Douglas S. Wingate

Healing Brain Injury with Chinese Medical Approaches
Integrative Approaches for Practitioners
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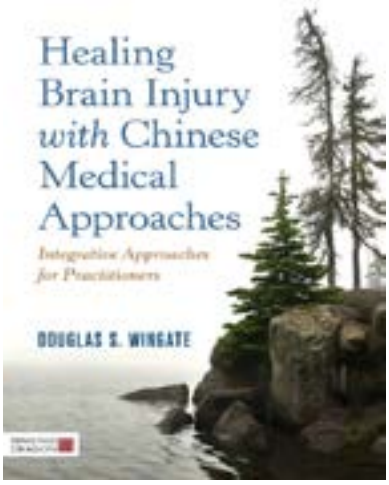

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Chapter 1

The "Silent Epidemic"

First, some definitions… Brain injuries that are neither congenital, hereditary, degenerative nor induced by birth trauma are collectively referred to as an acquired brain injury (ABI) and are the result of changes in neuronal activity that affect physical integrity, metabolic activity, or functional ability of the nerve cells of the brain. They can occur for a number of different reasons. An acquired brain injury can be placed into two general categories—traumatic and non-traumatic. Causes of each are listed in Table 1.1. Much of the treatment information in this text may be found useful for both, though it is primarily directed to cases of traumatic brain injury (TBI), which is defined as stemming from an external force. Direct impact injuries can be further divided into what are either closed injuries, in which brain lacerations or hemorrhages within the skull cause focal injuries, or open injuries, in which the tissue of the skull or the meninges have actually been breached.

TABLE 1.1 Causes of Brain Injury

Causes of Traumatic Brain Injury	Causes of Non-Traumatic Brain Injury
Falls	Stroke
Assaults	Infectious disease (meningitis, encephalitis)
Motor vehicle accidents	Seizure disorders
Sports and recreational injuries	Electric shock/lightning strike
Abusive head trauma/"shaken baby syndrome"	Tumors (surgery/radiation/chemo)
Gunshot wounds	Toxic exposures (substance misuse, lead ingestion, inhalation of volatile agents)
Workplace injuries	Metabolic disorders (insulin shock, diabetic coma, liver and kidney disease)
Child abuse	Neurotoxic poisoning (carbon monoxide, inhalants, lead exposure)
Domestic violence	Lack of oxygen to the brain (near drowning, airway obstruction, strangulation, cardiopulmonary arrest, hypoxia, anoxia)
Military actions/blast injuries	

Current medications and history of relevant psychotropic or anticonvulsive (for prophylaxis or a continuing seizure disorder) drugs and their efficacy should be noted and considered. Benzodiazepines can impair memory and coordination. Anticholinergic drugs can increase confusion. Food and drug allergies and intolerances should be noted and considered, especially in the case of prescriptions of herbal medications. Potential herb–drug interactions should also be assessed (commonly used pharmaceuticals noted in this text and their potential herb–drug interactions are listed in Appendix D).

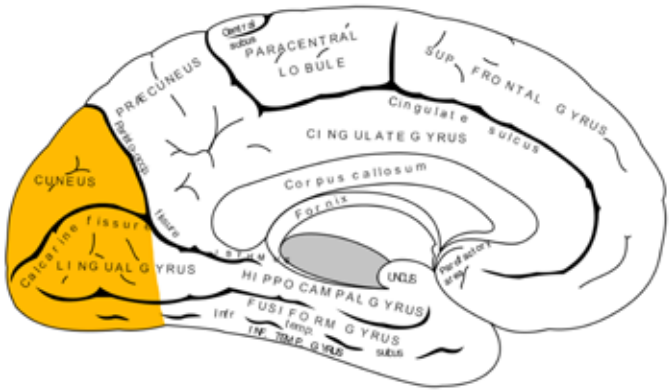
Relevant family medical and psychiatric history should be assessed to inform any potential impact on current emotional or psychological abnormalities or concerns. Inquiry into the individual's social history, family dynamic prior to and after injury, and other support structures are all relevant information as these can additionally play into mental–emotional states and the outlook of the individual toward healing. Losses and disruptions in an individual's social dynamic can provide severe additional stress which can act to slow the progress of recovery. Establishing new social connections or participating in leisure activities can become difficult, creating a sense of loneliness. If possible, a referral to a local support group should be considered where it is possible to socially interact with others who have experienced similar life events. Occupational status or pursuits are also of note as they can also be significant stressors.

TABLE 2.1 Traumatic Brain Injury Symptom Checklist

Cognitive	Level of consciousness Sensorium Attention/concentration Short-term memory Processing speed Thought processes Executive function (planning, abstract reasoning, problem solving, information processing, multi-tasking, insight, judgment, etc.)
Emotional	Mood/lability Depression Hypomania/mania Anxiety Anger/irritability Apathy
Behavioral	Impulsivity Disinhibition Anger dyscontrol Inappropriate sexual behavior Lack of initiative Change in personality

