Jean Elbaum *Editor*

Acquired Brain Injury

An Integrative Neuro-Rehabilitation Approach

Second Edition

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Jean Elbaum
Transitions of Long Island
Northwell Health
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Chapter 1
Introduction

Jean Elbaum

Thirty plus years in the field of neuro-rehabilitation, and each day still brings new challenges and new learning. The resilience of the brain and the exciting recoveries that are facilitated in survivors of acquired brain injuries (ABIs) reinforce the value and power of an integrated team effort. Shifting survivors from states of brokenness to productive, meaningful lives continues to be the chief reward.

The best way to achieve excellent outcomes for our clients and families is by ensuring a comprehensive, integrated approach that covers the continuum of care, allowing clients to be supported from the earliest stages of recovery throughout their rehabilitation, providing programming that is evidence based, purposeful and functional, as well as offering post rehabilitation options well matched to clients' needs.

A specialized team approach to neuro-rehabilitation with each member assuming a different, yet interconnected role is vital. The survivor and family must know that their care is being coordinated as well as the function of each of their clinicians. All rehabilitation team members must be knowledgeable about the different roles of their colleagues and maintain open communication that crosses interdisciplinary borders.

Much has changed over the last decade, primarily in concussion management as well as in the use of ever developing technology to facilitate recoveries. What has stayed the same is the criticality of helping clients remove barriers towards progress and teaching compensatory strategies to work around residual challenges. The true team effort includes not only the therapy team, client, and family, but may include the employer or school/university to which the client is reintegrating.

Thus, the goal of this text is to provide an introduction to many of the key members of the neuro-rehabilitation team, including their roles, approaches to evaluation, and treatment. The book was written for interdisciplinary students of

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neuro-rehabilitation as well as practicing clinicians interested in developing their knowledge in both their field as well as other discipline areas. It can also be useful for survivors and families to help untangle and clarify the complexities of the rehabilitation process. Case examples were included to help illustrate real life challenges.

Based on feedback from colleagues and students, the second edition excluded certain chapters and added others. Existing chapters were updated to include new research and current technologies. Kwan, Schneider, Narayan, and Ullman (Chap. 2) describe the role of the neurosurgeon in treating clients post acquired brain injuries. Duarte and Patel (Chap. 3) and Khan, Patel, and Vasquez-Cascals (Chap. 4) describe the central roles of neurology and physiatry in diagnosing and treating clients post-ABI. They highlight the importance of team collaboration and discuss topics such as neuroplasticity, spasticity management, concussion management, headaches, seizures, sleep disorders, and new areas of study such as stem cell research in acquired brain injury. Chang, Saul, and Volpe (Chap. 5) provide a state-of-the-art review of the efficacy of robotics in neuro-rehabilitation. Han (Chap. 6) describes common visual difficulties post-ABI and the role of the neuro-optometrist. Napoleone, Silberglied, L'Abbate, and Fried (Chap. 7) and Henderson, Jensen, Drucker, and Lutz (Chap. 9) discuss the essential roles of the occupational therapist and the speech/language pathologist on the neuro-rehabilitation team. Murray, Aquino, and Nugent (Chap. 8) provide a comprehensive review of physical challenges post acquired brain injury which was missing from our first edition. Scicutella (Chap. 10), Pachilakis and Mirra (Chap. 11), and Elbaum (Chap. 12) discuss the emotional, behavioral, and neuropsychological challenges post-ABI and the importance of addressing these difficulties through an integration of evaluation, proper medication management, and counseling. Specific family challenges and ways to meet their needs effectively through appropriate interventions are reviewed in a separate chapter (Chap. 13). Muscatello and Elbaum (Chap. 14) review the value of post-rehabilitation programs for clients who aren't ready or able to return to work or school. Outstanding recoveries are highlighted in the final chapter by a former client and current Broadcast Journalist, Jessica Moskowitz.

I'd like to thank all the clients and families that have been part of the Transitions' family over the last three decades. I'm impressed on a daily basis by the persistence, devotion, sacrifices, and constructive attitudes we see in the face of highly difficult and complicated situations.

I'd also like to thank my colleagues that worked on this new edition and approached the task with interest and enthusiasm. The goal was to assemble the key components of the neuro-rehabilitation team in an organized, meaningful, and engaging manner.

Chapter 2
Neurosurgery and Acquired Brain Injury

Kevin Kwan, Julia Schneider, Raj K. Narayan, and Jamie S. Ullman

Introduction

An integrated neuro-rehabilitation approach toward the treatment of acquired brain injuries begins with a fundamental understanding of neurosurgical pathologies. Improvements in the neurosurgical knowledge of not only the neuro-rehabilitation team but also the patient family unit allow more cohesive participation and improvements in patient's outcomes. The purpose of this chapter is to provide a framework for the *epidemiology*, *symptoms*, *diagnoses*, *treatment paradigm*, *and outcomes* of the most frequently encountered *operative neurosurgical pathologies* in the contemporary era. It is expected that this information can be utilized by the integrated neuro-rehabilitation team and family unit to improve patient outcomes.

Brain Anatomy and Physiology

The brain provides numerous essential functions. Besides providing information about the environment from our five senses, the brain also mediates cognition, recollection, speech, movement, touch, and systemic homeostasis. The brain is an organized structure, divided into three main components: *the cerebrum*, *the*

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cerebellum, *and the brain stem*. These vital structures are encased by the bones of the skull known as the cranium, protecting it from injury.

The cerebrum, which forms the major portion of the brain, is divided into two major parts: the right and left cerebral hemispheres. Each hemisphere is subsequently divided into different sections or lobes: the frontal, parietal, temporal, and occipital lobes. The frontal lobe is responsible for thinking, making judgments, planning, decision-making, and conscious emotions. The parietal lobe is mainly associated with spatial computation, body orientation, and attention. The temporal lobe is concerned with hearing, language, and memory. The occipital lobe is dedicated to visual processing. Any damage to a particular part of the brain may result in a relative loss of function dedicated to that area.

The cerebellum is located at the back of the brain beneath the occipital lobes. The cerebellum fine tunes motor activity or movement. It helps maintain central posture and fine tunes the movements of the peripheral limbs. The cerebellum is important in one's ability to perform rapid and repetitive actions such as playing a piano.

The brain stem is the lower extension of the brain, located in front of the cerebellum and connected to the spinal cord. It consists of three structures: the midbrain, pons, and medulla oblongata. The midbrain is an important center for ocular motion, while the pons is involved with coordinating eye and facial movements, facial sensation, hearing, and balance. The medulla oblongata controls breathing, blood pressure, heart rhythms, and swallowing. Messages from the cortex to the spinal cord and nerves that branch from the spinal cord are sent through the brain stem. Damage of this essential and primitive region of the brain, i.e., due to a stroke, may result in sudden death (AANS, 2018).

Initial Neurosurgical Evaluation

Upon presentation of a patient with acquired brain injury, often emergently, the priority for the multidisciplinary trauma team is for airway stabilization and cardiovascular circulatory optimization. Securing the airway may require the insertion of an endotracheal tube with ventilator support. Blood pressure stabilization may also require adjuvant pharmacological support. The team must also quickly assess the patient's neurologic exam using an abridged format, which is often denoted using the Glasgow Coma Scale (GCS). Noted in Fig. 2.1, this scale is divided into three segments, including eye opening (4 points), verbal response (5 points), and motor response (6 points) to stimuli, for a total of 15 points (Bateman, 2001). Any patient with evidence of trauma or with an impaired GCS score must have a computed tomography (CT) scan completed following initial stabilization. The CT scan of the head is sensitive for demonstrating the presence of hemorrhage or edema in the brain, as well as any evidence of a fracture within the cranium. Emergent neurosurgical management is subsequently dictated by the patient's history, physical exam, and radiographic findings.

Eye Opening (4)	Verbal Response (5)	Motor Response (6)
No Eye Opening (1)	No Verbal Response (1)	No Motor Response (1)
Eye Opening to Pain (2)	Incomprehensible (2)	Extensor posturing (2)
Eye Opening to Voice (3)	Inappropriate words (3)	Flexor posturing (3)
Spontaneous (4)	Confused Speech (4)	Withdrawal (4)
	Oriented Fully (5)	Localizing (5)
		Following Commands (6)

Fig. 2.1 Glasgow Coma Scale (Bateman, 2001)

Acquired Brain Injury

Acquired brain injury (ABI) refers to post-natal cerebral damage, rather than an insult occurring as part of a hereditary disorder (Ontario Brain Injury Association, 2018). ABI is classically subdivided into traumatic and nontraumatic subtypes (Prins, Greco, Alexander, & Giza, 2013). Consequences of ABI often require a major life alteration around the patient's new conditions, and making that modification has a critical influence on recovery and rehabilitation (Tate et al., 2014). This alteration, however, depends mainly upon the nature and severity of the specific neurologic injury.

Chapter Outline
This chapter will seek to illustrate the symptoms, diagnosis, treatment, and outcomes from ABI as a result of **traumatic brain injury** (concussion, epidural hematoma, subdural hematoma, and penetrating injury) or **nontraumatic brain injury** (spontaneous intracranial hemorrhage, malignant cerebral infarction, brain tumor, and aneurysmal subarachnoid hemorrhage).

