Ontario

Traumatic Brain Injury

Discussion paper prepared for

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This medical discussion paper will be useful to those seeking general information about the medical issue involved. It is intended to provide a broad and general overview of a medical topic that is frequently considered in Tribunal appeals.

Each medical discussion paper is written by a recognized expert in the field, who has been recommended by the Tribunal's medical counsellors. Each author is asked to present a balanced view of the current medical knowledge on the topic. Discussion papers are not peer reviewed. They are written to be understood by lay individuals.

Discussion papers do not necessarily represent the views of the Tribunal. A vice-chair or panel may consider and rely on the medical information provided in the discussion paper, but the Tribunal is not bound by an opinion expressed in a discussion paper in any particular case. Every Tribunal decision must be based on the facts of the particular appeal. Tribunal adjudicators recognize that It is always open to the parties to an appeal to rely on or to distinguish a medical discussion paper, and to challenge it with alternative evidence: see *Kamara v. Ontario (Workplace Safety and Insurance Appeals Tribunal)* [2009] O.J. No. 2080 (Ont Div Court).

TRAUMATIC BRAIN INJURY

Traumatic brain injury (TBI) is an injury from forces transmitted to the brain from an impact to the head. The brain injury may be trivial, and completely reversible by natural healing processes, or it may be serious with varying degrees of lasting damage. The term "head injury" implies trauma to the surface of the head, but does not necessarily imply any injury to the brain, and will not be used in this discussion paper.

In addition to a blow to the head, forces may be transmitted to the skull from a blow to the jaw, and in a few specific instances, from trauma to the spine. Although the skull protects the brain, it does not absorb the impact of a violent force. The brain is of gelatin-like consistency, and is surrounded by a thin layer of fluid ("cerebro-spinal fluid or "CSF"). An abrupt blow to the head, or violent deceleration, can cause the brain to be displaced inside the skull, and subjected to various strains and stresses. It is thought that the most common mechanism of injury to the brain is rotational acceleration/deceleration, with distortion, deformities, and sheer-strain forces in various areas of the brain. Other types of injury include contusion of the brain under the site of a blow, especially if the bone is fractured, or injury to the surface of the brain if the brain is forcibly displaced against the inner surface of the skull, often at an area opposite the site of the blow (called "contre-coup injury"). Thus, brain injuries often occur at areas remote from the site of a blow to the head.

There are billions of microscopic-sized nerve cells, called neurons, in the brain, located in the "grey matter" on the brain surface (the "cortex") and in the deeper parts of the brain including the "basal ganglia" and the "brain stem". Neurons can be likened to complex micro-chips. Neurons connect to each other through long processes, like miniature wires, called "axons". Axons are the means by which neurons communicate and network with each other. Axons travel in short or long bundles or pathways throughout the brain, and to the spinal cord and the nerves to the face, eyes and ears ("cranial nerves"). In an injury axons may be stretched or torn, often in the deeper parts of the brain. The process is termed "diffuse axonal injury". Damage to axons may be temporary or lasting. Tiny hemorrhages (called "petechiae"), due to rupture of miniature blood vessels, may be scattered diffusely in deeper parts of the brain that have been subjected to violent sheer stresses. Larger hemorrhages from rupture of larger blood vessels that have been injured, may occur within the brain ("intra-cerebral hemorrhage"), or on the surface of the brain ("sub-dural" or "extra-dural" hemorrhage). Brain swelling ("edema"), either localized or diffuse, often occurs in more severe injuries.

Thus there is a wide spectrum of injury, from trivial to serious, from reversible to permanent, and often at a site or sites remote from the site of the blow to the head. Brain injury usually occurs at the time of the trauma, but some pathological processes, such as edema or hemorrhage, or complex biochemical changes, may be delayed during the first days or even weeks.

Skull fracture. A significant amount of force is required to fracture the adult skull. The amount of injury to brain tissue may range from surprisingly little to very severe. Some or most of the energy of the blow may be expended in breaking the skull, rather than being transmitted through the brain; this can occur where the bone tends to be thin, such as the frontal region. However, the bone at the back of the skull ("occipital region") is very thick, and almost all of the energy of a blow to the back of the skull may be transmitted to the brain. When bone fragments are driven inward by a small object, the injury is called a "depressed skull fracture". In most depressed skull fractures, the energy from the blow is expended in crushing the bone underneath the blow, with insufficient energy remaining to cause injury to brain areas remote from the local injury. There may or not be localized injury to the brain surface underlying the depressed fracture.