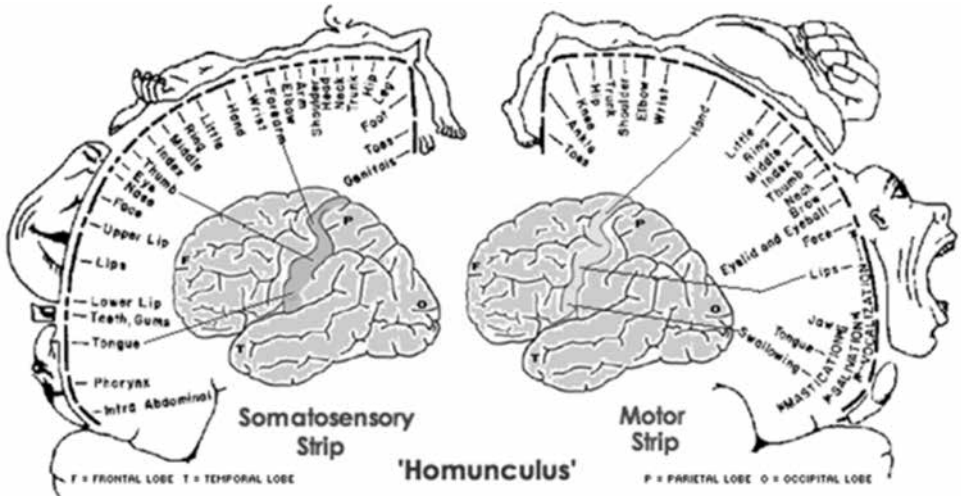
Chapter 6

Occipital Lobe



Functional Overview

The occipital lobe is composed of two basic subsections, the primary visual cortex and the visual association area. Simple and complex visual and central/foveal analysis is one of the main functions associated with the occipital lobe; however, neurons also tend to a number of modalities including vestibular, acoustic, visual, visceral, and somesthetic input. These are subdivided into four visual cortices. The primary visual cortex, also called V1, is located predominantly within the medial walls of the cerebral hemispheres and is concerned with the elementary aspects of form perception, transforming information from the retina and lateral geniculate nucleus into a basic code that enables visual information to be extracted by later stages of processing. It is particularly specialized in processing spatial information such as static and moving visual stimuli, colors, and pattern recognition, though the information coming into V1 from the retina is coded as an edge detection rather than discrete optical imagery. A spatial mapping of the subjective visual field with a specific location within V1 is very precise, to where even blind spots may be mapped onto it. This is the most ancient visual portion of the brain and found in most mammals.



Somesthetic (Supplementary) Association Area

This region has connections stemming from the primary somesthetic cortices, contralateral primary zone of opposite hemisphere (area 5ab only), motor association areas (area 6) in the frontal lobe, and posterior thalamus (i.e. ventral posterior complex). It controls and contains representations of both halves of the body. The trunkal area in particular is represented in this region in a way that the body is bilaterally represented in the secondary sensory areas. This being said, bilateral representation is mostly maintained in the right half of the brain. A detailed representation of the cutaneous surface, in particular the hand and face, is maintained here. A small percentage of cells in area 5 also appear to be concerned with more complex activities such as the movement of the hand and arm and the manipulation of objects. Electrical stimulation of area 5 can result in limb movements. Other neurons in area 5 are especially responsive to specific temporal–sequential patterns of sensation and can determine the direction and rhythm of movement. The determination of positional interrelationships is also interpreted here through comparisons with a more stable image of the body in areas 3ab, 1, and 2.

Polymodal Receiving Area (Area 7 and Superior–Posterior Parietal)

The polymodal receiving area is concerned with the analysis and integration of the highest order visual, auditory, and somesthetic information. Single neurons often have quite divergent capabilities and the ability to monitor many different body parts simultaneously. Through this, it is able to create a three-dimensional image of the body in space. It coordinates/guides gaze and whole-body–positional movement through visual and auditory space, continually updating information of relations between internal and external coordinates. Neurons in area 7 are thought to execute a matching function between the internal drive state of the

An mTBI is caused by transfer of kinetic energy into soft tissue, which can lead to chemical/metabolic changes in the brain or direct damage to physical structures such as brain cells. The most common form of damage is diffuse axonal injury. Axonal shearing can also occur in which axons become twisted and disconnected.

The most important chemical change that occurs is a massive release of the neurotransmitter glutamate. Very high levels of glutamate are toxic to the brain as they affect sodium/potassium balances that allow for proper neural signal transmission. ATP, the body's energy currency, also becomes compromised in a concussion by other chemical processes that starves the brain for energy. This may be further impacted by a general constriction of blood flow.

Symptoms

In cases of mTBI most symptoms resolve within a time of about two to four weeks, with only an estimated 10–15 percent of individuals experiencing chronic symptoms. Most symptoms can be categorized as either physical/somatic, cognitive, or behavioral/emotional, as laid out in Table 13.1.

TABLE 13.1 Symptoms Associated with Mild Traumatic Brain Injury

Physical/Somatic Symptoms	Cognitive Symptoms	Behavioral/Emotional Symptoms
Headache	Inattentiveness	Depression
Fatigue	Diminished concentration	Anxiety
Seizure	Poor memory	Agitation
Nausea	Impaired judgment	Irritability
Numbness	Slowed processing speed	Aggression
Poor sleep	Executive dysfunction	Impulsivity
Light/noise sensitivity		"Frontal release": disinhibition, emotional lability, social inappropriateness
Impaired hearing		
Blurred vision, convergence insufficiency		
Dizziness/loss of balance (BPPV, orthostatic hypertension)		
Neurologic abnormalities		

Chronic Traumatic Encephalopathy (CTE)

CTE is a rare, progressively degenerative condition of the central nervous system which typically follows repetitive brain trauma. This may be due to high-risk sports such as football or boxing. Here diffuse axonal injury causes a release of Tau proteins which are changed structurally by the metabolic breakdown of brain cells following trauma to create a chronic inflammatory state that causes a progressive degeneration of the central nervous system.

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