Traumatic Brain Injury

Concussion (Mild Traumatic Brain Injury)

Concussion is a diffuse subtype of mild traumatic brain injury and afflicts an estimated 1.4–3.8 million people in the United States per year (Laker, 2011). The diagnosis of a patient with a concussion is mainly clinical, with patients presenting

with nonspecific symptoms such as headache, dizziness, nausea, imbalance, or incoordination. Often patients may present in a delayed fashion, days, weeks, or even months after the initial traumatic event with persistent symptomatology (Kushner, 1998). Radiographic evaluation, usually with computed tomography (CT) or magnetic resonance imaging (MRI), is classically normal in nature. Patients with mild traumatic brain injury are often managed conservatively with medication for their symptomatology. Routine re-imaging may be necessary if symptoms persist or delayed focal neurologic deficits occur. Early involvement of rehabilitation specialists who focus on traumatic brain injury is essential to expedite patient recovery and resumption of activities of daily life (Fraser, Matsuzawa, Lee, & Minen, 2017).

Epidural Hematoma

Epidural hematomas (EDHs) represent 3% of head injuries, occurring mostly between 10 and 30 years of age as the dura is more attached to the cranium as one ages. EDHs may occur secondary to tearing of the middle meningeal artery, middle meningeal vein, or dural sinus (Bullock, 2006). Presentation can be acute, subacute, or chronic, but classically patients present with a lucid interval before deterioration. EDHs are often diagnosed on initial computed tomography (CT) scans on patient presentation, which often manifest with a lentiform biconvex appearance that does not cross suture lines due to dural attachments. Craniotomy or craniectomy for surgical evacuation of the hematoma is necessitated if patients have a neurologic deficit as a result of the mass effect (Williams, Levin, & Eisenberg, 1990).

Subdural Hematoma

Subdural hematomas (SDH) can occur in 10–35% of severe head injuries. SDH develop from ruptured bridging veins following acceleration, deceleration, and rotational forces to the cranium. Risk factors can include use of anticoagulation, alcoholism, or cerebral atrophy. Presentation can be acute, subacute, or chronic in nature. When diagnosed initially on CT, they tend to be crescent shaped and cross suture lines but not dural attachments. Surgical evacuation of the hematoma is necessitated if patients have a neurologic deficit as a result of the mass effect. Generally, if the SDH is acute in nature, a larger craniotomy is utilized for the surgical evacuation. Conversely, if the SDH is chronic in nature, a smaller burr hole or craniotomy is utilized for the surgical evacuation (Karibe et al., 2014).

Case Study: Subdural Hematoma

Clinical Presentation: A 75-year-old female with past medical history of moderate/severe Alzheimer's disease (nursing home resident with full assistance of activities of daily living) was found down by nursing staff with a left forehead abrasion. Upon arrival, patient was initially oriented to person and conversant, then deteriorated and became minimally responsive. Pt exam deteriorated to a Glasgow coma score of 7 (no eye opening, no verbal response, localized to painful stimuli), and she was intubated for respiratory protection.

Diagnostic Imaging: CT head initially showed a left SDH (blue arrow), with a left temporal parietal parenchymal hematoma (red arrow) with surrounding edema (Fig. 2.2). On a subsequent scan, there was greater than 1 cm midline shift with compression of the lateral ventricle.

Management: The patient was taken emergently to the operating room for a left craniotomy with placement of an intracranial pressure (ICP) monitor.

Clinical Course: Patient was started on video electroencephalography which showed increased risk for focal onset seizure bi-frontally. Patient was started on anti-epileptics. Intracranial pressures continued to be low and the ICP monitor was discontinued. The patient received a tracheostomy, percutaneous endoscopic gastrostomy tube and was transferred to a long-term rehabilitation facility. On long-term follow up, the patient still requires full assistance with activities of daily living.

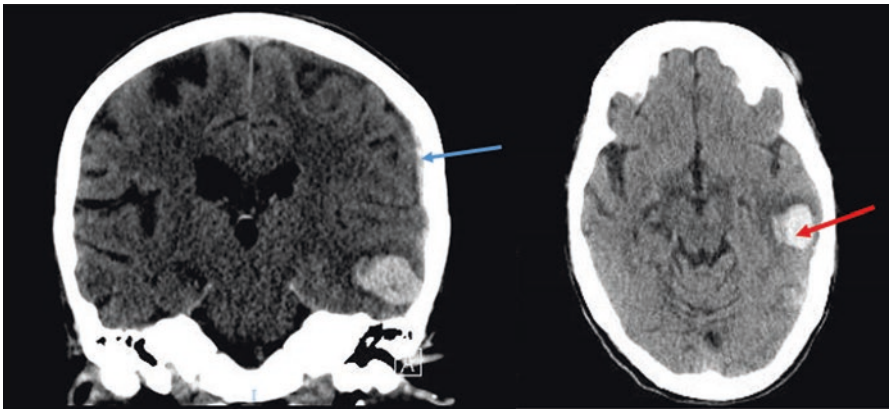

Fig. 2.2 Initial computed tomography scan of head, coronal slice (left) and axial slice (right), demonstrating a left temporal subdural hematoma and parenchymal hemorrhage

Penetrating Brain Injury

Penetrating brain injuries (PBI) are fortunately rare occurrences among the civilian populations and can be the result of violence, accidents, or even suicide attempts (Gutiérrez-González, Boto, Rivero-Garvía, Pérez-Zamarrón, & Gómez, 2008).

Following the initial stabilization of the patient in regard to the trauma guidelines, the neurosurgical evaluation begins with conducting a clinical exam with signs of increased ICP documented prudently. CT scan is the initial imaging modality of choice, with vascular imaging included if there is a suspicion for arterial or venous injury. Surgical treatment is recommended within 12 h (Helling, McNabney, Whittaker, Schultz, & Watkins, 1992), especially in the context of a neurologic deficit or deterioration, with the goal toward the safe removal of the object, if at all possible, followed by appropriate antibiotic prophylaxis to improve outcomes. The risk of post-traumatic epilepsy after PBI is between 45% and 53%, and therefore, the use of prophylactic anticonvulsants is recommended (Raymont et al., 2010; Salazar et al., 1985).

Case Study: Penetrating Brain Injury

Clinical Presentation: A 42-year-old male walks into the emergency room after shooting himself with a nail gun in the head and chest. The patient did not have any neurologic deficits. The patient received prophylactic broad-spectrum antibiotics and a tetanus vaccine.

Diagnostic Imaging: CT head showed a foreign object within the frontal interhemispheric fissure (Fig. 2.3). Vascular imaging was obtained and did not show evidence of arterial or venous injury.

Management: The patient was taken emergently to the OR for a bi-frontal craniotomy with removal of the foreign body.

Clinical Course: The patient had an uneventful postoperative recovery in the intensive care unit. The patient was subsequently transferred to the psychiatric department for management.

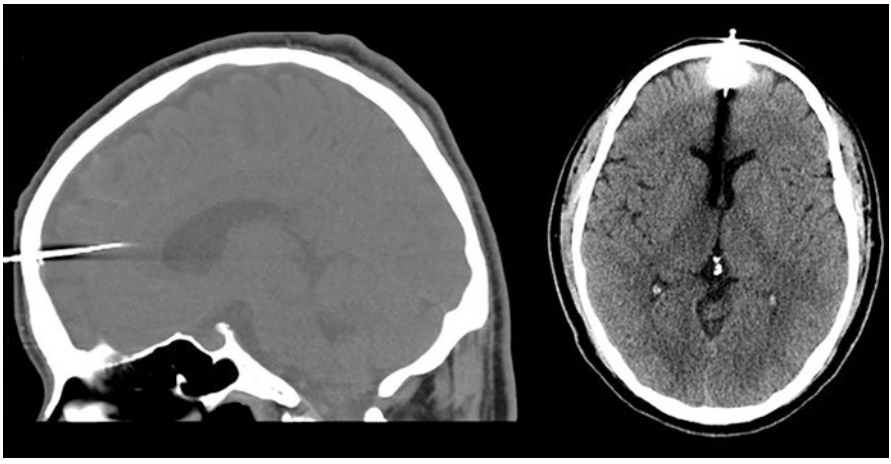

Fig. 2.3 Computed tomography scan of head, sagittal slice (left) and axial slice (right), demonstrating a left temporal subdural hematoma and parenchymal hemorrhage

Operative Nontraumatic Brain Injury

Spontaneous Intracranial Hemorrhage

Spontaneous nontraumatic intracerebral hemorrhage (ICH) is the second most prevalent subtype of stroke and is associated with high mortality and morbidity throughout the world (Kim & Bae, 2017). The pathogenesis of spontaneous ICH is diverse, including vascular disorders, amyloid angiopathy, tumor, vasculitis, hypertension, or reperfusion following a cerebral vascular accident. Initial workup entails a CT scan, with vascular imaging added on, especially in younger patients. Initial management of ICH is directed toward optimization of risk factors and control of ICPs. Surgical management of ICH remains controversial based on outcomes from randomized clinical trials (Broderick, 2005), but can be considered for superficial or cerebellar lesions, especially in the younger patient population (Alerhand & Lay, 2017). Results of a trial applying minimally invasive thrombolysis of spontaneous intracerebral hemorrhage showed no significant difference in outcome in 506 patients, but did suggest an advantage towards better outcomes in patients whose ICH was reduced to 15 ml in volume (Hanley, Thompson, Rosenblum, et al., 2019). Preliminary results for an endoscopic-guided, minimally invasive evacuation of basal ganglia ICH are promising, reducing in-house mortality (Goyal, Tzigoulis, Malhotra, et al., 2019).