Natural evolution/healing. There is a natural healing process after brain injury. Hemorrhages absorb, swelling (edema) subsides, nerve cells and their connections, if not permanently destroyed, can recover and resume function. New nerve pathways and connections can form and assume functions that have been lost to injured pathways and connections (so-called "plasticity" of the brain). However, nerve cells that have been badly injured, or axons that have been severed, do not recover.

Concussion. Concussion is traumatically induced transient loss of normal brain function. There may or may not be loss of consciousness. Concussion is characterized by immediate and transient impairment of mental function, such as confusion, disorientation, attentional dysfunction, or other transient neurological symptoms such as slurred speech or visual impairment.

There are degrees or grades of concussion, depending on its severity. There are several different grading scales (see boxes). In all of them, Grade 1 concussion is defined as mild, very brief, neurological disturbance such as confusion, without loss of consciousness. In Grade 3 there is loss of consciousness, either brief or more prolonged. In all grades, there may be a short period of amnesia.

Cantu Guidelines
1. Mild, no LOC, PTA <30 minutes 2. Moderate, LOC <5 minutes, PTA >30 minutes 3. Severe, LOC >5 minutes, PTA >24 hours LOC=loss of consciousness PTA=post traumatic amnesia

Congress of Neurological Surgery Guidelines
1. Mild, no LOC, transient neurological disturbance 2. Moderate, LOC with complete recovery in <5 minutes 3. Severe, LOC lasting >5 minutes

Obviously, a patient cannot know whether he/she was or was not unconscious, and this can only be determined by the description of a witness who observed the event. Loss of consciousness does not necessarily imply greater neural damage or greater cognitive impairment than does concussion without loss of consciousness.

American Academy of Neurology Guidelines
1. Transient confusion, no LOC, symptoms resolve in <15 minutes 2. Transient confusion, no LOC, symptoms last >15 minutes 3. Any LOC, either brief or prolonged

The duration of amnesia is calculated to the time that the patient regained continuous memory after the event. As a general rule, longer periods of amnesia tend to be associated with more severe brain injuries, and vice versa. Therefore accurate estimation of the duration of amnesia is important in the overall evaluation of the severity of the injury. The most accurate estimation of the duration of amnesia is made soon after the patient has recovered. Estimation made retrospectively by the patient months or years after the injury tend to be unreliable, as the patient's memory is likely to be cloudy or distorted by then, (often after frequent questioning by physicians, psychologists and others years later).

Glasgow coma scale. The Glasgow coma scale is a simple 15-point scale that measures the level of consciousness by measuring eye opening, verbal responses and limb movement. 15 is normal. The scale is useful in the immediate post injury period, and helps to follow a patient's progress during the first hours and days after a severe head injury.

Symptoms after brain injury.
Immediate symptoms depend on the severity of the injury. They can include, in addition to those described under "concussion", varying degrees of impairment of limb movement, vision, speech, cognitive function and coma.

Delayed neurological deficit, such as the above, may occur in the hours, days or weeks after the trauma, due to a delayed pathological process such as edema or bleeding.

As the process of healing proceeds, many of these symptoms may improve, sometimes completely, and sometimes minimally, with varying degrees of lasting residual neurological deficit. Recovery occurs most rapidly in the first few months after injury, but recovery does continue during the first year, and continues in the subsequent year or two, although at a slower rate. Symptoms from injured brain tissue gradually improve, not worsen.

Delayed deterioration, with cognitive decline or new neurological symptoms is uncommon. Rare delayed complications include conditions such as hydrocephalus (obstruction to the flow of cerebro-spinal fluid through the chambers in the brain), and sub-dural hematoma (delayed hemorrhage on the surface of the brain). Apart from these rare conditions, symptoms that are claimed to worsen progressively in the months and years after a brain injury are not the result of some delayed pathological process in the brain, but are due to other factors such as depression, non-organic factors such as psycho-social issues, or a concurrent illness.

Post concussion syndrome. The term "post concussion syndrome" (PCS) refers to symptoms that a patient may have following a concussion or mild traumatic brain injury. PCS may include physical (somatic), cognitive and/or affective (mood) symptoms. The commonest somatic complaint is headache. Other somatic complaints may be fatigue, dizziness, nausea, blurred vision, noise and light sensitivity, and insomnia. Cognitive problems may include impaired attention, difficulty concentrating, poor memory and slowed thinking or reaction time. Affective or mood problems may include depression or anxiety. Many of the symptoms of PCS are due to a combination of organic and non-organic factors. These symptoms usually improve within a few weeks after injury, and in almost all cases they resolve between 3 and 12 months. In February 2004, The World Health Organization task force on Mild Traumatic Brain Injury published a review of 428 published studies related to prognosis after MTBI. The task force provided their synthesis of "best evidence" on prognosis from those studies which they accepted after critical review. They conclude that for adults, cognitive deficits and symptoms are common in the acute stage, and the majority of studies report recovery for most within 3-12 months" (1).

