the last 15 years (Figure 9). Interestingly, in the year 2001, there were 733 new cases of MTBI (largest number in anyone of the 15 year period) (Figure 1) and yet the year 2001 represented one of the lowest total number of STD days paid for MTBI claimants (Figure 10).

Figure 9. <u>Median</u> number of days MTBI claimants on STD by Year.

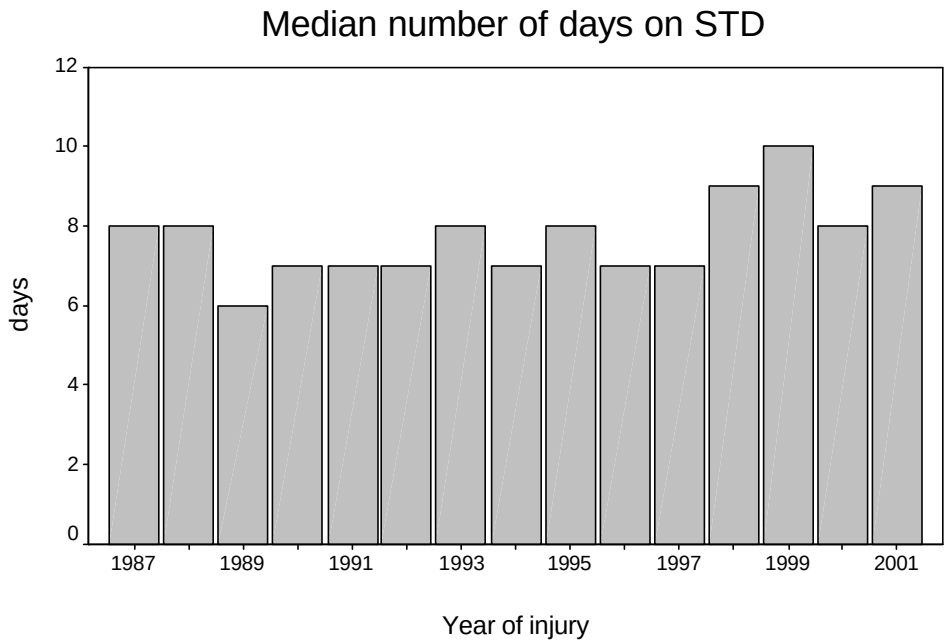
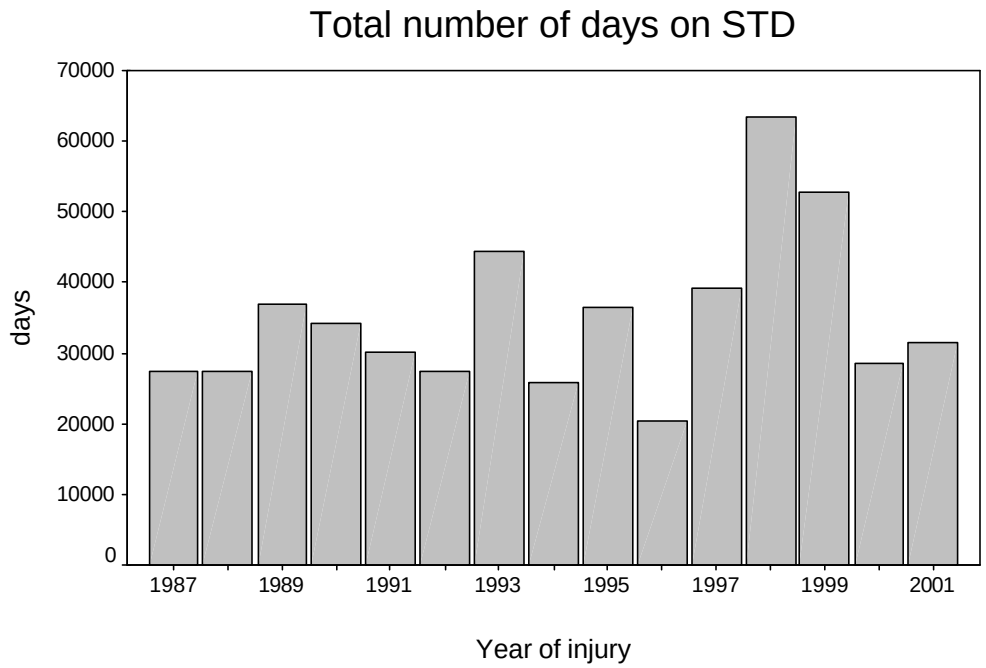
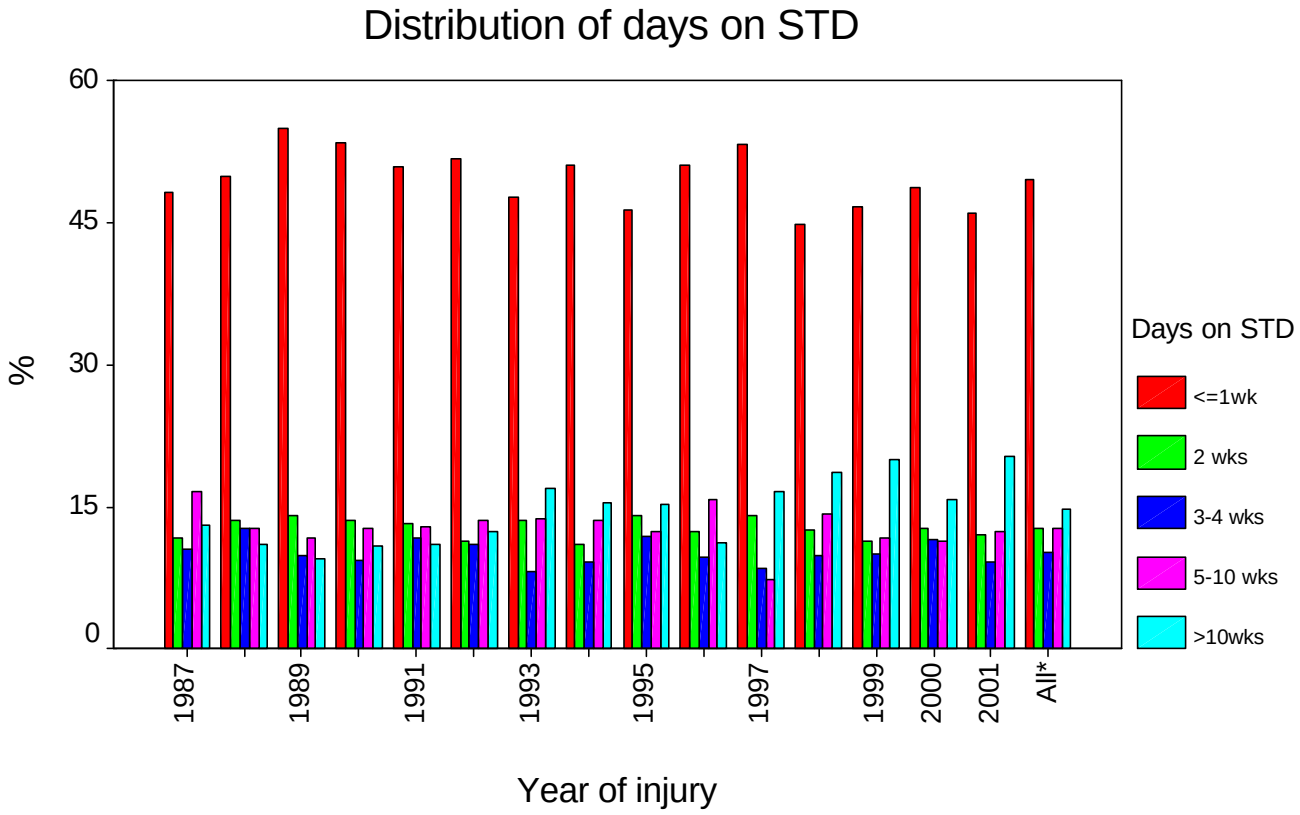


Figure 10. Total number of days MTBI claimants on STD by Year.



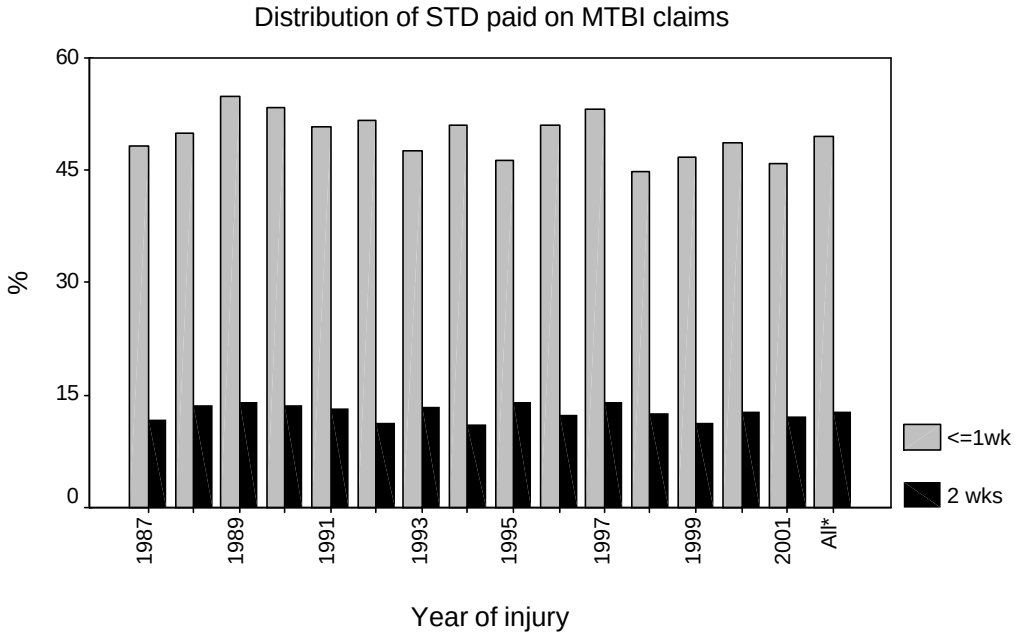
About half of the MTBI claims were on STD for ≤ 1 week, 12.8% for 2 weeks, 10.2% between 3-4 weeks, 12.8% between 5-10 weeks and 14.7% for > 10 weeks (Figure 11, 12, 13). In more detail, overall, 13.8% of the MTBI claims had STD for 1 day, 7.3% for 2 days, 10.2% for 3 days, 10.1% for 4 days, 4.6% for 7 days, 8.2% for 14 days and 8.7% for > 168 days (Figure 14).

Figure 11. Distribution of days MTBI claimants on STD by Year.