Malignant Cerebral Infarction

Malignant cerebral infarction (MCI) is characterized by the compromise of the entire territory supplied by the middle cerebral artery (MCA) with accompanying mass effect resulting from acute brain swelling. Peak swelling and symptomatology usually occur within the first 48 h after stroke. MRI can be utilized to visualize acute infarcts on diffusion weighted imaging sequences. Initial management involves optimization of risk factors and control of ICPs. No definitive surgical guidelines exist, but the general recommendation is to perform a hemicraniectomy within 48 h if the patient is less than 60 years of age. There is an 80% mortality associated with MCI (Simard, Sahuquillo, Sheth, Kahle, & Walcott, 2011).

Brain Tumors: Meningiomas

Meningiomas are the most common primary brain tumor, with an incidence of 3–3.5 per 100,000 persons (Hoffman, Propp, & McCarthy, 2006). They tend to occur more commonly in patients with genetic predispositions, including neurofibromatosis type 2 or multiple endocrine neoplasia type 1 (Asgharian et al., 2004; Perry et al., 2001). The majority are histologically benign and asymptomatic and incidentally found on radiographic imaging (Chamoun, Krisht, & Couldwell, 2011).

Clinical presentation can include initial nonspecific symptoms (headache, nausea, altered mental status) with progression to focal neurologic deficits. Classical radiographic appearance is a well-circumscribed calcified contrast-enhancing extra-axial mass with vasogenic edema and dural attachment (Watts et al., 2014). In symptomatic cases or those with radiographic serial increases in tumor volume, patients are offered definitive surgical treatment with gross total resection (GTR) when feasible (Condra et al., 1997). Adjuvant therapy with postoperative radiation may be advised in cases of subtotal resection (STR). Prognosis is generally excellent in patients with meningiomas, with greater risks of progression with higher WHO grade or subtotal resection (Rogers et al., 2015).

Brain Tumors: Diffuse Gliomas

In the 2016 CNS WHO grading, all diffusely infiltrating gliomas (whether astrocytic or oligodendroglia) are now characterized collectively. This grading scheme is based not only on their histopathological behaviors but also on their genetic drivers (Louis et al., 2016). One of the most malignant tumors in the group, glioblastoma multiforme (GBM), tends to occur in patients aged 45–60 years of age, with an increased incidence in men. MRI with contrast tends to show GBM in the deep frontotemporal regions following white matter tracts, with invasion of the gray-white junction. Radiographically, they are represented by a central necrotic core with heterogeneous ring enhancement. Standard treatment involves initial gross total resection of the lesion, if possible, followed by chemotherapy and radiation. Prognosis of GBM remains bleak (8–18 month survival), but is better for tumors with specific genetic mutations, younger patients, and those with better preoperative Karnofsky performance scores (Nam & de Groot, 2017).

Case Study: Glioblastoma Multiforme

Clinical Presentation: A 59-year-old right-handed man with a recent history of altered mental status and left-sided paresis.

Diagnostic Imaging: MRI showed a large right parietal cystic lesion just below the cortical surface and a smaller frontal lesion and was most consistent with GBM (Fig. 2.4). The motor strip was slightly more anterior and superior to the most posterior lesion. PET scan of the body was negative for peripheral metastatic origins. Functional MRI showed the patient had left-sided hemispheric speech dominance.

Management: The patient underwent a right fronto-parietal craniotomy with resection of tumor. Given the proximity of the lesion to eloquent areas of the cortex, the case utilized functional cortical mapping. Intraoperative MRI was utilized to ascertain gross total resection.

Clinical Course: The patient's postoperative course was uneventful. The patient was seen by neuro-oncologist within initiation of chemo-radiotherapy 1 month following surgery. On 6-month follow up, MRI did not show evidence of reoccurrence.

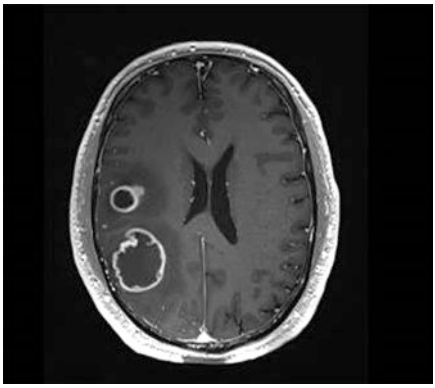

Fig. 2.4 Magnetic resonance image of head with contrast (axial slice), demonstrating right frontal and parietal enhancing cystic lesions

Spontaneous Subarachnoid Hemorrhage (Cerebral Aneurysm)

Spontaneous subarachnoid hemorrhage (SAH), usually secondary to a rupture of a cerebral aneurysm (85% cases), is fatal in >50% of cases. Initial imaging with CT imaging classically shows a stellate pattern where the hemorrhage collects around the basal cisterns, with additional hemorrhagic foci around the culprit vascular territory. Follow-up vascular imaging in the form of a CT angiography (CTA) or formal digital subtraction angiography (DSA) is required to visualize the external and internal cerebral arteries. Treatment is required immediately for ruptured aneurysms and can involve either open surgical or endovascular treatment as determined by the neurosurgeon in collaboration with a neuro-endovascular specialist. Of people with

Case Study: Subarachnoid Hemorrhage

Clinical Presentation: A 50-year-old male was at church when he developed the worst headache of his life. He presented to the emergency room with nausea and vomiting. He denied history of hypertension, smoking history, or family history of aneurysms. He had no focal deficits on neurologic exam.

Diagnostic Imaging: CTH demonstrated diffuse SAH with effacement of the basal cisterns (Fig. 2.5). Follow up CTA demonstrated a medially projecting right internal carotid artery (ICA) terminus aneurysm.

Management: The patient was taken emergently for a right craniotomy for clipping of ICA terminus aneurysm. Intraoperative angiography was utilized and demonstrated total obliteration of the aneurysm following surgical clipping.

Clinical Course: The patient's clinical course was complicated by symptomatic postoperative cerebral vasospasm on post-bleed day 8 which did not improve with maximal medical therapy. CTA demonstrated evidence of vasospasm of the right anterior cerebral artery (red arrow, Fig. 2.5, right). The patient was taken to the angiography suite and treated with intra-arterial verapamil, a calcium channel blocker, with improvement of symptoms. The patient was discharged on postoperative day 14 to a rehabilitation facility. On outpatient follow-up 1 month later, the patient remained neurologically intact.

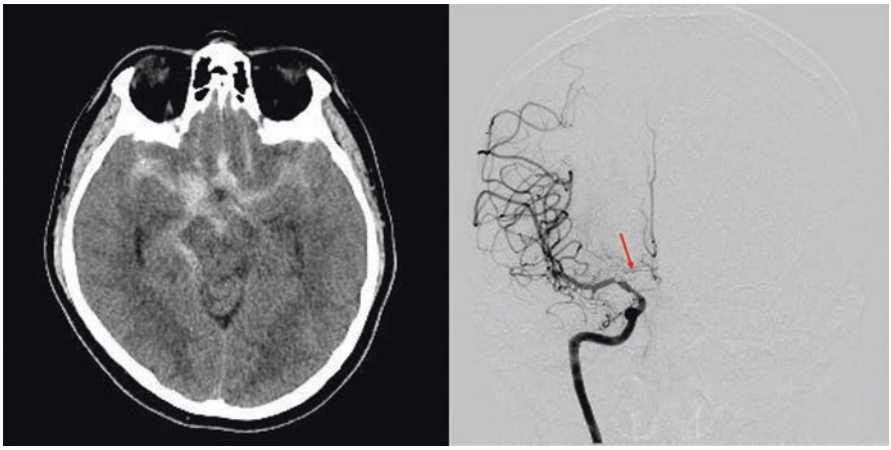

Fig. 2.5 Computed tomography scan of head (axial, left) demonstrating subarachnoid hemorrhage in basal cisterns with early hydrocephalus. Digital subtraction angiography of brain (anterior-posterior, right internal carotid injection, image right) demonstrating right anterior cerebral artery vasospasm

spontaneous SAH, 60% return to normal, 20% succumb to the disease, and 20% have severe permanent disability (Grasso, Alafaci, & Macdonald, 2017).

Conclusion

The causes of ABI, whether traumatic or not, are numerous and multifaceted. Treatment outcome relies upon the proper diagnosis of causes accountable for the symptomatology. Cautious deliberation must be given to somatic, neurological, neuropsychological, emotional, motivational, and social factors that contribute to a patient's pathology. Subsequent intervention should not only be directed toward the surgical treatment of the disease but also involve a multidisciplinary team to allow more cohesive participation. Improvements in patient outcomes will no doubt hinge not only on the acute perioperative care received but also on the long-term therapy provided to improve functional impairments. Education of the patient and family about realistic expectations and anticipated recovery over time is paramount.