Persistent post concussion syndrome. In a small subset of patients, the symptoms of PCS do not recover, and may even worsen with time. Such symptoms are termed "Persisting Post Concussion Syndrome" (PPCS). There is continuing controversy in the medical community regarding the

patho-physiology of PPCS. One school proposes that the symptoms are a direct consequence of brain injury. Another school proposes that the symptoms are functional, and represent psychological or emotional sequelae of brain injury. There is evidence to support some aspects of both schools. Alexander (2,3) stresses that the symptoms and cognitive deficits in PPCS have no specificity, that is, they may occur in a variety of conditions and are not specific or unique to brain injury. There is great risk of inappropriately attributing PPCS to brain injury. It is important to carefully distinguish among the many possible causes of the symptoms, before attributing them to injured brain tissue. From their research, Saltz et al (4) conclude that it is unclear whether an initial mild head injury is even necessary or sufficient to cause the symptoms like those of PPCS. Unfortunately, some well-intentioned physicians and head injury treatment programs may reinforce the symptoms in the patient's mind, and persuade the patient that the symptoms are the result of serious brain damage.

Headaches are not caused by injury to the brain itself, for there are no pain-sensitive nerve endings within brain tissue. Pain nerve endings capable of generating headaches are located in the scalp, muscles outside the skull, the membrane covering the brain (dura mater) and large blood vessels. Headaches do not mean there has been brain injury. (Persisting severe headaches are uncommon after brain surgery or severe traumatic brain injury). Post traumatic headaches are considered in the WSIAT discussion paper on headaches.

Seizures. Post traumatic seizures may result from mild brain injury, but are more common following severe brain injury. The onset may be delayed many months after injury. The incidence of seizures following mild brain injury is in the range of 1% to 2%. The diagnosis of seizures by a neurologist depends upon the history and description of the attacks, together with EEG findings. Unfortunately some patients without a clearly established diagnosis of seizures, perhaps with "pseudo-seizures", or with non-specific EEG abnormalities, are inappropriately treated with anti-seizure medication on a speculative basis.

Diagnostic Tests

Diagnostic tests that may be used in evaluating a patient who may have had a brain injury include tests used shortly after the injury, to rule out a complication such as a blood clot that might require urgent intervention, and tests used weeks and months later in attempt to determine the cause of persisting symptoms. Procedures commonly used shortly after injury include

x-rays of the skull to rule out a fracture, and CT scan. Diagnostic tests that may be used to evaluate later symptoms such as post concussion syndrome are listed below.

- CT scan
  - Non-enhanced (non-contrast or plain)
  - Contrast (enhanced) with intravenous radio-opaque contrast material
- MRI
  - Non-enhanced
  - Enhanced (with intravenous contrast)
  - Using a variety of pulse sequences or 'weightings' (e.g. FLAIR, DWI)
  - Functional MRI (fMRI)
- Electrophysiological tests
  - EEG, Evoked potentials, "brain mapping"
- SPECT scan
- PET scan
- Neuro-psychological tests

CT and MRI are described as "anatomical imaging", as they can furnish knowledge of abnormalities in anatomical structures within the skull. SPECT scans, PET scans and fMRI (functional MRI) are described as "functional imaging", as they may provide insights into the patho-physiological and functional sequelae of brain injury, i.e. how various parts of the brain are functioning.

Anatomical Imaging

CT scans. Computerized Tomography is an x-ray picture of "slices" of the brain. X-ray beams are passed through the brain, the density of the various components of the tissue is measured, and a computer program arranges the images into slices. Each slice is usually 1 cm or 5mm thick. The brain can be sliced from front to back or from top to bottom. CT is most useful in detecting abnormalities such as large hemorrhage or brain contusion in the hours or days following trauma, in order to identify abnormalities that may require treatment, such as removal of a blood clot. Skull fractures can be seen on CT scans. The resolution of CT is insufficient to show tiny or microscopic lesions in the brain. Therefore CT is of little value in evaluating a patient months or years after a mild traumatic brain injury, other than to rule out an unexpected lesion, such as a tumor or hydrocephalus (obstruction to the flow of CSF within the chambers of the brain), to explain the patient's symptoms.