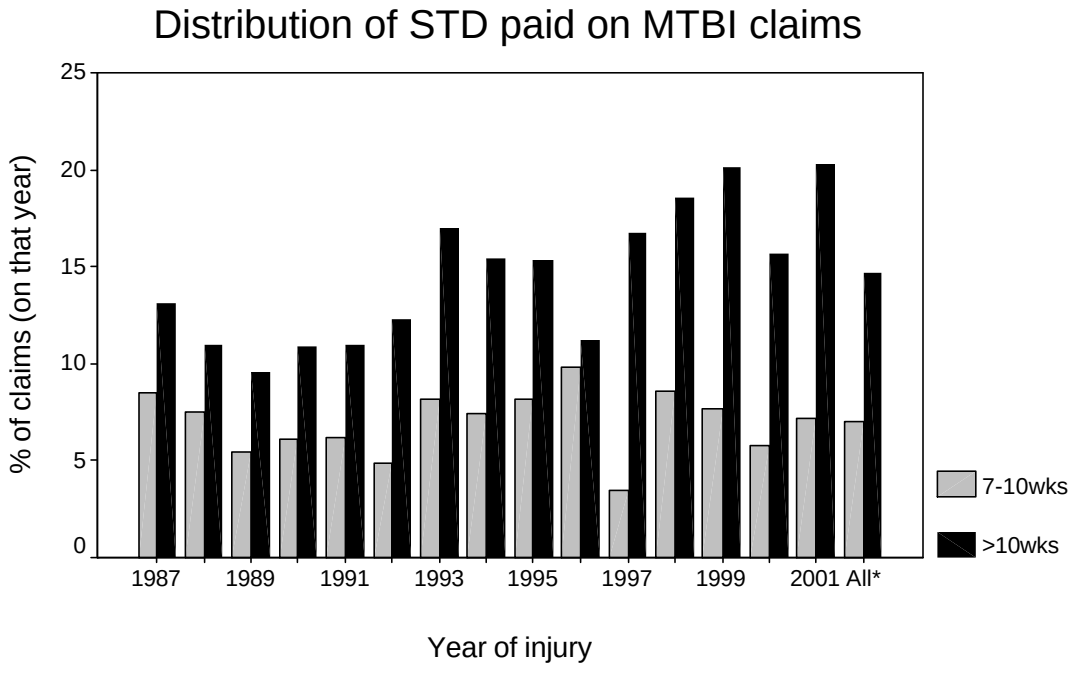
All* is from 1987-2001

Figure 12 represents a select portion of 'left hand tail' of Figure 11.



All* from 1987-2001

Figure 13 represents a select portion of 'right hand tail' of Figure 11.



All * is from 1987 - 2001

Figure 14. Distribution of days MTBI claimants on STD, year 1987 - 2001.

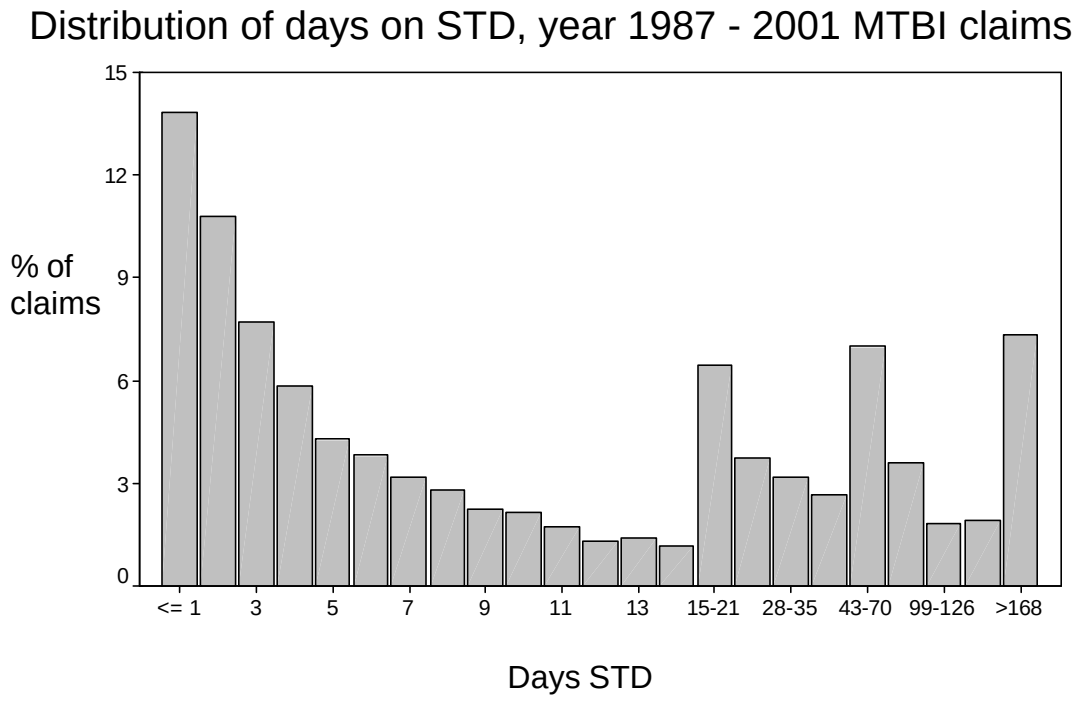


Figure 14 shows that there are 4 'peaks' on the STD graph across times i.e. at 1 day, 15-21 days, 43-70 days and > 168 days. This pattern is also observed when annual data were analyzed separately. Examples on this pattern are presented for 2001 (Figure 15), 2000 (Figure 16) and 1999 (Figure 17) claims.

Figure 15. Distribution of days on STD. Year 2001 claims only.

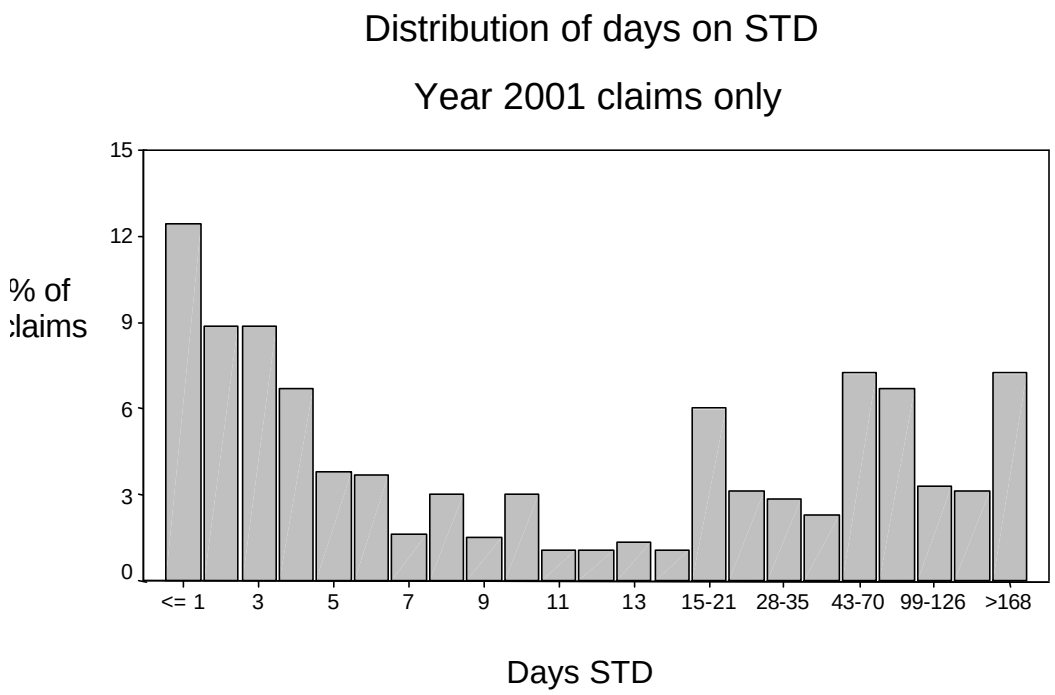


Figure 16. Distribution of days on STD. Year 2000 claims only.

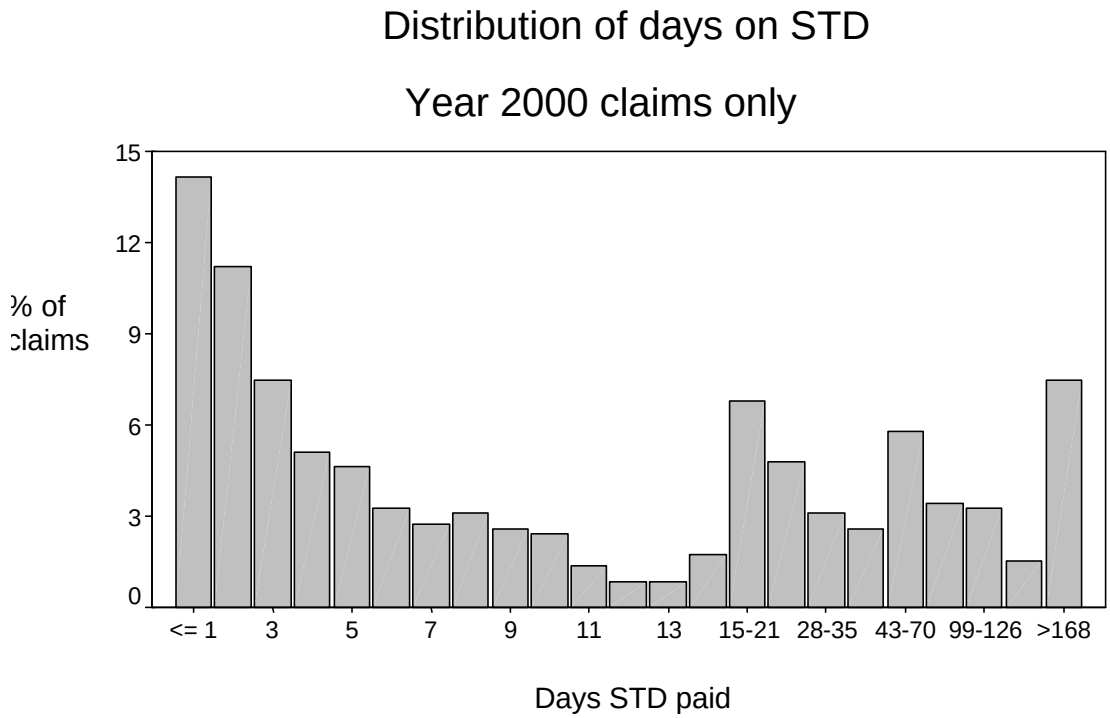
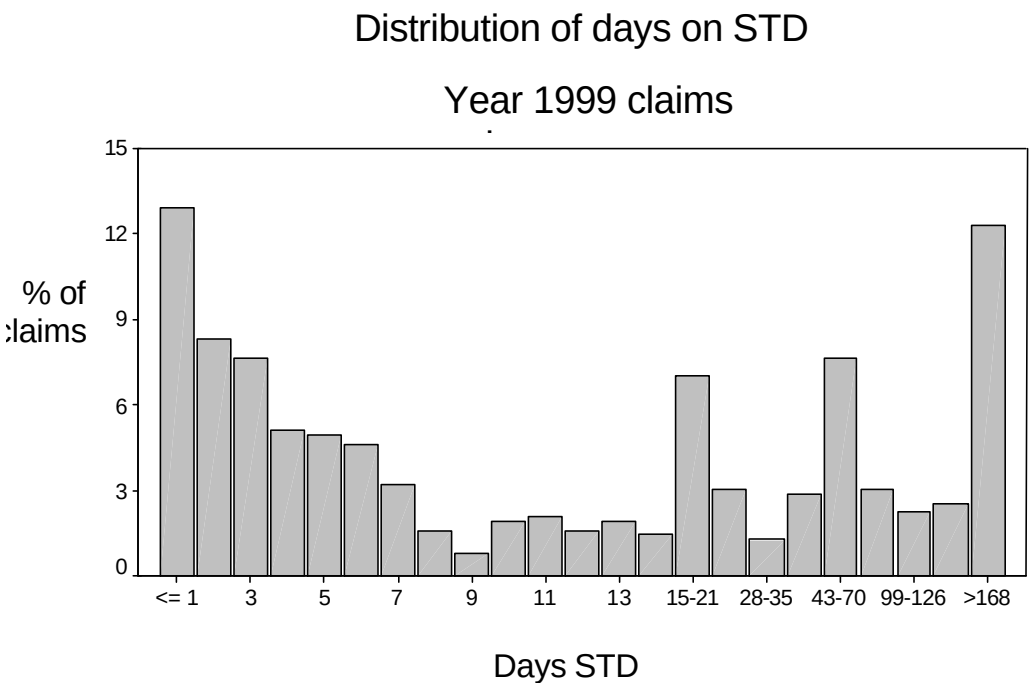


Figure 17. Distribution of days on STD. Year 1999 claims only.