References

Alerhand, S., & Lay, C. (2017). Spontaneous intracerebral hemorrhage. *Emergency Medicine Clinics of North America, 35*(4), 825–845. https://doi.org/10.1016/j.emc.2017.07.002. Review.

American Association of Neurological Surgeons. (2018). Retrieved June, 2018, from http://www.aans.org/Patients/Neurosurgical-Conditions-and-Treatments/Anatomy-of-the-Brain

Asgharian, B., Chen, Y. J., Patronas, N. J., Peghini, P. L., Reynolds, J. C., Vortmeyer, A., … Jensen, R. T. (2004). Meningiomas may be a component tumor of multiple endocrine neoplasia type 1. *Clinical Cancer Research, 10*, 869–880.

Bateman, D. (2001). Neurological assessment of coma. *Journal of Neurology, Neurosurgery, and Psychiatry, 71*(Suppl 1), i13–i17. https://doi.org/10.1136/jnnp.71.suppl_1.i13

Broderick, J. P. (2005). The STICH trial: What does it tell us and where do we go from here? *Stroke, 36*(7), 1619–1620.

Bullock M. R., Chesnut R, Ghajar J, Gordon D, Hartl R, Newell D. W., Servadei F,Walters B. C., Wilberger J. E. (2006). Surgical Management of Traumatic Brain Injury AuthorGroup. Surgical management of acute epidural hematomas. *Neurosurgery*. 58(3 Suppl):S7-15; discussion Si-iv. Review. PubMed PMID: 16710967.

Chamoun, R., Krisht, K. M., & Couldwell, W. T. (2011). Incidental meningiomas. *Neurosurgical Focus, 31*(6), E19.

Condra, K. S., Buatti, J. M., Mendenhall, W. M., Friedman, W. A., Marcus, R. B., Jr., & Rhoton, A. L. (1997). Benign meningiomas: Primary treatment selection affects survival. *International Journal of Radiation Oncology, Biology, Physics, 39*, 427–436.

Fraser, F., Matsuzawa, Y., Lee, Y. S. C., & Minen, M. (2017). Behavioral treatments for post-traumatic headache. *Current Pain and Headache Reports, 21*(5), 22. https://doi.org/10.1007/s11916-017-0624-x. Review.

Goyal N., Tsivgoulis G., Malhotra K., Katsanos A. H., Pandhi A., Alsherbini K. A. Chang J. J., Hoit D., Alexandrov A. V., Elijovich L., Fiorella D., Nickele C., Arthur A.S. (2019) Minimally invasive endoscopic hematoma evacuation vs best medical management forspontaneous basal-ganglia intracerebral hemorrhage. *Journal of NeurolnterventionaI Surgery, 11*(6), 579–583. https://doi.org/10.1136/neurintsurg-2018-014447.

Grasso, G., Alafaci, C., & Macdonald, R. L. (2017). Management of aneurysmal subarachnoid hemorrhage: State of the art and future perspectives. *Surgical Neurology International, 8*, 11. https://doi.org/10.4103/2152-7806.198738

Gutiérrez-González, R., Boto, G. R., Rivero-Garvía, M., Pérez-Zamarrón, A., & Gómez, G. (2008). Penetrating brain injury by drill bit. *Clinical Neurology and Neurosurgery, 110*, 207–210.

Hanley, D. F., Thompson, R. E., Rosenblum, M., et al. (2019). Efficacy of minimally invasive surgery with thrombolysis in intracerebral haemorrhage evacuation (MISTIE III): A randomized, controlled, open-label, blinded endpoint phase 3 trial. *Lancet*. https://doi.org/10.1016/S0140-6736(19)30195-3

Helling, T. S., McNabney, W. K., Whittaker, C. K., Schultz, C. C., & Watkins, M. (1992). The role of early surgical intervention in civilian gunshot wounds to the head. *The Journal of Trauma, 32*, 398–400.

Hoffman, S., Propp, J. M., & McCarthy, B. J. (2006). Temporal trends in incidence of primary brain tumors in the United States, 1985–1999. *Neuro-Oncology, 8*, 27–37.

Karibe, H., Hayashi, T., Hirano, T., Kameyama, M., Nakagawa, A., & Tominaga, T. (2014). Surgical management of traumatic acute subdural hematoma in adults: A review. *Neurologia Medico-Chirurgica (Tokyo), 54*(11), 887–894.

Kim, J. Y., & Bae, H.-J. (2017). Spontaneous intracerebral hemorrhage: Management. *Journal of Stroke, 19*(1), 28–39. https://doi.org/10.5853/jos.2016.01935

Kushner, D. (1998). Mild traumatic brain injury toward understanding manifestations and treatment. *Archives of Internal Medicine, 158*(15), 1617–1624. https://doi.org/10.1001/archinte.158.15.1617

Laker, S. R. (2011). Epidemiology of concussion and mild traumatic brain injury. *PM & R: The Journal of Injury, Function, and Rehabilitation, 3*(10 Suppl 2), S354–S358. https://doi.org/10.1016/j.pmrj.2011.07.017

Louis, D. N., Perry, A., Reifenberger, G., von Deimling, A., Figarella-Branger, D., Cavenee, W. K., … Ellison, D. W. (2016). The 2016 World Health Organization classification of tumors of the central nervous system: A summary. *Acta Neuropathologica, 131*(6), 803–820. https://doi.org/10.1007/s00401-016-1545-1

Nam, J. Y., & de Groot, J. F. (2017). Treatment of glioblastoma. *Journal of Oncology Practice/ American Society of Clinical Oncology, 13*(10), 629–638. https://doi.org/10.1200/JOP.2017.025536

Ontario Brain Injury Association. (2018). *What is acquired brain injury*. Retrieved January 10, 2018, from http://obia.ca/

Perry, A., Giannini, C., Raghavan, R., Scheithauer, B. W., Banerjee, R., Margraf, L., … Gutmann, D. H. (2001). Aggressive phenotypic and genotypic features in pediatric and NF2-associated meningiomas: A clinicopathologic study of 53 cases. *Journal of Neuropathology and Experimental Neurology, 60*, 994–1003.

Prins, M., Greco, T., Alexander, D., & Giza, C. C. (2013). The pathophysiology of traumatic brain injury at a glance. *Disease Models & Mechanisms, 6*, 1307–1315. https://doi.org/10.1242/dmm.011585

Raymont, V., Salazar, A. M., Lipsky, R., Goldman, D., Tasick, G., & Grafman, J. (2010). Correlates of posttraumatic epilepsy 35 years following combat brain injury (CME). *Neurology, 75*(3), 224–229. https://doi.org/10.1212/WNL.0b013e3181e8e6d0

Rogers, L., Barani, I., Chamberlain, M., Kaley, T. J., McDermott, M., Raizer, J., … Vogelbaum, M. A. (2015). Meningiomas: Knowledge base, treatment outcomes, and uncertainties. A RANO review. *Journal of Neurosurgery, 122*(1), 4–23. https://doi.org/10.3171/2014.7.JNS131644

Salazar, A. M., Jabbari, B., Vance, S. C., Grafman, J., Amin, D., & Dillon, J. D. (1985). Epilepsy after penetrating head injury, I. Clinical correlates: A report of the Vietnam Head Injury Study. *Neurology, 35*, 1406–1414.

Simard, J. M., Sahuquillo, J., Sheth, K. N., Kahle, K. T., & Walcott, B. P. (2011). Managing malignant cerebral infarction. *Current Treatment Options in Neurology, 13*(2), 217–229. https://doi.org/10.1007/s11940-010-0110-9

Tate, R., Kennedy, M., Ponsford, J., Douglas, J., Velikonja, D., Bayley, M., & Stergiou-Kita, M. (2014). INCOG recommendations for management of cognition following traumatic brain injury, Part III: Executive function and self-awareness. *The Journal of Head Trauma Rehabilitation, 29*(4), 338–352. https://doi.org/10.1097/HTR.0000000000000068

Watts, J., Box, G., Galvin, A., Brotchie, P., Trost, N., & Sutherland, T. (2014). Magnetic resonance imaging of meningiomas: A pictorial review. *Insights into Imaging, 5*(1), 113–122. https://doi.org/10.1007/s13244-013-0302-4

Williams, D. H., Levin, H. S., & Eisenberg, H. M. (1990). Mild head injury classification. *Neurosurgery, 27*, 422–428.

Chapter 3
The Role of the Neurologist in the Assessment and Management of Individuals with Acquired Brain Injury

Robert A. Duarte and Neisha Patel

Introduction

The specialist in neurology is trained to make a targeted diagnosis of specific ailments involving the brain, spinal cord, and peripheral nerves by obtaining a thorough history and a detailed neurological examination. Additionally, neurologists work with other neuro-rehabilitation specialists in setting up a proper rehabilitation program designed to maximize the patient's physical and neuro-cognitive recovery as well as provide the patient with tools to help cope with newfound deficits. Typical conditions that are evaluated and treated by a neurologist include traumatic brain injury (TBI), cerebrovascular accident (CVA), seizures, headaches, pain, and sleep disorders.