CT may be performed after intravenous injection of contrast material, which, because it is denser than brain tissue, blocks the passage of x-rays through tissues such as vascular tissue or tumours, where it may be concentrated. "*Contrast-enhanced CT"* helps to reveal structures and abnormalities that cannot be seen on plain or "*non-contrast CT"*. When reviewing a radiologist's report of a CT scan, it is helpful to note whether the scan was performed with or without contrast, as in some situations the examination may be considered incomplete if it was not contrast-enhanced.

MRI.[1] Magnetic Resonance Imaging. MRI provides extremely high-resolution images of the brain, with detail down to a very few millimeters. Any part of the brain can be imaged in any direction, in very thin slices. Each MRI examination is tailored to the specific information being sought in order to highlight possible abnormalities, and the radiologist does so by altering parameters of the magnetic field and radio-frequency (RF) energy being used.

Routine T1 and T2 images are sufficient to demonstrate larger lesions from trauma, such as contusion and hemorrhages, or focal loss of brain tissue. More subtle effects of trauma may or may not be detected in conventional images. There are a number of special techniques that may be used. Tiny hemorrhages from brain injury, deep in brain tissue, may not be seen in conventional imaging. *Gradient echo imaging* is a technique that highlights hemosiderin, which is an iron pigment product from blood, and when present, confirms areas of previous hemorrhage. *Diffusion weighted imaging* (DWI) can detect very small areas of edema. *Fluid attenuated inversion recovery* (FLAIR) images suppress the image of cerebro-spinal fluid, and are much more sensitive than standard spin echo images for certain tissues.

Magnetic resonance angiogram (MRA) is a technique to outline blood vessels by detecting flowing blood.

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[1] **How it's done:** When the patient lies in the scanner, a strong magnetic field aligns the hydrogen protons in various tissues. Then radio-frequency (RF) energy of a specific frequency is beamed through the tissues being examined, causing the protons to be temporarily "excited" and then relax, releasing RF energy which can be measured. Magnetic resonance imaging is a complex interaction between protons, the magnetic field and the RF energy. Standard "spin-echo" technique includes T1 and T2 images, which measure the rate of return of protons to equilibrium, or "relaxation times", and which vary in different tissues and disease processes. Sometimes *contrast material* is injected intravenously; it alters the magnetic field and enhances or highlights certain normal and abnormal tissue seen in the images. DELETE THIS LINE

When reviewing a radiologist's report of a MRI scan, it is important to determine whether the type of scan was appropriate to the information being sought by the patient's physician

Functional Imaging

Functional MRI (fMRI) is a technique to demonstrate how various areas of the brain are functioning. It does not provide images of anatomical structures. In fMRI, the patient, while in the MRI scanner, is asked to perform certain tasks, such as speech, cognitive, memory, sensory, or motor, and appropriate areas of the brain can be seen to "light up". fMRI is still a research tool.

Positron emission tomography (PET scanning) is essentially a research tool. PET scanners are few and extremely expensive. A radionuclide, which emits positrons, is combined with a sugar and injected into the patient, and the scanner, which looks much like a CT scanner, detects these positrons. PET scan can measure blood flow, oxygen consumption, glucose metabolism, neuro-transmitter concentrations in various areas of the brain. The images obtained can detect areas where nerve cells are working during a particular mental task, and detect areas that are not functioning normally at the time the scan is being done. As traumatic brain injury is an evolving dynamic process, areas of abnormal function do not necessarily represent areas of anatomical damage, nor predict clinical outcome.

Single photon emission computed tomography (SPECT scan) is a method of observing the physiologic behaviour of the brain. A radiotracer (99mTC) attached to a chemical such as HMPAO is injected into a patient. It emits gamma rays, which can be detected by a scanner (single photon tomography). The distribution of the tracer reflects the regional blood flow ("perfusion") and glucose metabolism, and thus can provide some information about the functional integrity (not anatomical integrity) of various brain areas at the time of the scan. Although SPECT images do not provide the same quality of information as PET scan images, they are fairly *sensitive* in detecting regions of decreased brain perfusion (presumably metabolic changes). However, SPECT scanning cannot determine the pathological cause of areas of hypo-perfusion, and thus lacks *specificity*. Areas of decreased perfusion may occur with certain medications, depression, and a variety of other conditions. The utility of SPECT scanning in mild traumatic brain injury is controversial and not proven, and there is a lack of adequate clinical trials. As noted under PET scans, traumatic brain injury is an evolving dynamic process; areas of decreased radio-tracer uptake in a SPECT scan may simply represent temporary metabolic changes, and do not necessarily represent areas of permanent anatomical or cellular damage, nor do they predict clinical outcome.