Females had significantly less median days on STD compared to males (Figure 18). Figure 19 shows that there is somewhat a 'dose-response relationship' between age at injury and median days on STD. Overall, the older the claimants the longer the median STD. This pattern is also observed when data were analyzed separately for each year from 1987-2001. Longer median day is observed especially among claimants older than 40 year.

Figure 18. Median days on STD by Sex.

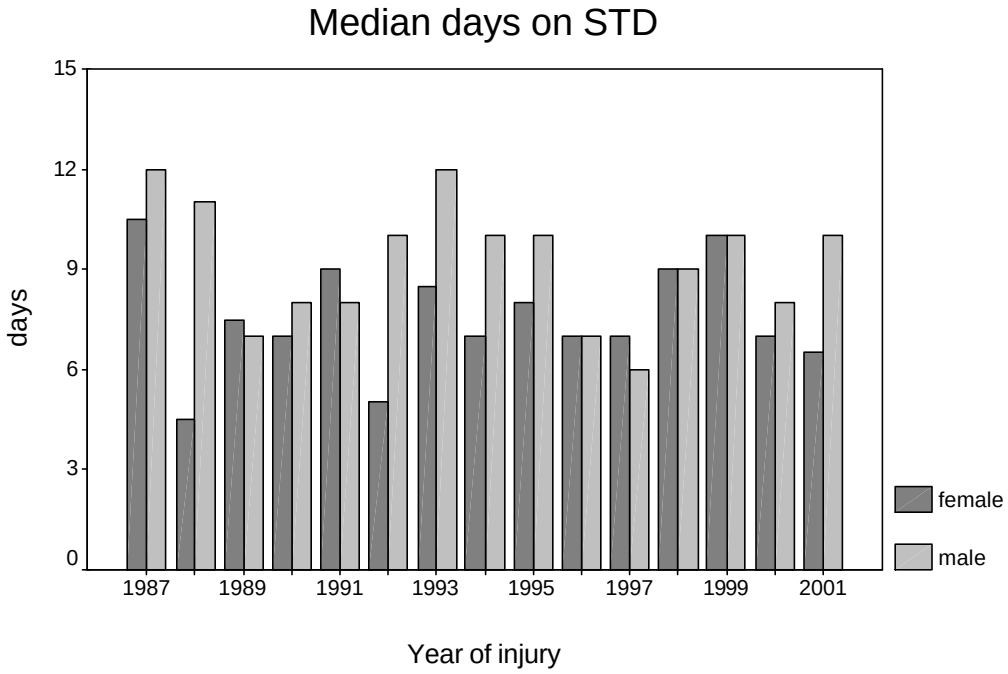
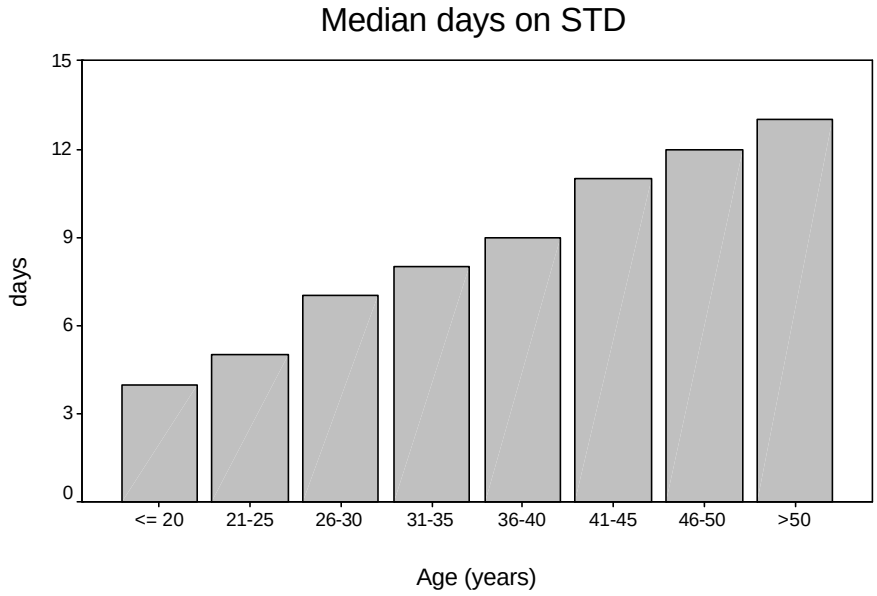


Figure 19. Median days on STD by Age group.



III.2.2. Claim costs

Even though there is a trend that the median claim costs for each MTBI claim is rising over time (Figure 20), the opposite tendency is observed for the annual total claim costs. The annual total claim costs for MTBI has been declining with the year 2000 and 2001 among the lowest in the last 15 years (Figure 21). This is probably due to a shorter duration of claimants under workers' compensation benefits.

Figure 20. Median amount of MTBI claim costs.

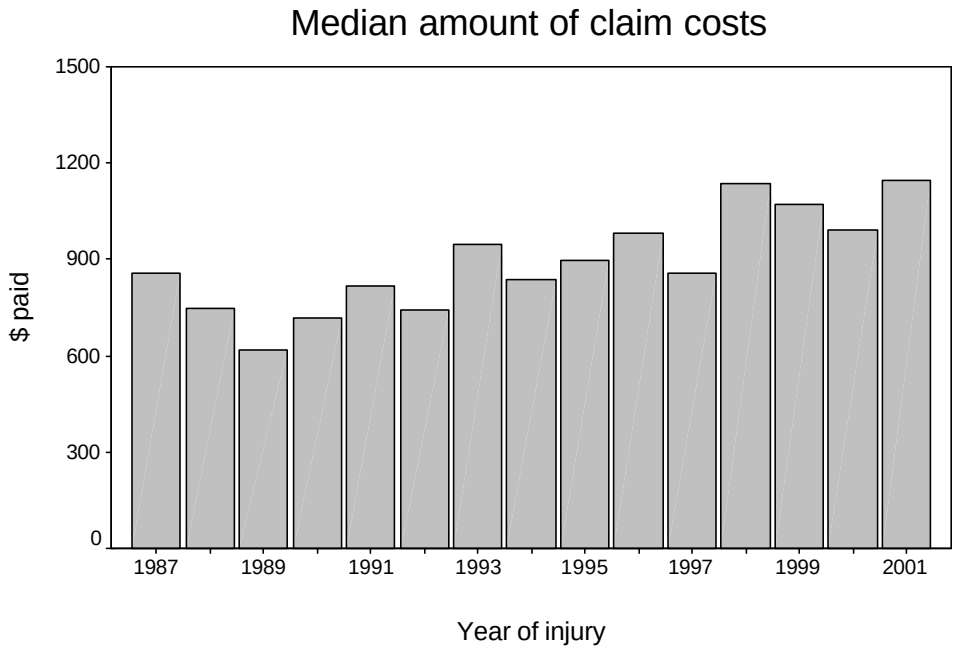
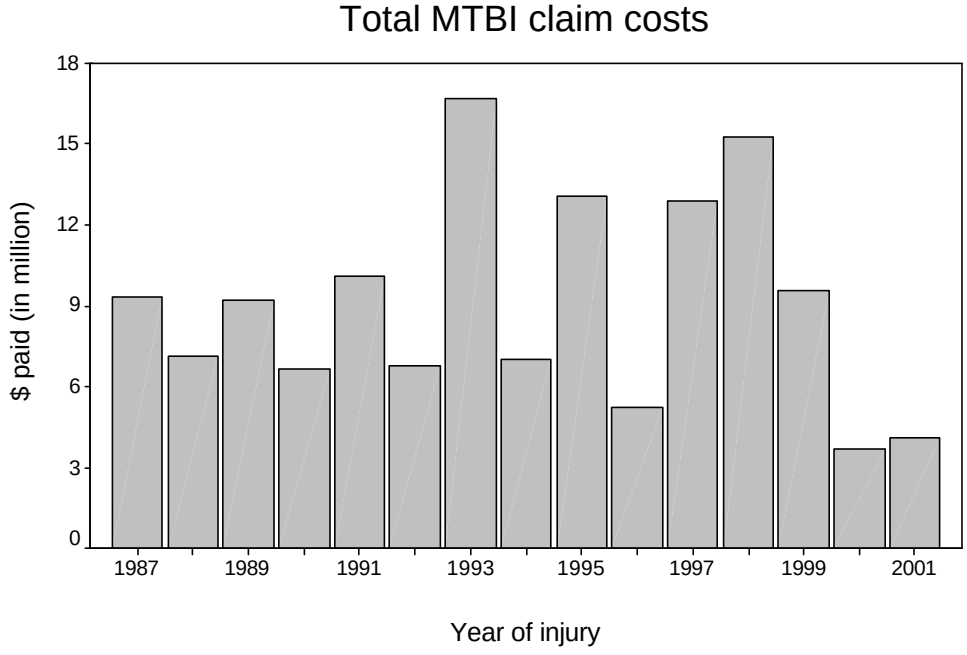


Figure 21. Total MTBI claim costs.



Median claim costs for female are about ½ - ? of their male counterparts (Figure 22). This is in direct contrast to that reported in the literature (section VI.2). However, due to the smaller number of female claims on MTBI, as is expected the total claim cost for females is significantly less than males (Figure 23).

Figure 22. <u>Median</u> amount of claim costs by Sex.

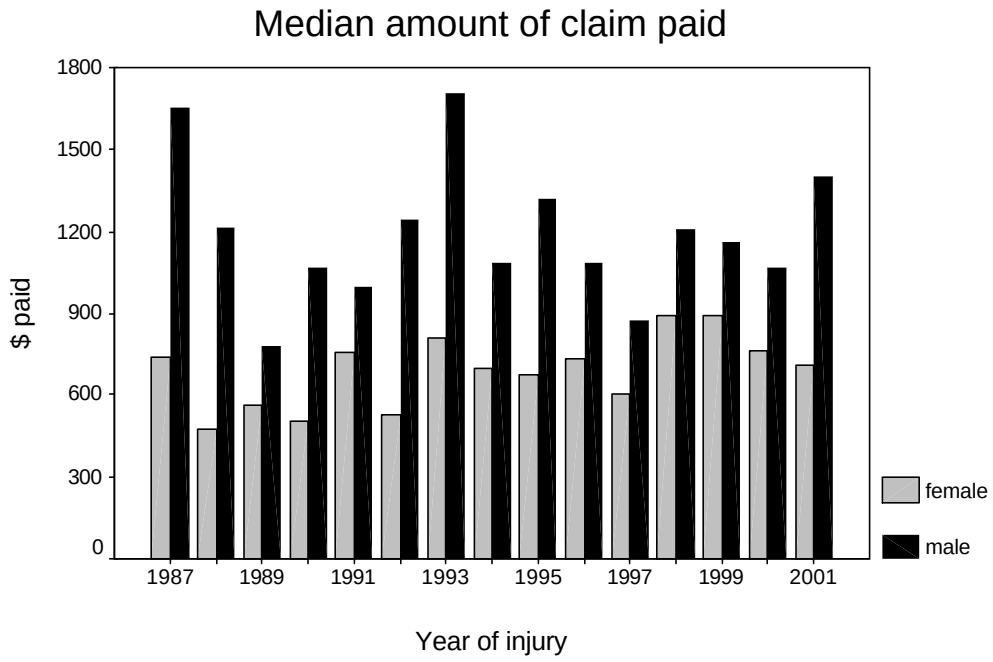
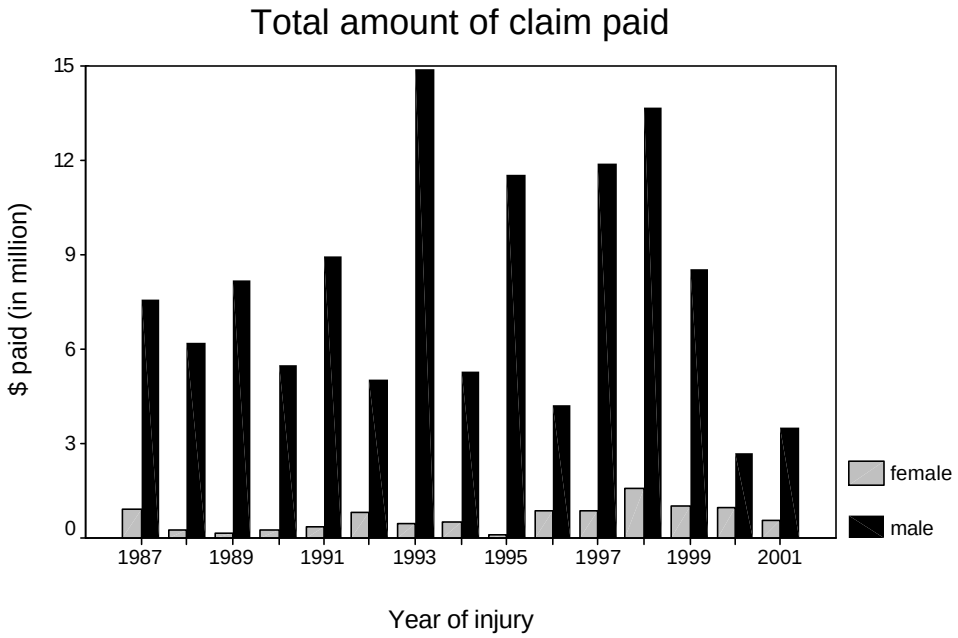


Figure 23. <u>Total</u> amount of claim costs by Sex.