Role of the Neurologist

Neurologists usually become involved with patients suffering from an ABI in the emergency room setting. Following a TBI, the patient's overall neurological status has been traditionally assessed by using the Glasgow Coma Scale (GCS) (see Chap. 2). The GCS is a reliable and significant indicator of the severity of TBI and should be used repeatedly to identify improvement or deterioration over time. While GCS remains one of the most popular tools for the assessment of patients with TBI, it is by no means the only one. Additionally, the usefulness of GCS in patients who are intubated is limited because their verbal responses may not be assessed properly. Preferably the GCS should be measured prior to sedative administration or paralytic

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agents or after the drugs are metabolized. Wijdicks, Bamlet, Maramattom, Manno, and McClelland (2005) proposed a Full Outline of UnResponsiveness (FOUR) score, which evaluates patients on the basis of eye response, motor response, brainstem reflexes (pupillary, corneal, and cough), and respiratory pattern, thus avoiding the limitations of the GCS score when evaluating patients with severe TBI who are intubated and therefore unable to communicate verbally. GCS and FOUR scores are crucial to the initial assessment because they are well correlated with intracranial pathology and hence necessitate further investigation such as neuroimaging, looking for possible surgically correctable causes of depressed mental status. In addition to performing coma scales of choice, the neurologist must perform a detailed neurological examination, including but not limited to the assessment of higher cortical functioning, language, speech, spatial and temporal orientation, and signs of aphasia, apraxia, visual field cuts and other signs of hemispheric dysfunction.

The neurological examination is the primary ancillary tool of every neurologist. Skilled examination combined with a thorough history provides clues to diagnosis in a majority of cases; therefore, proper examination techniques and ability to interpret findings become of paramount importance. Neurological evaluation is typically performed in a traditional sequence beginning with a mini-mental state examination, followed by cranial nerve examination, motor function, sensory testing, deep tendon reflexes, and lastly coordination and gait (Table 3.1).

The most common bedside test for assessing cognitive function is the Folstein Mini-Mental State Examination (MMSE). MMSE is a brief screening tool that takes approximately 8 min to administer in individuals who are cognitively unimpaired and up to 15 min in patients with dementia. It assesses several cognitive domains, namely, orientation, memory, language, praxis, attention, and concentration. The MMSE yields scores ranging from 0 to 30. Though MMSE score is dependent on a patient's level of education, a score below 24 points has been the traditional cut-off for patients with cognitive impairment. This test has often been criticized for diagnostic validity in dementia, mild cognitive impairment, and delirium. MMSE does not perform well as a confirmatory tool for dementia, mild cognitive impairment, or delirium, but it does perform adequately in a rule-out capacity (Mitchell, 2017).

Also, this test has poor sensitivity to subtle changes, as many brain injury patients may have a normal MMSE but show significant cognitive impairments upon more detailed neuropsychological testing. Thus, good performance on the MMSE should be combined with testing (by a neuropsychologist) to ascertain a patient's ability to perform his or her pre-injury home, community, work, or school roles.

Table 3.1 Components of the neurological examination

Mini-mental state examination (MMSE)
Cranial nerves I–XII
Motor (tone, bulk, strength, abnormal movements)
DTRs (biceps, brachioradialis, triceps, patellar, and Achilles; Babinski reflex)
Sensory (touch, temperature, pain, vibration, proprioception)
Coordination and gait

Following the mental status examination, the nerves supplying the head and neck region must be evaluated. This portion of the examination is known as the cranial nerve (CN) examination. Cranial nerve I, commonly known as the olfactory nerve, is responsible for the sense of smell. Due to their location between the inferior frontal lobes and the base of the skull, olfactory nerves are often disrupted after a traumatic brain injury. A lesion within the olfactory pathway leads to alterations in sense of smell (parosmia) or total absence of smell (anosmia).

Cranial nerve II, optic nerve, carries visual information garnered in the retina by rods and cones to the lateral geniculate body, where neurons synapse and the optic pathway continues via temporal and parietal lobes to the occipital lobe. The function of the optic nerve is evaluated by testing visual acuity utilizing the Snellen chart, pupillary reaction to light, color recognition, and visual field testing. Lesions along the optic pathway may produce pupillary abnormalities, defects in visual fields, and color desaturation. Fundoscopic examination is performed with the examiner using an opthalmoscope. Abnormalities in the fundus known as papilledema can reflect elevations in intracranial pressure, often seen in cerebral structural abnormalities such as brain tumors.

Cranial nerves III (ophthalmic), IV (trochlear), and VI (abducens) are usually tested together, as they work collectively to provide full range of eye movements. Testing begins by observing eyes in a position of primary gaze while observing for proper ocular alignment and the presence of ptosis (droopy eyelid). Then, conjugate eye movement in six principal directions of gaze is observed by having the patient follow the target outlining the letter H. CN III palsy with unilateral eyelid drooping, dilated pupil, and an externally deviated eye can be seen in a unilateral hemispheric lesion, such as a stroke or tumor.

CN VI (abducens) palsy frequently occurs in the setting of increased intracranial pressure, particularly due to its long intracranial course. Brain trauma frequently produces trochlear nerve palsy, wherein the patient may have trouble looking down and will frequently complain of trouble walking down the stairs.

Cranial nerve V (trigeminal) supplies sensation to the face and controls the muscles of mastication. Its function is usually evaluated by using a wisp of cotton for fine touch, pin to test for pain, and cool tuning fork to test for temperature. Muscles of mastication are rarely affected in brain injury, unless facial injuries occur concomitantly.

Cranial nerve VII (facial) is responsible for the facial motor muscles and is evaluated by asking the patient to smile, show teeth, close the eyes tightly, and wrinkle the forehead. A lesion above the level of facial nerve nucleus located in the brainstem will spare the forehead, as it is bilaterally innervated. It is important to differentiate a lower motor nerve lesion from an upper motor nerve lesion of the facial nerve, with the latter implying hemispheric lesion and the former a lesion within the brainstem or periphery.

Cranial nerve VIII (auditory) is necessary for hearing. Since hearing pathways project bilaterally early on, hearing is rarely affected in brain injury unless there is a fracture of the internal auditory canal, thus damaging the nerve itself. CN VIII is grossly tested by the examiner rubbing his/her fingers together next to the patient's

ears, asking which stimulus the patient hears louder. The type of hearing disturbance can be further clarified using Weber and Rinne tests, which allow for distinction between sensorineural hearing loss and conductive hearing loss.

CN IX (glossopharyngeal) and CN X (vagus) are usually tested together, as they provide coordination in the swallowing process. The patient will be typically asked to open his/her mouth wide, protrude the tongue, and say "AAAAH," while the examiner observes for palatal movement. Gag reflex is tested by using a tongue depressor and touching the pharyngeal surface with a cotton swab, comparing side to side. While testing gag reflex is commonly taken to represent the function of the ninth and tenth nerves, the presence of gag does not provide any information about the patient's ability to swallow. Additionally, up to 20% of the normal healthy population may have a depressed or absent gag. The best-known means of evaluation of swallowing is the modified barium swallow or cine-esophagram, which allows the observation of movement of the food bolus during deglutition and swallowing. These are usually performed in conjunction with the speech/swallow therapist and gastroenterologist. Voice hoarseness in the absence of laryngeal process may be an indication of bulbar dysfunction. Alternately, swallowing difficulties and hoarseness could be caused by diffuse bilateral hemispheric dysfunction.

Cranial nerve XI (spinal accessory nerve) innervates the trapezius and sternocleidomastoid muscles and is usually tested by asking the patient to shrug his/her shoulders and to turn his/her head against resistance.

Cranial nerve XII (hypoglossal) is necessary for tongue movement and is tested by asking the patient to protrude the tongue and move it side to side. Mild head injuries (Glasgow Coma Scale score 14–15) can result in cranial nerve palsies with a similar distribution to moderate or severe brain injuries. The CNs associated with the highest incidence of palsy are olfactory, facial, and oculomotor nerves. The trigeminal and lower CNs are rarely damaged. Oculomotor nerve injury can have a good prognosis, with a greater chance of recovery if no lesion is demonstrated on the initial scan (Coello, Canals, Gonzalez, & Martin, 2010).

The motor examination usually consists of evaluating muscular bulk, tone, strength, and the presence of involuntary movements. The presence of muscle atrophy usually indicates either a primary muscle disorder or a peripheral denervating process. Atrophy may frequently coexist with muscle fasciculations. Muscle tone is the permanent state of partial contraction of a muscle and is assessed by passive movement. Increased tone can be divided into spasticity or rigidity both secondary to a brain or spinal cord injury known as an upper motor neuron lesion. Hypotonia is defined as decreased tone and may be seen in lower motor neuron lesions often seen in peripheral nerve injuries. Strength is typically graded on a scale from 0 to 5, where 0 signifies the absence of voluntary muscle contraction and 5 is full strength (Table 3.2). Patterns of muscle weakness can provide clues to lesion localization. For example, if weakness involves the face, arm, and leg equally, then the lesion is likely affecting corticospinal tracts in a deep subcortical location; if weakness is more severe in the face and arm rather than the leg, then the lesion is likely more cortical and superficial.