SPECT after head injury may reveal perfusion abnormalities that are not seen in as anatomic changes on CT or MRI, but "*the clinical significance of these changes is uncertain. The scientific literature does not now support the routine use of SPECT for the evaluation of patients with closed head injury or post concussion syndrome……. SPECT should continue to be used as an investigational tool for the study of mild head trauma*" (5)

As with all diagnostic tests, the findings of SPECT scan must be interpreted in the light of, and conclusions must be consistent with, the clinical picture and all the other diagnostic tests. It is inappropriate to use SPECT findings as confirmatory evidence that there is brain damage, or that any abnormal findings are the result of previous trauma, or to use SPECT as evidence to provide support for abnormal psychological findings.

ELECTRO-PHYSIOLOGICAL STUDIES (EEG, evoked potentials, and 'brain mapping'). EEG records electrical activity of areas of the brain from multiple scalp electrodes. EEG may sometimes be used in the evaluation of a patient with mild traumatic brain injury. After concussion EEG recordings are usually normal, or show non-specific changes. EEG is especially useful in a patient who has had seizures. There are no specific EEG abnormalities diagnostic of traumatic brain injury. 'Brain mapping' is still a research tool and its usefulness in the evaluation of a patient with mild traumatic brain injury has not been established.

Neuro-psychological Tests

Neuro-psychological (NP) testing can be useful in the assessment of patients with traumatic brain injury. Deficits such as disturbances in new learning and memory, reduced attention and slowed reaction time are often present in the first weeks after mild concussion. Significant improvement usually occurs within the first three months. More severe cognitive dysfunction may follow severe head injury, and tends to recover more slowly or incompletely. Because cognitive deficits gradually improve after brain injury, NP assessment is most useful in patients whose recovery is complete, or has largely plateaued, usually some months or longer after injury.

NP tests do have established validity and reliability, and their findings can be regarded with confidence provided they have been conducted by a competent and experienced neuro-psychologist. Many factors may influence NP findings, including the effects of pre-injury personality, aging, education, ethnicity and cultural influences, depression, psychiatric disturbances,

medication and substance abuse. The relevance of NP findings, in evaluating a patient, must be interpreted in the context of the nature and severity of the injury, and also in conjunction with all the clinical, imaging and laboratory information. The Therapeutics and Technology Assessment Subcommittee of the American Academy of Neurology published the following position statement: "*Neuro-psychological assessment is not intended to provide a diagnosis or to indicate the precise localization of a focal brain lesion*" (6). Dr. Donald Stuss, in a presentation to the Tribunal in 2002, stated "*There is (at present) no neuro-psychological test that provides unequivocal evidence of brain damage*" (7). For a better understanding of NP testing in patients who have suffered a possible brain injury, the reader is encouraged to review the excellent articles by Alexander (2) and by Stuss (8), copies of which are available in the Tribunal's library.

Treatment

After severe brain injury, acute management focuses on support of the comatose patient and detection and treatment of large blood clots, brain swelling and other complications. After mild traumatic brain injury, treatment is conservative and supportive, encouraging the patient to resume normal activities as soon as possible. Physiotherapy, occupational therapy and counseling may play a role. The cause of any late or persisting symptoms, beyond the expected time of recovery, must be sought and treated. There is always a risk that a treatment program may convince a patient with a minimal brain injury that there has been significant "brain damage", and thus reinforce residual symptoms.

Conclusions

Head injury does not necessarily mean brain injury. Brain injury does not necessarily mean brain damage. The severity of a suspected brain injury is measured by the acute injury characteristics such as the nature and severity of the trauma and the early post injury clinical state, and not by the severity of late symptoms. Improvement of some type occurs with almost all levels of severity of traumatic brain injury.

After mild traumatic brain injury, most symptoms clear within 3-12 months. In patients with persisting post concussion symptoms, there is no single test (such as neuro-psychology, anatomical or functional imaging, EEG) that can

prove or disprove that the symptoms are due to brain injury. In order to be valid, the findings of each test must be compatible with and appropriate to the particular injury, the post injury symptoms and their subsequent course. The findings of all the tests must be congruous with each other.

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