Overall, the median cost of MTBI claim is highest among those older than 45 year (Figure 24). This is consistent with the world literature on this subject (section IV.3). There seems to be a cyclical pattern with regard to median claim costs among different age groups across time. There is a tendency towards an increase in the median claim costs among those aged 36 - 45 years (Figure 25).

Figure 24. Median amount of claim costs by Age group.

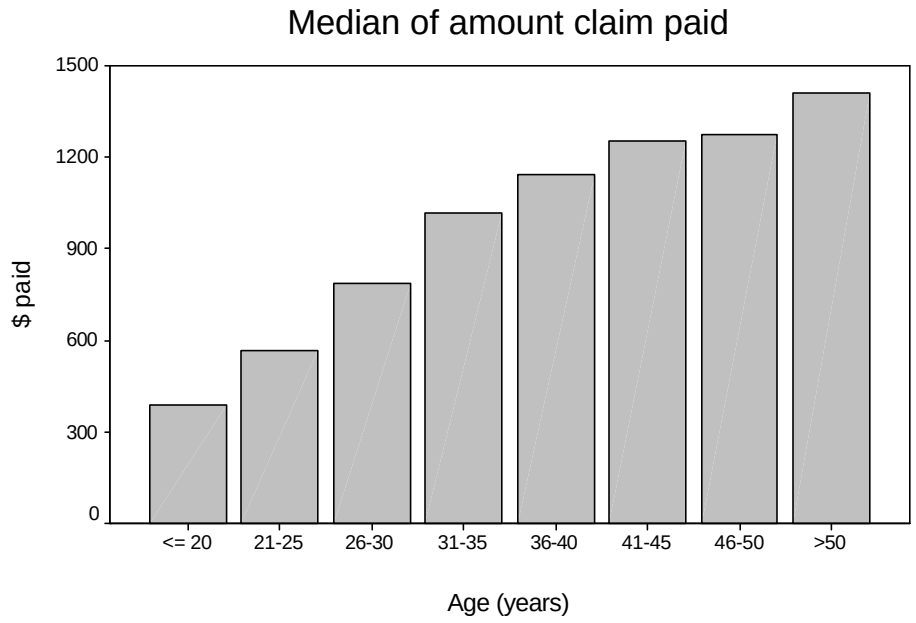
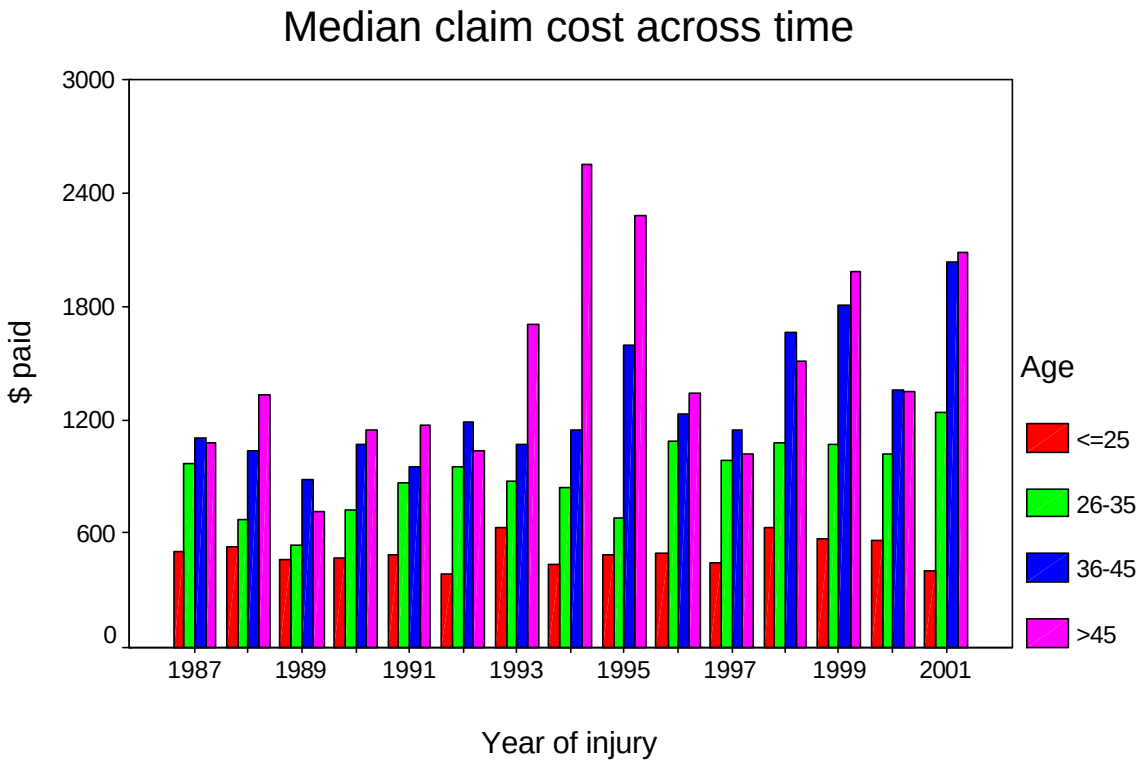
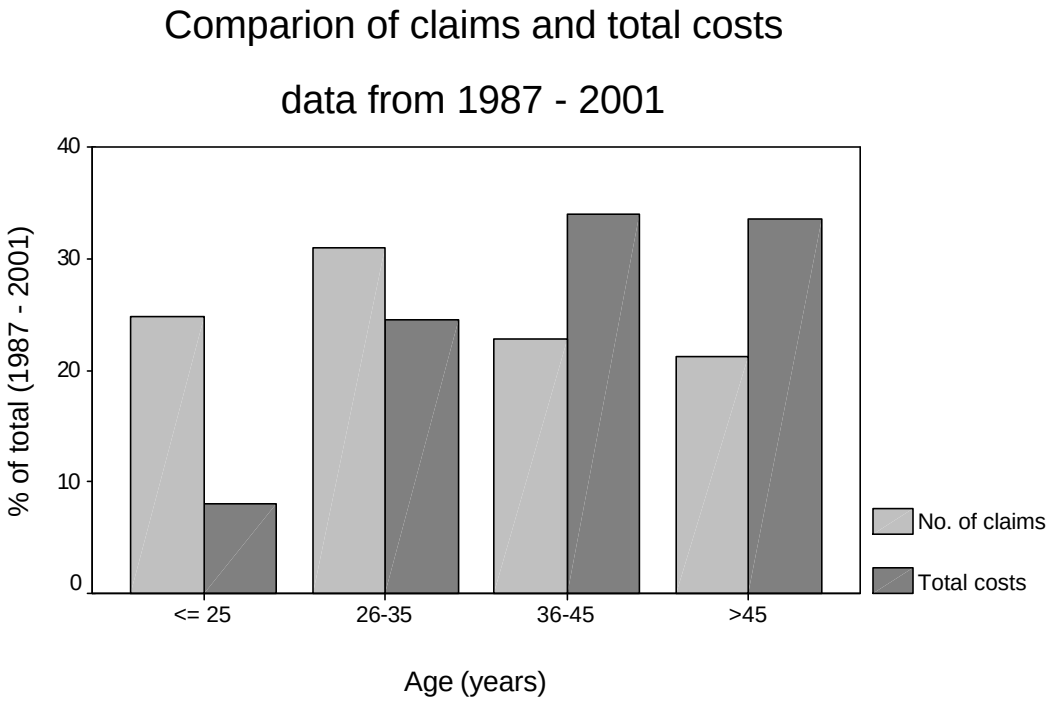


Figure 25. Median amount of claim costs across time, by Age group.



Even though, there are only 44.1% of MTBI claimants older than 35 years in 1987 - 2001, the total claim costs in this age group represent 67.4% of the overall MTBI claim costs in the same period (Figure 26).

Figure 26. Comparison of number of claims and total costs, 1987 - 2001.



III.2.3. Claim outcome

Claim outcome is defined as either the claimant being awarded health care only, short term disability (STD), long term disability (LTD) or if it is a fatal claim. In the period of 1987 - 2001, 0.5% of MTBI claims were for health care only benefit, 96.2% were for STD, 3.1% were for LTD and 0.1% were fatal (Figure 27 and 28). There is no relationship of duration between injury and claim submission with the outcome of the claims.

Figure 27. Percentage of Fatal, Health care only and LTD on MTBI claims, per year.