Table 3.2 Grading of muscle strength in neurological examination

0	No muscle contraction is detected
1	A trace contraction is noted in the muscle by palpating the muscle while the patient attempts to contract it
2	The patient is able to actively move the muscle when gravity is eliminated
3	The patient may move the muscle against gravity but not against resistance from the examiner
4	The patient may move the muscle group against some resistance from the examiner
5	The patient moves the muscle group and overcomes the resistance of the examiner; this is normal muscle strength

Sensory examination usually involves testing—touch, temperature, pain, vibration, and proprioception. Touch is usually tested by touching the patient on the face, testing all three divisions of the trigeminal nerve separately, and touching the patient on the extremities and asking the patient to compare sensation from side to side. Pain is usually tested by a disposable pin in a similar manner. Vibration is tested by using a 256-Hz tuning fork. Temperature is tested by using a cool tuning fork or a reflex hammer, in a similar manner. Proprioception is tested by isolating the patient's joint of interest, such as the distal phalangeal joints; asking the patient to close both eyes; and then, while holding the patient's finger on the sides, moving the finger up or down. The patient should be able to specify whether the finger is in the up or down position. If he/she has difficulty with small excursions, larger excursions should be attempted. If large excursions provide no clue to joint position, the examiner should move to a larger joint located more proximally, that is, wrist or elbow. Lower extremities may be tested in a similar manner. Additionally, Romberg's sign, which was previously thought to be significant for cerebellar dysfunction, actually tests proprioception (knowing where one is in space) in lower extremities. The patient is asked to stand with his/her feet together and eyes closed; instability and falling over in this position is considered a positive Romberg's sign and is revealing of diminished lower extremity proprioception often seen in a patient with syphilis or vitamin B12 deficiency.

The portion of the deep tendon reflex (DTR) examination includes the biceps, brachioradialis, and triceps involving the upper extremities and patellar and Achilles reflex in lower extremities. The presence of hyperactive DTRs in a weak extremity suggests corticospinal tract dysfunction, often seen in a stroke victim, whereas hypoactive DTRs are usually indicative of lower motor neuron dysfunction often seen in chronic diabetics with neuropathy. The Babinski reflex is tested by stroking the outer aspect of the sole from the heel toward the fifth digit on the foot; flexor response with downgoing toes is normal, and extensor response with upgoing toes is nonspecific but indicative of corticospinal tract dysfunction. The presence of brisk reflexes with associated extensor plantar response and/or clonus is abnormal and should be further investigated.

Coordination is primarily a function of the cerebellum and its connection to the cortex. It is usually tested by asking the patient to alternately touch his/her nose and the examiner's finger that moves within the patient's visual field. The patient with cerebellar dysfunction will exhibit dysmetria; that is, he/she will point beyond

the examiner's finger, or he/she will have marked oscillations on the way there. The lower extremity is usually tested by asking the patient to place the heel on the shin of the other leg and to slide the foot up and down the shin.

Stance is tested by asking the patient to stand with his/her eyes open and feet together. Then, the patient is asked to walk, with the examiner watching for circumduction of a lower extremity, which could be a sign of hemiparesis. Wide-based gait is a sign of cerebellar dysfunction. The patient should also be asked, if possible, to walk on the tiptoes and heels; this allows for detection of subtle gastrocnemius and tibialis anterior weakness, respectively. Finally, the patient should be asked to walk one foot in front of another, known as tandem gait.

Neurological Workup

A typical workup to evaluate for the presence of acquired brain injury involves an imaging study. Typically, computed tomography (CT) and magnetic resonance imaging (MRI) are utilized the most. CT scans are widely employed and available in nearly every emergency room. CT scans are fast and reliable and therefore remain a staple of the emergent neurological examination. Images in CT scans are acquired by means of thin X-ray beams rotating around examining part and detectors measuring the amount of radiation passing though. A computer analyzes these measurements, creating cross-sectional images of the area being scanned. By stacking these images—also known as "slices"—the computer can assemble three-dimensional models of the organs in a human body. Typically, a CT scan of brain without contrast material is the first neuro-imaging procedure utilized in the evaluation of a patient with a traumatic brain injury to rule out cerebral hemorrhage and identify possible skull fractures or bony lesions in the emergency setting. It is usually used to rule out cerebral hemorrhage and identify possible skull fractures in the setting of the emergency department. CT scan is superior to MRI in the evaluation of bony lesions and is just as good in the evaluation for the presence of blood. For these purposes, CT scans are typically obtained without intravenous contrast.

While a CT scanner is composed of X-ray generator, detector array, and processing unit, an MRI scanner consists of a large magnet, detector array, and processing unit. The MRI machine applies a radio-frequency pulse that is specific only to hydrogen. The system directs the pulse toward the area of the body being examined. Unlike CT scans, an MRI scan does not expose the patient to ionizing radiation and has a greater resolution for soft tissues. Often enough MRI scans require contrast; it is typically gadolinium-based and inert, and unlike CT contrast medium, which is usually iodinated, it is safe for kidneys and hypoallergenic.

However, MRI scanners have a few limitations, namely, MRI scans are contraindicated for someone with a pacemaker, old ferromagnetic aneurysm clips, or bullet fragments; the presence of extensive dental work, implants, or braces may introduce an artifact that will produce a poor quality image. Additionally, claustrophobic patients and those who cannot lie supine may experience difficulties in a scanner,

as the MRI examination will typically require the patient to stay still in a relatively closed space for 30–40 min at a time. Nonetheless, image quality obtained with an MRI is superior to that obtained with CT and therefore justifies its preference by most physicians and remains the gold standard in non-emergent evaluation of brain injury.

Additional testing modalities that are frequently employed by neurologists include transcranial and carotid Doppler ultrasound, which will be discussed in the "Stroke" section of this chapter, and electroencephalography (EEG), which is discussed in the "Epilepsy" section.

Seizures

Patient is a 49-year-old male who presented to the hospital after a motor vehicle accident at 40 miles/h, in which the patient was unrestrained and his head struck the windshield. On initial examination, the patient's GCS is 8 (best eye score 2/4, best verbal score 2/5, best motor score 4/6) Chap. 2 there is marked bruising of the forehead with multiple facial lacerations. During evaluation in the emergency room, the patient is observed to have a single generalized tonic–clonic seizure lasting 45 s, associated with tongue biting. CT scan of the head revealed frontal and occipital hemorrhagic contusions. Patient was loaded with intravenous phenytoin (Dilantin) and transferred to the intensive care unit for monitoring and neurological checks.

Seizures are a common complication of traumatic brain injury (TBI). A seizure is defined as a disturbance or disruption in the electrical activity of the brain, which results in uncontrollable changes to behavior, motor functions, or a change in sensory perception. The presence of intracranial pathology predisposes a patient to having seizures and consequently developing a seizure disorder. Epilepsy, as opposed to seizures, is usually defined as two or more unprovoked seizures on separate days, generally 24 h apart. An unprovoked seizure refers to a seizure that occurs in the absence of an acute brain insult or systemic disorder. Early seizures are thereby defined as acute symptomatic, but they are not representative of epilepsy, as seizures are provoked by the presence of an acute lesion. Post-traumatic epilepsy (PTE) refers to epilepsy that develops after TBI. Most investigators agree that PTE is to be distinguished from repeated seizures in the early stage following TBI, while the brain is acutely traumatized, inflamed, and metabolically disrupted. Therefore, a common set of definitions adopted by many researchers is the following: (1) immediate seizures usually defined as those occurring within 24 h after the injury, (2) early seizures which occur less than 1 week after the injury, and (3) late seizures which occur more than a week after the injury. Since the risk of recurrence after a single late post-traumatic seizure is over 70%, most investigators consider a single late post-traumatic seizure as being sufficient for the diagnosis of PTE. Although these are the most widely accepted definitions, there is controversy. Some narrow the definition of immediate seizures to those occurring at impact or within minutes

of the injury, classifying seizures occurring hours after trauma as early seizures. Others extend the time of early seizures to as long as 30 days after the injury (Ding, Gupta, & Diaz-Arrastia, 2016).

TBI is a significant risk factor for developing early seizures, as well as late epilepsy. Studies have shown that the presence of severe TBI increases one's chances of developing epilepsy as much as 74 times over the baseline rate at 2-year post-injury mark, with maximal risk of seizures in the first 7 days post-injury, followed by slow decline over a 5-year period. Risk factors for the development of post-traumatic epilepsy in the brain-injured patient highly correlate with severity of injury. A variety of classifications of TBI severity exist, but most investigators currently use the following schema: (1) mild TBI, characterized by loss of consciousness less than 30 min, post-traumatic amnesia less than 1 day, and normal neuroimaging; (2) moderate TBI, characterized by loss of consciousness more than 30 min and less than 24 h and post-traumatic amnesia between 1 and 7 days with or without abnormal neuroimaging; and (3) severe TBI, characterized by loss of consciousness greater than 24 h and post-traumatic amnesia more than 7 days, usually with abnormal neuroimaging such as contusion, cerebral hematoma, or extra-axial hematoma (Ding et al., 2016).

This nosology while robust and usually easily applied has many limitations, foremost among them the fact that it does not take into account findings from modern neuroimaging studies.

In 2003, the American Academy of Neurology (AAN) published guidelines on antiepileptic drug prophylaxis in acute severe TBI (Chang et al., 2003). It is recommended that patients with severe TBI be loaded with IV phenytoin (Dilantin) as soon as possible after the injury in order to prevent post-traumatic seizures within the first 7 days. Continued prophylactic treatment should not extend beyond 7 days (level B recommendation).