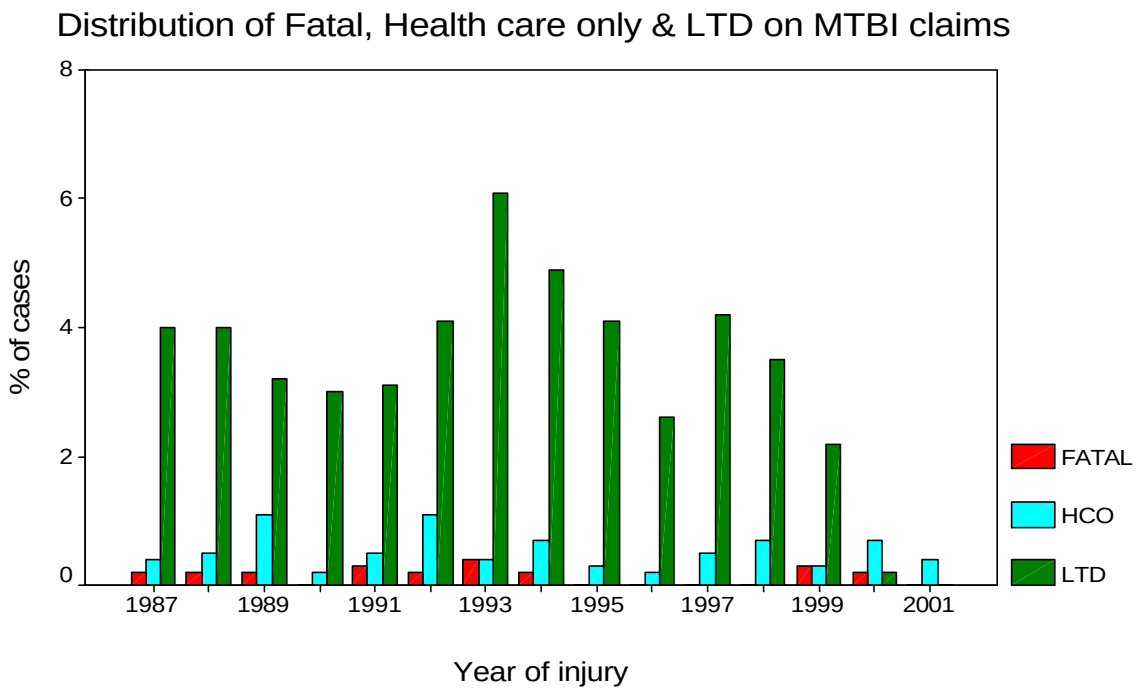
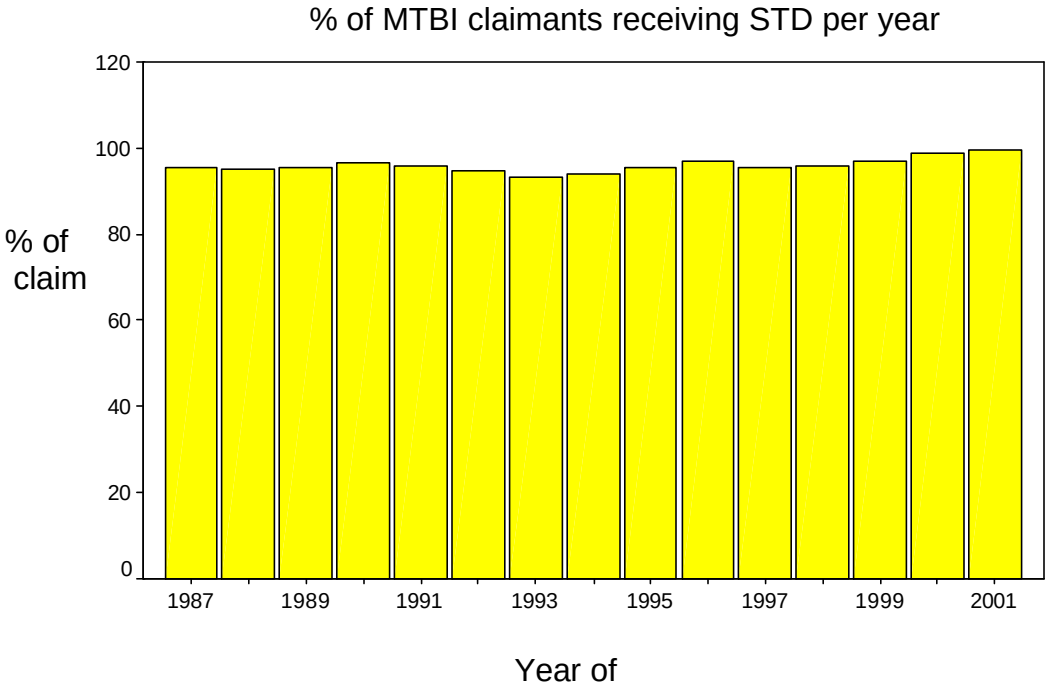


Figure 28. Percentage of MTBI claims receiving STD per year.



III.3. Data modelling.

In an attempt to find which factors affect the duration of STD, multiple linear regression analysis was undertaken. In this model, the duration of STD was the dependent factor with age, sex and duration between injury and claim submission as the independent (explanatory) factors.

Multiple linear regression analysis revealed that being female reduced the number of days on STD by about 42 days. On the other hand, for every year of increase in age, there was an increase in the number of days on STD by about 3 days. For every one day increase of duration between injury and claim submission, there was approximately a one day longer duration of claimants being on STD.

IV. Summary.

- On average, the WCB of BC receives about 551 new MTBI related claims annually. Even though MTBI claims comprise about 0.3% of the total annual claims, the claim costs associated with MTBI is about 1% - 2% of the total claim costs in that year.
- Half of the MTBI claimants submitted their claims within a week post injury. The median duration between injury and claim submission was 7 days.
- Almost half of the MTBI claims were aged 21 - 35 years. Males were 3-4 times more likely to file MTBI claim compared to females.
- Half of all MTBI claimants were on STD for 7 days. Only about 15% were on STD for > 10 weeks.
- Females spent less day on STD as compared to males. Older claimants(> 45 years) recorded longer median STD times.
- The median claim cost for MTBI has been rising across time, however, the annual total cost of MTBI has been declining. Costs of MTBI claims among females was less than males. The highest median cost of claims was observed among those aged 45 year or older.
- About 0.1% of MTBI claims were fatal; more than 95% were for STD only. 3.1% of MTBI claims are on LTD.

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Table 1. Diagnostic criteria for Mild Traumatic Brain Injury.

Signs/symptoms at Presentation	Organization/Expert									
	US-CDC	EFNS	SNC	ACRM	Colorado	EAST	ICD-9 CM	Alexander	Bernstein	Bazarian
Preceding event: blunt trauma or contact or acceleration/deceleration or rotation trauma of the head	Ö	Ö	Ö	Ö	Ö	Ö	Head injury	Ö	Ö	Ö
Glasgow Coma Scale at presentation/admission	13 - 15	13 - 15	14 - 15	13 - 15	13 - 15	13 - 15	-	13 - 15	13 - 15	15
Length of unconsciousness	≤ 30 min	≤ 30 min	0 - 5 min	< 30 min	≤ 30 min	≤ 20 min	0 - > 24hrs or unspecified	0- minutes	< 20 min	< 10 min
Post traumatic amnesia	Ö	< 60 min	0 - 5 min	< 24 hrs	≤ 24 hrs	brief	-	≤ 24 hrs	< 60 min	< 10 min
Focal neurological sign	Ö	Neg	Neg	Pos / neg	Pos / neg	Neg	-	Neg	Neg	Neg
Alteration in mental state at the time of accident	-	-	-	Pos	Pos	Pos	-	-	-	-
Neuroimaging intracranial lesion	-	-	-	-	Neg	Neg	-	Neg	Neg	Neg
Other cause for alteration of mental status	-	-	-	-	Neg	-	-	-	-	-
Skull fracture	-	-	-	-	-	-	Neg	-	Neg	-
Coding number	-	-	-	-	-	-	850.0 - .9			
Level of evidence	5	4	4	5	5	4	n/a	4	4	5
Reference number:	3	2	6	10	12	13	14	15	16	17

NB. All criteria requires the fulfillment of the preceding event and ≥ 1 sign or symptom. Neg = not present. Pos = present
US-CDC = United States, Center for Disease Control and Prevention, EFNS = European Federation of Neurological Societies. SNC = Scandinavian Neurotrauma Committee. ACRM = American Congress of Rehabilitation Medicine. Colorado = Workers' Compensation Board, State of Colorado, USA. EAST = EAST Practice Management Guidelines Work Group.
Alteration of mental status includes dazed, confusion, disorientation.

Table 2. Prevalence of signs and symptoms during follow-up.

	Van der Naalt et al[23] N = 67 (%)				Chambers et al[13] N = 940 (n, %)	
Symptoms	1 month	3 months	6 months	12 months	At discharge	3 months
No symptom	4	18	11	14	457 (41.2)	183 (33.8)
Headache	37	26	19	32	249 (22.5)	61 (11.3)
Dizziness	59	42	33	25	20 (1.8)	16 (2.9)
Balance disorders	29	16	13	14	-	-
Tinnitus	29	21	17	20	0	6 (1.1)
Hearing loss	18	10	14	12	-	-
Drowsiness	55	58	48	42	-	-
Fatigue	57	61	45	45	-	-
Memory problem	53	44	38	42	43 (3.9)	17 (3.1)
Poor concentration	51	44	44	42	-	-
Slowness	39	29	25	25	-	-
Irritability	35	27	26	34	-	-
Noise intolerance	53	40	25	28	-	-
Alcohol intolerance	6	11	17	20	-	-
Anxiety	20	19	19	26	-	-
Dry mouth	17	15	6	9	-	-
Neck pain	27	21	14	22	-	-
Neck stiffness	14	7	3	9	-	-
Arm pain	22	24	16	17	-	-
Itching	18	23	16	9	-	-
Weakness	-	-	-	-	22 (1.9)	7 (1.3)
Nausea	-	-	-	-	14 (1.3)	0
Numbness	-	-	-	-	0	12 (2.2)
Double vision	-	-	-	-	0	6 (1.1)
Headache and memory problems	-	-	-	-	38 (3.4)	13 (2.4)
Headache and dizziness	-	-	-	-	41 (3.7)	13 (2.4)
Headache and nausea	-	-	-	-	27 (2.4)	0
Headache and weakness	-	-	-	-	17 (1.5)	0
Headache and numbness	-	-	-	-	12 (1.1)	0
Headache and tinnitus	-	-	-	-	0	9 (1.7)
Numbness and tinnitus	-	-	-	-	0	7 (1.3)
Headache, memory problem and dizziness	-	-	-	-	0	6 (1.1)
Headache, dizziness and tinnitus	-	-	-	-	0	6 (1.1)

Table 3. Summary of neuroimaging modalities in MTBI.

Modality	Underlying principal	Advantages	Limitations	Possible/Role in MTBI	Strength of evidence
CT scan	x-rays acquired in multiple planes are reconstructed to indicate tissue densities	Rapid image acquisition, widely available, relatively low cost. Mainly detect haemorrhage and surgical lesions	Relatively low sensitivity. Doesn't detect changes in brain function. High x-ray exposure	Screening for structural and surgically correctable lesions, blood	Various systematic reviews and good randomized controlled trials (review in this section)
MRI	While in a magnetic field RF pulses excite protons in tissue and receiver coils detect relaxation of spins. In conventional MRI, T1, T2 and PD weighted planar images show normal tissue and pathology	Higher sensitivity compare to CT scan. High spatial resolution, widely available	Longer image acquisition (30-60 min). Doesn't detect changes in brain function	Higher sensitivity relative to CT Scan for detecting various lesions. T1 & T2 for acute hemorrhagic lesions. T2 for hemorrhagic diffuse axonal injury lesions or old hemorrhagic shear injuries	Various well designed comparison studies between CT and MRI (review in this section)
Volumetry	High spatial resolution of MRI allows volumetric quantification of various brain structure and regions. Complex structures require multiplanar visualization	Allows quantification of structural changes over time. Can be highly reproducible by experienced operators	Time consuming, not routinely available, requires experience rater and special software. Some structure boundaries are difficult to trace. Doesn't detect changes in brain function	Quantification of atrophy of various structures e.g. corpus callosum, hippocampus	No studies yet on MTBI alone. Several studies on mix severity showed atrophy of corpus callosum, hippocampus and increased ventricle to brain ratio
MRS	Chemical composition of selected voxels or planes is analyzed using NMR. Brain metabolites (NAA, Cho, Cr) are quantified in tissue by detection of characteristic peaks in spectra.	Measure of regional neuronal integrity and metabolic milieu. May detect areas of neuronal dysfunction in absence of detectable structural damage	Limited region assessed at a given time. Unclear relationship to clinical status. Usually doesn't detect changes in brain activity	Assessment of neuronal integrity. Detection of dysfunctional tissue which otherwise appear normal	Data on TBI of mized severity showed ratio of n-acetyl-aspartate over creatine differences

Table 3. (continue)

Modality	Underlying principal	Advantages	Limitations	Possible role in MTBI	Strength of evidence
DWI/DTI	DWI detects regional changes in membrane permeability to Na & K ions and intracellular water. Apparent diffusion coefficient maps reflect edema & ischemia. In DTI, tensor field is computed to map diffusional of water which is anisotropic in axonal white matter as compared to isotropic in other tissue	DWI: evidence of sensitivity of axonal & dendritic injury & edema after human & experimental TBI. DTI: unique in providing visualization of white matter pathways & a measure of their functional integrity.	DWI: interpretation is dependent on time since brain injury. Still little data on MTBI DTI: doesn't detect state related changes in brain activity. Complex post processing is required. Not available widely. Little data on MTBI	Assessment of white matter pathway integrity	Studies only available on 10 TBI mix severity cases
MTI	A pulse selectively suppresses signal from protein-bound water found in brain tissue vs. mobile water e.g. cerebrospinal fluid. Contrast is enhanced between water and fat containing tissue	Measure regional neuron integrity. May detect microscopic neural dysfunction in absence of visible structural changes. Sensitive to breakdown of blood brain barrier after gadolinium contrast agent	Doesn't detect change in brain function. Relationship between reduced magnetization transfer ratio and clinical status still unclear. Little data on MTBI	Characterization of dysfunctional neuronal tissue in both normal and abnormal appearing region on conventional MRI	Studies on 13 subjects with mix severity available so far
SPECT	Photon emitting radioisotopes distribute evenly in blood volume over several hours. Gamma camera is used to detect activity and map regional cerebral blood flow	Good indicator of regional cerebral blood flow. May be sensitive to TBI including MTBI (several studies available). Relatively widely available	No absolute quantification. Low spatial resolution. Requires registration to MRI. Doesn't permit imaging of transient cognitive events due to low resolution. Little information about white matter	Assessment of localized perfusion deficits especially in persistently symptomatic patients	Few studies on mixed severity suggested SPECT showed perfusion deficits, some correlation with cognitive deficits in the absence of structural abnormalities

Table 3. (continue)

Modality	Underlying principal	Advantages	Limitations	Possible role in MTBI	Strength of evidence
PET	Positron emitting radioisotopes can be used to measure resting metabolic rate (18-fluoro-deoxyglucose), task related changes in cerebral blood flow reflecting neural activity or neurotransmitter receptor density	Can be used to map glucose metabolism, cerebral blood flow and receptor populations. O^{15} method can be used to assess brain response to cognitive tasks	Quantitative studies need arterial line. Limited temporal and spatial resolution. Requires registration to MRI. Requires a cyclotron. Not available in most centres	Assessment of regional glucose utilization in persistently symptomatic MTBI patients	Four small case series suggesting areas of abnormal activity in symptomatic patients with normal CT Scan and MRI
fMRI	Blood oxygen level dependent contrast is based on the greater magnetic susceptibility of deoxy-haemoglobin than oxy-haemoglobin. This leads to increased T2 signal after neural activity because of increased local cerebral blood flow and surplus oxy-haemoglobin delivery	Non-invasive. Repeatable mapping of brain activation. Temporal resolution of several seconds or less allows imaging of transient cognitive events. Event related tasks paradigms can image activities during correct or incorrect responses. May be sensitive to MTBI	Susceptible to movement artifact. Unable to image neurotransmitters or receptor populations. Signal change in arbitrary units	Assessment of neurophysiological basis of cognitive complaints and deficits after MTBI	Small studies showed abnormalities of regional brain activation in MTBI on various memory tasks
MSI	Combines magneto encephalography (MEG) with MRI to examine structure and function. MEG and MRI are co-registered for analysis	Detects dendritic electrical activity with very high temporal resolution. Use this activity detection with MRI spatial imaging. Can use event-related paradigm	Technically very difficult. Limited mainly to cortical surface activity. Little data in MTBI	Assessment of abnormal regional dendritic electrical activity in persistently symptomatic patients	Only 1 study (30 cases) showed that MEG abnormalities were found in 65% of symptomatic MTBI.

Appendix 1. NICE's suggested written discharge advice card for people aged over 12 years and for carers of adults who have sustained a head injury.

1. for people aged over 12 years who have sustained a head injury.

We think that it is all right for you to leave hospital now. We have checked your symptoms and you seem well on the road to recovery. When you get home it is very unlikely that you will have any further problems. But, if any of the following symptoms do return, we suggest you come back, or get someone to bring you back to your nearest hospital A&E department as soon as possible:

- unconsciousness, or lack of full consciousness (for example, problems keeping eyes open)
- any confusion (not knowing where you are, getting things muddled up)
- any drowsiness (feeling sleepy) that goes on for longer than 1 hour when you would normally be wide awake
- any problems understanding or speaking
- any loss of balance or problems walking
- any weakness in one or more arms or legs
- any problems with your eyesight
- very painful headache that won't go away
- any vomiting – getting sick
- any fits (collapsing or passing out suddenly)
- clear fluid coming out of your ear or nose
- bleeding from one or both ears
- new deafness in one or both ears

Things you shouldn't worry about:

You may feel some other symptoms over the next few days which should disappear in the next 2 weeks. These include a mild headache, feeling sick (without vomiting), dizziness, irritability or bad temper, problems concentrating or problems with your memory tiredness, lack of appetite or problems sleeping. If you feel very concerned about any of these symptoms in the first few days after discharge, you should go and see your own doctor to talk about them.

If these problems do not go away after 2 weeks, you should go and see your doctor. We would also recommend that you seek a doctor's opinion about your ability to drive a car or motorbike.

Things that will help you get better:

If you follow this advice you should get better more quickly and it may help any symptoms you have to go away:

- DO NOT stay at home alone for the first 48 hours after leaving hospital.
- DO make sure you stay within easy reach of a telephone and medical help.

- DO have plenty of rest and avoid stressful situations.
- DO NOT take any alcohol or drugs.
- DO NOT take sleeping pills, sedatives or tranquilisers unless they are given by a doctor.
- DO NOT play any contact sport (for example, rugby or football) for at least 3 weeks without talking to your doctor first.
- DO NOT return to your normal school, college or work activity until you feel you have completely recovered.
- DO NOT drive a car, motorbike or bicycle or operate machinery unless you feel you have completely recovered.

Telephone number to call at the hospital: _____

Long-term problems:

Most patients recover quickly from their accident and experience no long-term problems. However, some patients only develop problems after a few weeks or months. If you start to feel that things are not quite right (for example, memory problems, not feeling yourself), then please contact your doctor as soon as possible so that we can check to make sure you are recovering properly.

2. for <u>care givers</u> of adults who have sustained a head injury.

We think that it is all right for your friend/relative/client to leave hospital now. We have checked their symptoms and they seem well on the road to recovery. When you get them home it is very unlikely that they will have any further problems. But, if any of the following symptoms do return, we suggest you bring them back to their nearest hospital A&E department as soon as possible:

- unconsciousness, or lack of full consciousness (for example, problems keeping eyes open)
- any confusion (not knowing where they are, getting things muddled u)
- any drowsiness (feeling sleepy) that goes on for longer than 1 hour when they would normally be wide awake
- difficulty waking the patient up
- any problems understanding or speaking
- any loss of balance or problems walking
- any weakness in one or more arms or legs
- any problems with their eyesight
- very painful headache that won't go away
- any vomiting – getting sick
- any fits (collapsing or passing out suddenly)
- clear fluid coming out of their ear or nose
- bleeding from one or both ears
- new deafness in one or both ears

<u>Things you shouldn't worry about:</u>

They may feel some other symptoms over the next few days which should disappear I the next 2 weeks. These include a mild headache, feeling sick (without vomiting), dizziness, irritability or bad temper, problems concentrating or problems with their memory, tiredness, lack of appetite or problems sleeping. If you feel very concerned about any of these symptoms in the first few days after discharge, you should bring the patent to their doctor to talk about them.

If these problems do not go away after 2 weeks, you should bring the patient to see their doctor. We would also recommend that they seek a doctor's opinion about their inability to drive a car or motorbike.

<u>Things that will help the patient get better</u>:

If the patient follows this advice it should help them get better more quickly and it may help any symptoms they have to go away:

- DO have plenty of rest and avoid stressful situations.
- DO NOT take any alcohol or drugs.
- DO NOT take sleeping pills, sedatives or tranquilisers unless they are given by a doctor.

- DO NOT return to their normal college or work activity until they feel they have completely recovered.
- DO NOT play any contact sport (for example, football) for at least 3 weeks without talking to their doctor first.
- DO NOT drive a car, motorbike or bicycle or operate machinery unless they feel they have completely recovered.

Things you should do to make sure the patient is OK:

- DO NOT leave the patient alone in the home for the first 48 hours after leaving hospital.
- DO make sure that there is a nearby telephone and that the patient stays within easy reach of medical help.

Telephone number to call at the hospital: _____

Long-term problems:

Most patients recover quickly from their accident and experience no long-term problems. However, some patients only develop problems after a few weeks or months.

If you start to feel that things are not quite right for your friend/relative/client (for example, memory problems, not feeling themselves), then please contact your doctor as soon as possible so that we can check to make sure they are recovering properly.

Appendix 2. Head Up: preventing brain injury.

Please see separate PDF file entitled 'CDC-preventing head injury'.

Heads Up
Preventing Brain Injuries

Brain injuries are caused by a bump or blow to the head. These injuries sometimes are called "concussions" or "traumatic brain injuries" (TBIs) and can range from mild to severe.

Most mild brain injuries cause no harm. But sometimes even mild brain injuries can cause serious, long-lasting problems. The best way to protect yourself and your family from brain injuries is to prevent them from happening in the first place.

How to Prevent a Brain Injury

Here are some tips from the Centers for Disease Control and Prevention (CDC) and the Brain Injury Association of America to reduce the chances that you or your family members will have a brain injury.

- Wear a seat belt every time you drive or ride in a motor vehicle.
- Always buckle your child into a child safety seat, booster seat, or seat belt (according to the child's height, weight, and age) in the car.
- Never drive while under the influence of alcohol or drugs.
- Wear a helmet and make sure your children wear helmets when:
  - Riding a bike, motorcycle, snowmobile, or all-terrain vehicle;
  - Playing a contact sport, such as football, ice hockey, or boxing;
  - Using in-line skates or riding a skateboard;
  - Batting and running bases in baseball or softball;
  - Riding a horse; or
  - Skiing or snowboarding.
- Avoid falls in the home by:
  - Using a step stool with a grab bar to reach objects on high shelves;
  - Installing handrails on stairways;
  - Installing window guards to keep young children from falling out of open windows;
  - Using safety gates at the top and bottom of stairs when young children are around;
  - Removing tripping hazards such as small area rugs and loose electrical cords;
  - Using non-slip mats in the bathtub and on shower floors;
  - Putting grab bars next to the toilet and in the tub or shower;
  - Maintaining a regular exercise program to improve strength, balance, and coordination; and
  - Seeing an eye doctor regularly for a vision check to help lower the risk of falling.
- Make sure the surface on your child's playground is made of shock-absorbing material, such as hardwood, mulch, and sand.
- Keep firearms stored unloaded in a locked cabinet or safe. Store bullets in a separate secured location.

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Over please >>

When to Call the Doctor: Signs and Symptoms of Brain Injury

Heads Up
Preventing Brain Injuries

Here is a list of common symptoms of a brain injury (concussion). If you or a family member has a head injury and you notice any of the symptoms on the list, call your doctor right away. Describe the injury and symptoms, and ask if you should make an appointment to see your own doctor or another specialist.