In addition to TBI, any other ABI also substantially increases the risk of developing epilepsy. Szaflarski et al. (2008) found that the overall incidence of acute seizures within the first 24 h of stroke was 3.1%. Kammersgaard and Olsen (2005) reported that PTE occurred in about 3% of all patients with stroke within 7 years after stroke. One meta-analysis showed that seizures occurred in about 6.93% of people with stroke. This variation in the incidence of early seizures, late seizures, and PTE reflects the differences in the patient populations, definition of the term for PSE, study design, diagnostic criteria, and the duration of follow-up.

This accounts for the increased prevalence of epilepsy in older adults, as cumulative brain lesion load increases with age. There have been a few studies on the cellular mechanisms underlying acquired epilepsy, and it has been shown that injury-induced alterations in intracellular calcium concentration levels and calcium homeostatic mechanisms play a role in the development and maintenance of acquired epilepsy by producing long-term neuroplasticity changes underlying epileptic phenotype (Delorenzo, Sun, & Deshpande, 2004).

The patient has not had any further seizures after the first episode; his mental status improved over the next week, and CT of the brain has reflected initial stages

of resolution. Phenytoin has been discontinued after 1 week and the patient has been successfully discharged to a rehabilitation facility. Seven months after the accident, the patient presented to the emergency room with another seizure that started as a rhythmic twitch of the arm, progressing into a generalized tonic–clonic seizure.

Epilepsy is classified according to the International League Against Epilepsy (ILAE) which has recently been updated by Ding et al. (2016). The definition of epilepsy has remained the same since 2004. The classification states that epilepsy and seizures are not the same. An epileptic seizure is a transient occurrence of signs and/or symptoms due to abnormal, excessive, or synchronous neuronal activity in the brain. Epilepsy is a disease characterized by an enduring predisposition to generate epileptic seizures and by the neurobiological, cognitive, psychological, and social consequences of this condition. This means a seizure is an event and epilepsy is a disease involving recurrent unprovoked seizures. The revisions provided by the ILAE focus on classifications of seizures. For decades, the most common words to describe seizures were grand mal and petit mal. Although the medical meaning of these terms was fairly precise, some used these terms loosely when referring to any big or little seizure. For over 35 years, the terms partial and generalized seizures were used to describe types of seizures. This system divided seizures into partial (starting in one area or side of the brain) and generalized (starting in both sides of the brain at the same time). Partial seizures were then defined as whether a person was aware or conscious during the seizure. In simple partial seizures, the person has awareness during the event, whereas in complex partial seizures, the patient may be confused, partially aware, or not aware at all. The old classification system worked for many years but didn't capture every type of seizure. The new basic seizure classification is based on three key features including where the seizure originates in the brain, the level of awareness during the seizure, and other features. The 2017 ILAE classifications of seizure types keep the framework of the basic classification but add more seizure types and subheadings. In the following image, the types of features under motor and non-motor seizures are listed for all times: focal, generalized, and unknown onset. Under focal onset, there is differentiation between awareness and impaired awareness and motor onset versus non-motor onset. Under generalized and unknown onset, the distinction is made between motor and non-motor such as absence seizures. Therefore, obtaining a thorough history of seizures, evoking a history of deja vu or jamais vu, olfactory sensations, intense fear, or nausea preceding a seizure is essential, as it would provide additional clues about localization of the epileptic lesion. The workup for a patient who has suffered a seizure episode includes blood tests, imaging studies, and electroencephalographic monitoring. Laboratory studies usually include a chemistry panel to evaluate for the presence of electrolyte and glycemic abnormalities.

Additionally, some epileptologists advocate obtaining a serum prolactin level after a seizure. Recent AAN guidelines on the use of prolactin in diagnosing epileptic seizures (Chen, So, & Fisher, 2005) suggest that elevation of prolactin within 20 min of the event can be used as an adjunct in distinguishing true seizures from a

nonepileptic event. Elevations in prolactin levels can also be seen in pregnancy, in cases of a prolactin-secreting tumor, and in patients on dopamine-blocking agents. A complete blood count should be performed since an elevation in the white blood cell count may point toward an underlying infection. If infection is indeed considered, a lumbar puncture may need to be performed to evaluate for the presence of encephalitis. Finally, both blood and urine toxicology screens should also be performed in order to rule out possible environmental or recreational drug use.

Routine electroencephalogram (EEG) is useful in the evaluation of a patient with seizures. This test is painless and usually takes a total of 60 min and is usually administered by a technician. The treating physician may request the patient to be sleep-deprived prior to the procedure, as it may provoke the appearance of epileptiform discharges. During an EEG, patients are typically asked to hyperventilate for a short period of time, and they may also be requested to look at a flashing strobe light. These maneuvers are known to elicit epileptiform discharges. Approximately 50% of people with epilepsy will have normal results on their first EEG; however, the sensitivity goes up to 90% after the third EEG (Binnie & Stefan, 1999). It is very rare that a seizure episode is captured during routine EEG, but prolonged video EEG monitoring in a medically supervised environment allows physicians to observe a patient's seizures directly. Observing seizure semiology (i.e., appearance) coupled with EEG correlate makes it possible to characterize ictal events and differentiate bona fide seizures from nonepileptic events and provides a longer time sample for capture of epileptiform discharges. Nonetheless, electroencephalographic studies are not useful in predicting the likelihood of post-traumatic seizures in any given patient.

The treatment of epilepsy is essential in maintaining a patient's health and lifestyle. Fortunately, the arsenal of a neurologist treating epilepsy has been significantly expanded over the last 15 years. In addition to standard drugs for epilepsy, that is, phenytoin (Dilantin), valproic acid (Depakote), carbamazepine (Tegretol), and phenobarbital (Luminal), there have been a substantial number of new drugs developed and tested over the years. According to the recent American Academy of Neurologists (AAN) guidelines on the efficacy and tolerability of newer drugs, several second-generation AEDs are effective for new-onset focal epilepsy. Data are lacking on the efficacy in new-onset generalized tonic–clonic seizures, juvenile myoclonic epilepsy, or juvenile absence epilepsy and on the efficacy of third-generation AEDs in new-onset epilepsy. Lamotrigine (LTG), levetiracetam, and zonisamide (ZNS) may be considered in decreasing seizure frequency in adults with new-onset focal epilepsy. LTG and gabapentin may be considered in decreasing seizure frequency in patients greater than 60 years of age with new-onset focal epilepsy. Unless there are compelling adverse effect–related concerns, ethosuximide or valproic acid should be considered before LTG to decrease seizure frequency in treating absence seizures in childhood absence epilepsy (level B). In general, individual choice of antiepileptic drug (AED) therapy should indeed be guided by patient-specific co-morbidities, that is, a patient with a prominent headache syndrome may benefit from topiramate, while patients with severe mood disturbances may benefit from mood-stabilizing properties of lamotrigine.

The patient was examined in the emergency room; routine laboratory studies as well as toxicology screen were negative. CT scan of the brain revealed the presence

of encephalomalacia in regions of prior trauma. EEG was obtained and revealed frontal slowing with rare epileptiform discharges. Though the patient did not formally meet the criteria for epilepsy, the decision was made to start the patient on antiepileptic drugs, as this seizure most likely represented a remote symptomatic seizure originating from the focus of encephalomalacia. Since the patient has suffered from concomitant mood disturbances, the decision was made to start lamotrigine, which is noted for its mood-stabilizing properties. The patient was instructed not to drive. He was able to tolerate the medication well and did not develop a rash; he is currently on 200 mg of lamotrigine twice a day maintenance and has not experienced any further seizures.

The question of whether a patient should be allowed the right to drive following a seizure remains an important issue, while the occurrence of a fatal motor vehicle accident due to a seizure is overestimated (Nguyen & Tellez Zenteno, 2009).

Legally, the rules regarding driving in seizure patients vary from state to state. Therefore, each practitioner should check in with the state's motor vehicles' authority regarding the proper procedure for reporting and counseling a patient. The neurologist should also gather input from the interdisciplinary team on the patient's attentional skills, processing abilities, and reaction time. In New York State, patients who have had a seizure or have been unconscious following an ABI must file an MV-80U.1 form with the Motor Vehicles Department in Albany to be cleared to return to driving. Occupational therapists (see Chap. 7) will often refer patients for a specialized driving evaluation to assess safety awareness and driving skills post-ABI. The patient suffering from epilepsy must be educated on the potential stressors that may precipitate a seizure. These include poor sleeping habits; alcohol or other recreational drug use; and exposure to environmental, dietary, physical, or emotional stressors. Patients should be advised never to be alone when bathing, swimming, or climbing heights. Family members need to be informed on how to manage their loved one who is having a seizure, with the emphasis on preventing a further brain injury. After a seizure, the patient should be turned to the side so as to allow any fluid in the oral cavity to drain, thus preventing aspiration. Most seizures are self-limited and last less than 5 min; however, emergency services should be contacted for any prolonged seizure. The risk of post-traumatic seizures decreases with time and reaches a normal value for the general population approximately 5 years after the brain injury. About half of the patients who develop late post-traumatic epilepsy have only three or fewer seizures and go into spontaneous remission thereafter. Nonetheless, a decision to stop antiepileptic agents should be made after careful consideration of risks versus benefits.