Symptoms of a Concussion

In Adults

- Headaches or neck pain that won't go away
- Trouble with such mental tasks as remembering, concentrating, or decision-making
- Slow thinking, speaking, acting, or reading
- Getting lost or easily confused
- Feeling tired all the time, having no energy or motivation
- Mood changes (feeling sad or angry for no reason)
- Changes in sleep patterns (sleeping a lot more or having a hard time sleeping)
- Feeling light-headed or dizzy, or losing your balance
- An urge to vomit (nausea)
- Increased sensitivity to lights, sounds, or distractions
- Blurred vision or eyes that tire easily
- Loss of sense of smell or taste
- Ringing in the ears

In Children

- Feeling tired or listless
- Being irritable or cranky (will not stop crying or cannot be consoled)
- Changes in eating (will not eat or nurse)
- Changes in sleep patterns
- Changes in the way the child plays
- Changes in performance at school
- Lack of interest in favorite toys or activities
- Loss of new skills, such as toilet training
- Loss of balance, unsteady walking
- Vomiting

When you visit the doctor, here are some important questions to ask:

- How long should I expect these symptoms to last?
- What should I do for this condition?
- Is it safe to get back to my normal daily routine, such as school, work, or playing sports and doing other physical activities?
- What can I do to keep from injuring myself again?

For more information…

The Brain Injury Association of America (BIAA)
BIAA has information on brain injury statistics and prevention as well as services for persons who have a brain injury, and how to contact the Brain Injury Association in your state. You can call BIAA toll-free at 1-800-444-6443, or visit BIAA on the Internet at www.biausa.org.

Centers for Disease Control and Prevention (CDC)
CDC has a wide variety of information about prevention of mild traumatic brain injury and other types of injuries. Find CDC's injury prevention resources at www.cdc.gov/ncipc/ncipchm.htm.

National Bicycle Safety Network (NBSN)
The NBSN web site at www.cdc.gov/ncipc/bike/ has information about preventing brain injuries through bicycle helmet use.

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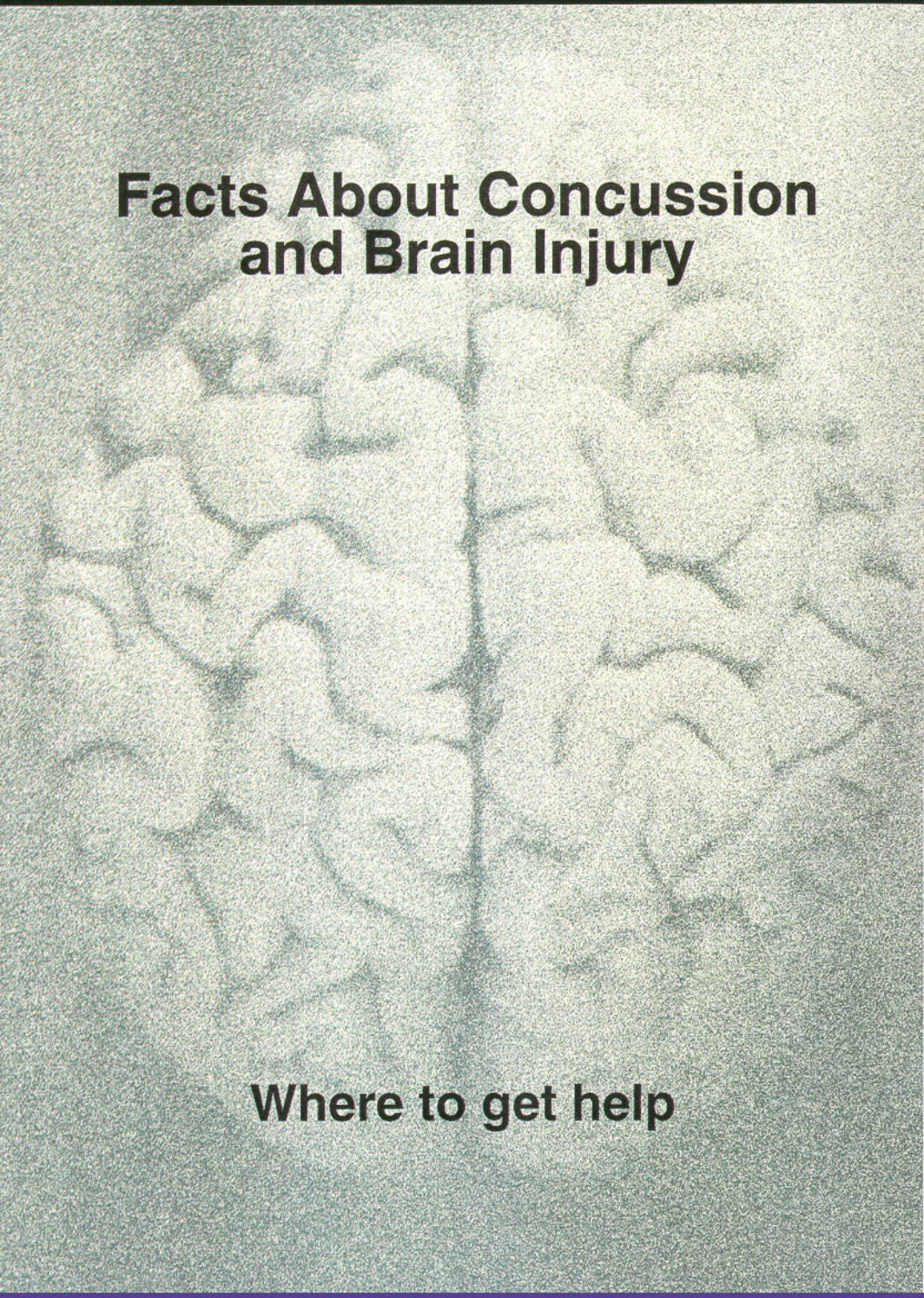
Appendix 3. Facts about concussion and brain injury.

Please see separate PDF file entitled 'CDC-Facts about concussion'.

Facts About Concussion and Brain Injury



Where to get help

Keep this...

Brain Injury Association National Help Line: 1-800-444-6443
Brain Injury Association Web site: www.biausa.org
Centers for Disease Control and Prevention Web site:
www.cdc.gov/ncipc/tbi

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About Brain Injury

A blow or jolt to the head can disrupt the normal function of the brain. Doctors often call this type of brain injury a “concussion” or a “closed head injury.” Doctors may describe these injuries as “mild” because concussions are usually not life threatening. Even so, the effects of a concussion can be serious.

After a concussion, some people lose consciousness or are “knocked out” for a short time, but not always — you can have a brain injury without losing consciousness. Some people are simply dazed or confused. Sometimes whiplash can cause a concussion.

Because the brain is very complex, every brain injury is different. Some symptoms may appear right away, while others may not show up for days or weeks after the concussion. Sometimes the injury makes it hard for people to recognize or to admit that they are having problems.

The signs of concussion can be subtle. Early on, problems may be missed by patients, family members, and doctors. People may look fine even though they’re acting or feeling differently.

Because all brain injuries are different, so is recovery. Most people with mild injuries recover fully, but it can take time. Some symptoms can last for days, weeks, or longer.

In general, recovery is slower in older persons. Also, persons who have had a concussion in the past may find that it takes longer to recover from their current injury.

This brochure explains what can happen after a concussion, how to get better, and where to go for more information and help when needed.

Medical Help

People with a concussion need to be seen by a doctor. Most people with concussions are treated in an emergency department or a doctor's office. Some people must stay in the hospital overnight for further treatment.

Sometimes the doctors may do a CT scan of the brain or do other tests to help diagnose your injuries. Even if the brain injury doesn't show up on these tests, you may still have a concussion.

Your doctor will send you home with important instructions to follow. For example, your doctor may ask someone to wake you up every few hours during the first night and day after your injury.

Be sure to carefully follow all your doctor's instructions. If you are already taking any medicines — prescription, over-the-counter, or "natural remedies" — or if you are drinking alcohol or taking illicit drugs, tell your doctor. Also, talk with your doctor if you are taking "blood thinners" (anticoagulant drugs) or aspirin, because these drugs may increase your chances of complications. If it's all right with your doctor, you may take acetaminophen (for example, Tylenol®* or Panadol®*) for headache or neck pain.

*Use of trade names is for identification only and does not imply endorsement by the U.S. Department of Health and Human Services.

Danger Signs — Adults

In rare cases, along with a concussion, a dangerous blood clot may form on the brain and crowd the brain against the skull. Contact your doctor or emergency department right away if, after a blow or jolt to the head, you have any of these danger signs:

- Headaches that get worse
- Weakness, numbness, or decreased coordination
- Repeated vomiting

The people checking on you should take you to an emergency department right away if you:

- Cannot be awakened
- Have one pupil — the black part in the middle of the eye — larger than the other
- Have convulsions or seizures
- Have slurred speech
- Are getting more and more confused, restless, or agitated

Danger Signs — Children

Take your child to the emergency department right away if the child has received a blow or jolt to the head and:

- Has any of the danger signs for adults listed on page 4
- Won't stop crying
- Can't be consoled
- Won't nurse or eat

Although you should contact your child's doctor if your child vomits more than once or twice, vomiting is more common in younger children and is less likely to be an urgent sign of danger than it is in an adult.

Symptoms of Brain Injury

Persons of All Ages

"I just don't feel like myself."

The type of brain injury called a concussion has many symptoms. These symptoms are usually temporary, but may last for days, weeks, or even longer. Generally, if you feel that "something is not quite right," or if you're "feeling foggy," you should talk with your doctor.

Here are some of the symptoms of a concussion:

- Low-grade headaches that won't go away
- Having more trouble than usual:
  - Remembering things
  - Paying attention or concentrating
  - Organizing daily tasks
  - Making decisions and solving problems
- Slowness in thinking, acting, speaking, or reading
- Getting lost or easily confused
- Neck pain

- Feeling tired all the time, lack of energy
- Change in sleeping pattern:
  - Sleeping for much longer periods of time than before
  - Trouble sleeping or insomnia
- Loss of balance, feeling light-headed or dizzy
- Increased sensitivity to:
  - Sounds
  - Lights
  - Distractions
- Blurred vision or eyes that tire easily
- Loss of sense of taste or smell
- Ringing in the ears
- Change in sexual drive
- Mood changes:
  - Feeling sad, anxious, or listless
  - Becoming easily irritated or angry for little or no reason
  - Lack of motivation

Young Children

Although children can have the same symptoms of brain injury as adults, it is harder for young children to let others know how they are feeling. Call your child's doctor if your child seems to be getting worse or if you notice any of the following:

- Listlessness, tiring easily
- Irritability, crankiness
- Change in eating or sleeping patterns
- Change in the way they play
- Change in the way they perform or act at school
- Lack of interest in favorite toys
- Loss of new skills, such as toilet training
- Loss of balance, unsteady walking

Older Adults

Older adults with a brain injury may have a higher risk of serious complications such as a blood clot on the brain. Headaches that get worse or an increase in confusion are signs of this complication. If these signs occur, see a doctor right away.

Getting Better

"Sometimes the best thing you can do is just rest and then try again later."

How fast people recover from brain injury varies from person to person. Although most people have a good recovery, how quickly they improve depends on many factors. These factors include how severe their concussion was, what part of the brain was injured, their age, and how healthy they were before the concussion.

Rest is very important after a concussion because it helps the brain to heal. You'll need to be patient because healing takes time. Return to your daily activities, such as work or school, at your own pace. As the days go by, you can expect to gradually feel better.

If you already had a medical problem at the time of your concussion, it may take longer for you to recover from your brain injury. Anxiety and depression may also make it harder to adjust to the symptoms of brain injury.

While you are healing, you should be very careful to avoid doing anything that could cause a blow or jolt to your head. On rare occasions, receiving another concussion before a brain injury has healed can be fatal.

Even after your brain injury has healed, you should protect yourself from having another concussion. People who have had repeated brain injuries, such as boxers or football players, may have serious problems later in life. These problems include difficulty with concentration and memory and sometimes with physical coordination.

Tips for Healing — Adults

Here are a few tips to help you get better:

- Get plenty of sleep at night, and rest during the day.
- Return to your normal activities gradually, not all at once.
- Avoid activities that could lead to a second brain injury, such as contact or recreational sports, until your doctor says you are well enough to take part in these activities.

- Ask your doctor when you can drive a car, ride a bike, or operate heavy equipment because your ability to react may be slower after a brain injury.
- Talk with your doctor about when you can return to work or school. Ask your doctor about ways to help your employer or teacher understand what has happened to you.
- Consider talking with your employer about returning to work gradually and changing your work activities until you recover.
- Take only those drugs that your doctor has approved.
- Don’t drink alcoholic beverages until your doctor says you are well enough to do so. Alcohol and certain other drugs may slow your recovery and can put you at risk of further injury.
- If it’s harder than usual to remember things, write them down.

- If you're easily distracted, try to do one thing at a time. For example, don't try to watch TV while fixing dinner.
- Consult with family members or close friends when making important decisions.
- Don't neglect your basic needs such as eating well and getting enough rest.

Tips for Healing — Children

Parents and caretakers of children who have had a concussion can help them heal by:

- Having the child get plenty of rest.
- Making sure the child avoids activities that could result in a second blow or jolt to the head — such as riding a bicycle, playing sports, or climbing playground equipment — until the doctor says the child is well enough to take part in these activities.
- Giving the child only those drugs that the doctor has approved.

- Talking with the doctor about when the child should return to school and other activities and how to deal with the challenges the child may face.
- Sharing information about concussion with teachers, counselors, babysitters, coaches, and others who interact with the child so they can understand what has happened and help meet the child's needs.

Where to Get Help

Help for People With Brain Injuries

"It was the first time in my life that I couldn't depend on myself."

There are many people who can help you and your family as you recover from your brain injury. You don't have to do it alone.

Show this brochure to your doctor or health care provider and talk with them about your concerns. Ask your doctor whether you need specialized treatment and about the availability of rehabilitation programs.

Your doctor may be able to help you find a health care provider who has special training in the treatment of concussion. Early treatment of symptoms by professionals who specialize in brain injury may speed recovery. Your doctor may refer you to a neurologist, neuropsychologist, neurosurgeon, or specialist in rehabilitation.

Keep talking with your doctor, family members, and loved ones about how you are feeling, both physically and emotionally. If you do not think you are getting better, tell your doctor.

For more information, see *Resources for Getting Help* on page 17.

Help for Families and Caregivers

"My husband used to be so calm. But after his injury, he started to explode over the littlest things. He didn't even know that he had changed."

When someone close to you has a brain injury, it can be hard to know how best to help. They may say that they are "fine" but you can tell from how they are acting that something has changed.

If you notice that your family member or friend has symptoms of brain injury that are getting worse or are not getting better, talk to them and their doctor about getting help. They may also need help if you can answer YES to any of the following questions:

- Has their personality changed?
- Do they get angry for no reason?
- Do they get lost or easily confused?
- Do they have more trouble than usual making decisions?

You might also want to talk with people who have experienced what you are going through. The Brain Injury Association can put you in contact with people who can help (see page 17).

Resources for Getting Help

"I thought I was all alone, but I'm not. There are lots of people out there who understand what I've been through."

Several groups help people with brain injury and their families. They provide information and put people in touch with local resources, such as support groups, rehabilitation services, and a variety of health care professionals.

Among these groups, the Brain Injury Association (BIA) has a national office that gathers scientific and educational information and works on a national level to help people with brain injury. In addition, 44 affiliated state Brain Injury Associations provide help locally.

You can reach the BIA office by calling the toll-free **BIA National Help Line at 1-800-444-6443**. You can also get information through the national **BIA Web site at www.biausa.org.** Both the Help Line and the Web site can provide you with information about your closest state Brain Injury Association.

More information about brain injury is available through the Centers for Disease Control and Prevention (**CDC) Web site at www.cdc.gov/ncipc/tbi.**

For More Information:

- **BIA National Help Line: 1-800-444-6443**
- **BIA Web site: www.biausa.org**
- **CDC Web site: www.cdc.gov/ncipc/tbi**
- **State Brain Injury Association**

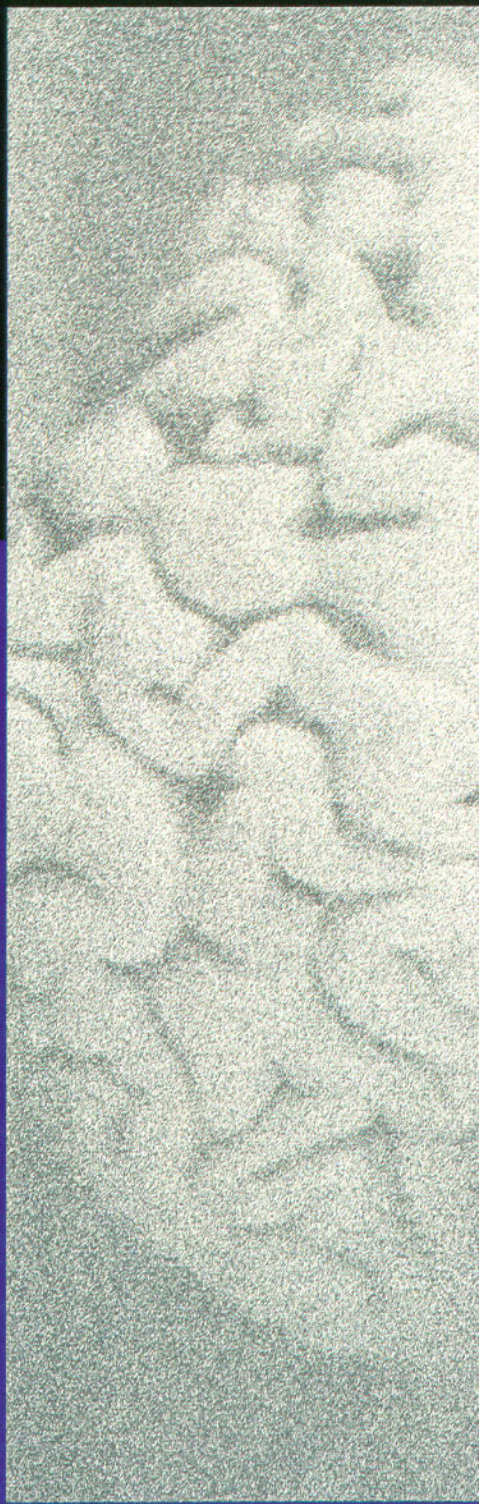
A blow or jolt to the head can cause a type of mild brain injury called a concussion.

Some symptoms of a concussion are:

- **Persistent low-grade headaches**
- **Having more trouble than usual remembering things, concentrating, or making decisions**
- **Feeling tired all the time**
- **Feeling sad, anxious, or listless**
- **Becoming easily irritated for little or no reason**

For more information on danger signs, symptoms, tips for getting better, and where to go for help, look inside this brochure.

U.S. DEPARTMENT OF HEALTH AND HUMAN SERVICES
Centers for Disease Control and Prevention
National Center for Injury Prevention and Control



Appendix 4.
Scandinavian Guideline: Brain concussion information to patients and their families

You have been examined and/or observed in the hospital after a head injury with possible brain concussion. We observed no signs of severe brain injury and found it therefore safe to discharge you. This is to inform you about some problems that may arise after discharge.

Could serious complications appear?

Acute complications are rare after careful evaluation in the hospital. The following symptoms should, however, lead to prompt contact with a physician or the hospital for repeated evaluation:

- increasing severe headache
- repeated vomiting
- reduced level of consciousness (difficult to wake up)
- confusion

Which symptoms are normal?

A mild head injury may transiently cause some of the following symptoms:

- moderate headache
- nausea
- dizziness
- reduced memory
- poor concentration

These symptoms are common during the first days after the injury. They usually resolve spontaneously, but some patients may experience moderate symptoms for weeks and months.

What should you and your family do?

You should not be alone and you should be woken up twice during the first night after the injury, to make sure that your reactions are still normal. During the next days, we recommend that you restrict the following activities until your symptoms have resolved:

- long lasting TV watching or reading
- computer/video games
- alcohol
- sports involving risk of a new head injury (i.e., football, downhill skiing)

You may use a prescription-free analgesic drug (i.e., paracetamol) if you have a headache. Your doctor may order a brief sick leave depending on your condition and occupation.

Should you see a doctor again?

If you have persistent symptoms despite following our advice, contact your GP for further advice and eventual prolongation of your sick leave.

Appendix 5. Definitions and acronyms

- Closed head injury is injury to the head due to blunt, contact or acceleration-deceleration type of trauma to the head which does not involve loss of consciousness, amnesia or focal neurological signs. Open head injury ?
- Brain injury is defined as damage to the brain, which occurs after birth and is not related to a congenital or a degenerative disease. Brain injury usually involves loss of consciousness, amnesia or focal neurological sign depending on the area of the brain involved. The impairments may be temporary or permanent and cause partial or functional disability or psychosocial maladjustment.
- MTBI - **M**ild **T**raumatic **B**rain **I**njury is defined as the mild injury to the brain tissue as the result of closed head injury on which the patient had Glasgow Coma Scale of 13-15 at admission (to the emergency department for example) and loss of consciousness ≤ 30 minutes and or post traumatic amnesia ≤ 24 hours with no focal neurological signs and negative intra-cranial lesions as shown by CT scan
- GCS - **G**lasgow **C**oma **S**cale is a tool to measure the depth of coma. GCS has a maximum score of 15. GCS is a composite score of 3 measurements involving eye opening (max score 4), best verbal response (max score 5) and best motor response (max score 6)
- PTA - **P**ost **T**raumatic **A**mnesia is a condition on which brain injured patient forget about the event before (retrograde) or after (anterograde) the traumatic event/injury
- LOC - **L**oss **O**f **C**onsciousness
- Malingering - is the intentional production of false or greatly exaggerated symptoms for the purpose of attaining some identifiable external reward. Some areas of potential exaggeration include pain, stiffness, dizziness, depression, memory disturbance, poor concentration, personality changes, blindness or visual loss, numbness, mobility restriction or range of motion, amnesia.
- PubMed is a database on medical literature that is developed the National Library of Medicine and managed by the National Center for Biotechnology Information. It contains the bibliographic information (most of the time include the abstract) of published medical literatures on any topics since 1966. PubMed is available for free.
- Cochrane Library Database is a commercial database that is developed and maintained by the Cochrane Collaboration Group. There various aims of the group, one of the aim is to synthesize and to provide best evidence in health care.
- NICE - **N**ational **I**nstitute for **C**linical **E**xcellence of England and Wales
- DARE - **D**atabase of **A**bstracts of **R**eviews of **E**ffects (at the University of York)
- AHRQ - **A**gency for **H**ealthcare **R**esearch and **Q**uality of the US Department of Health and Human Resources
- US-CDC - **U**nited **S**tates **C**enter for **D**isease **C**ontrol and Prevention
- INAHTA - **I**nternational **N**etwork of **A**gencies for **H**ealth **T**echnology **A**ssessment
- Systematic review is a concise summary of the best available evidence that address sharply defined clinical questions. It is developed by employing explicit and rigorous methods to identify, critically appraise and synthesize relevant studies. In its process, systematic review assembles and examines all of the available high quality evidence that are relevant to the clinical questions being asked.

- DAI - **D**iffuse **A**xonal **I**njury is the disruption of axon and associated small blood vessels along the longitudinal axis of the brain due to sudden deceleration forces in the brain.
- PCS - **P**ost **C**oncussive/**C**oncussion **S**yndrome
- PPCS - **P**ersistent **P**ost **C**oncussive/**C**oncussion **S**yndrome
- CT scan - **C**omputed **T**omography Scanning (see Table 3)
- MRI - **M**agnetic **R**esonance **I**maging (see Table 3)
- PET scan - **P**ositron **E**mission **T**omography scanning (see Table 3)
- SPECT - **S**ingle **P**hoton **E**mission **C**omputed **T**omography (see Table 3)
- SBU - Swedish Council on Technology Assessment in Health Care