Stroke

The patient is a 68-year-old female with a past medical history of hypertension and diabetes who was found by her family members lying on the floor next to her bed and unable to talk or get up. The patient was rushed to the emergency room, where she was found to have dense right hemiparesis, face and arm more than leg, and

global aphasia. Since the time of onset was not known, the patient was excluded as a candidate for thrombolysis. Routine labs were within normal limits. CT of the head revealed the presence of a large hypodense area in the left fronto-temporal region. Since EKG revealed the new onset of atrial fibrillation, the patient was admitted to the telemetry service.

The impact of stroke on the healthcare system is staggering, according to the CDC, costing an estimated 34 billion dollars annually in both direct healthcare cost and lost income. Stroke is the leading cause of disability in adults, with 30% of stroke survivors requiring assistance with activities of daily living, 20% requiring assistance with ambulation, and 16% needing institutionalized care (Benjamin et al., 2017).

With industrialized nations' ever-increasing life expectancy, it is obvious that stroke is becoming one of the most important and most expensive public health hazards. Cerebrovascular disease is a multifactorial disorder, and the risk of a stroke could be greatly decreased by minimizing correctable factors, that is, tightened glycemic control, blood pressure medication, smoking cessation, and aggressive cholesterol-lowering treatments.

Most authorities agree upon stroke classification on the basis of vessel size ("small" vs. "large" vessel), mechanism of obstruction (thrombotic vs. embolic), or the presence of bleeding (ischemic vs. hemorrhagic). Small-vessel infarctions are representative of end-artery obstructions by microscopic cholesterol plaques within the lumen of the vessel. Large-vessel strokes occur due to obstruction of a main territory-supplying vessel such as the middle cerebral artery. In large vessels, obstruction is typically caused by progressive accumulation of intra-arterial thrombus or by embolization from a remote source, that is, the carotid artery or heart. Common impairments caused by stroke include motor weakness (77%), cognitive deficits (44%), dysphagia (45%), bladder/bowel dysfunction (48%) (Lawrence et al., 2001), visuo-spatial deficits (15%) (Linden, Samuelsson, Skoog, & Blomstrand, 2005), and dementia (28%) (Linden, Skoog, Fagerberg, Steen, & Blomstrand, 2004).

A typical diagnostic workup in a patient status post stroke involves CT or MRI of the brain in order to delineate the anatomy and observe the extent of the damage. If available, magnetic resonance angiography (MRA) of intracranial vessels is often advocated, as it allows determining the presence of intracranial vessel narrowing. While MRA is a static study of anatomical peculiarities, carotid Doppler ultrasonography allows for the evaluation of flow patterns within intracranial vessels and is considered useful in the evaluation of anterior circulation strokes. Transcranial Dopplers serve a similar purpose in posterior circulation strokes. A Holter monitor is useful in the detection of atrial fibrillation, as the heart is the most common site of embolism. Transesophageal echocardiogram (TEE) is the test of choice for the detection of left atrial thrombus. Low ejection fraction and valvular disease predispose to cerebrovascular disease as well. Laboratory studies usually include lipid profile, as hyperlipidemia is a correctable risk factor and hemoglobin A1C as a screen for hyperglycemic state. Workup of a cerebrovascular event in a young person without risk factors deserves additional testing for the presence of hypercoagulable state, evaluation for the presence of embolism from venous circulation to

arterial circulation through patent foramen ovale, and possible search for venous outflow obstruction.

Until 1995, acute stroke therapy consisted of controlling modifiable risk factors and managing immediate and remote consequences of acute stroke. Based on randomized controlled trials, the Federal and Drug Administration (FDA) approved the use of intravenous tissue plasminogen activator (tPA) for the treatment of acute ischemic stroke in June of 1996, thereby giving neurologists a tool to potentially reverse the neurological deficits secondary to an ischemic stroke. Other novel therapies include combined use of intra-arterial and intravenous tPA, intra-arterial tPA alone, clot retrieval devices, glycoprotein IIb/IIIa inhibitors, and neuroprotection through hypothermia.

While admitted, the patient had a complete neurological workup. MRI of the brain revealed a large left fronto-temporal lesion on diffusion-weighted imaging sequence, corresponding to the CT finding. MRA of the brain showed drop-off of the signal in the left middle cerebral artery, likely due to a clot obstructing the lumen. Carotid Doppler ultrasonography revealed 60% reduction of flow in the area of the left internal carotid artery. Transesophageal echocardiogram showed large left atrium without clots present. Patient was anticoagulated with coumadin and was discharged to the acute rehabilitation facility. Follow-up appointment with a vascular surgeon was made regarding carotid stenosis.

Secondary stroke prevention usually involves seeking out modifiable risk factors as well as placing a patient on an antiplatelet agent. According to the 2018 Guidelines for the Early Management of Patients with Acute Ischemic Stroke by the American Heart Association/American Stroke Association, a patient with acute ischemic stroke presenting within 48 h of symptom onset should be given aspirin (160–325 mg/day) to reduce stroke mortality and decrease morbidity. Among patients with acute TIA or minor ischemic stroke, starting aspirin/clopidogrel within 24 h of symptom onset reduces the 90-day stroke incidence without increasing bleeding rates, when compared to aspirin monotherapy, per the CHANCE trial. Ticagrelor is not recommended (over aspirin) in the acute treatment of patients with minor stroke.

Hypertension, diabetes mellitus, heart disease, dyslipidemia, smoking, carotid stenosis, and oral contraceptive use are some of the modifiable risk factors that can be addressed in conjunction with the patient's primary care physician. First-line cholesterol-lowering drug therapy, unless contraindicated, is moderate- to high-intensity statin. High-intensity statin therapy is recommended to reduce the risk of stroke among patients with ischemic stroke or TIA presumed to be of atherosclerotic origin and an LDL level $\geq$ 100 mg/dL with or without evidence for other ASCVD. For patients with ASCVD or diabetes mellitus, consideration should be given to the use of moderate- or high-intensity statin therapy, irrespective of baseline atherogenic cholesterol levels. Elevated blood pressure (BP) is the leading modifiable risk factor for stroke, and the benefit of BP-lowering therapy on the stroke risk reduction is well established. The optimal BP target for preventing stroke and other vascular events has been controversial, but the evidences from epidemiological studies and randomized controlled trials (RCTs) support intensive BP lowering for greater vascular protection, particularly for stroke prevention. For secondary

stroke prevention, the evidence of intensive BP-lowering benefit is limited since only a single RCT for patients with lacunar infarctions was conducted and most data were driven by exploratory analyses (Hong, 2017).

Most authorities agree that first-line hypertension treatment for secondary stroke prevention is the administration of angiotensin-converting enzyme (ACE) inhibitors, angiotensin receptor blockers, and thiazide diuretics. In cases where carotid stenosis is identified, the 2011 AHA/ACCF guidelines recommend stenting as a reasonable alternative to CEA among symptomatic patients with stenosis >70% and low risk of peri-procedural complications recommending that CEA might be preferred among older patients or those with anatomy not amenable to stenting. The guidelines suggest that prophylactic stenting for select asymptomatic patients with stenosis >70% by Doppler may be reasonable. It was an accepted standard of care to anticoagulate patients with nonvalvular atrial fibrillation with warfarin (Coumadin). Cochrane review of available literature in 2001 showed that, assuming a baseline risk for 45 strokes per 1000 patients, warfarin could prevent 30 incidences of stroke at the expense of 6 major bleeding episodes (Aguilar, Hart, & Hart, 2005). However more recent data, according to the ARISTOTLE trial, suggested that in patients with nonvalvular atrial fibrillation and with ≥1 risk factor, apixaban is associated with a greater reduction in rates of stroke or systemic embolism while having a lower rate of lower bleeding than warfarin (Granger et al., 2011).

The treatment of atrial fibrillation in a stroke patient requires a multidisciplinary approach and should be done in conjunction with an internal medicine practitioner as well as a cardiologist. Hyperbaric oxygen therapy (HBOT) has been evaluated as another potential avenue of treatment for patients with acute ischemic stroke. The primary purpose of HBOT is to increase the amount of salvageable tissue located within ischemic penumbra as well as to provide neuroprotection through a decrease in edema. Most recently in 2014 updated of the Cochrane review examining available data on utility of HBOT in acute ischemic stroke and in TBI (Bennett et al., 2014). It has been postulated that hyperbaric oxygen therapy (HBOT) may reduce the volume of the brain that will die by greatly increasing available oxygen, and it may further improve outcomes by reducing brain swelling. However, no good evidence to show that HBOT improves clinical outcomes when applied during acute presentation of ischemic stroke. Although evidence from the 11 RCTs is insufficient to provide clear guidelines for practice, the possibility of clinical benefit has not been excluded. Further research is required to better define the role of HBOT in this clinical setting (Bennett et al., 2014).

Secondary complications following a stroke may include increased intracranial pressure, systemic hypertension, seizures, hemorrhagic transformation, and obstructive hydrocephalus. According to the most current AHA/ASA guidelines for early management of patients with acute ischemic stroke, in patients with BP ≥220/120 mmHg who did not receive IV alteplase or EVT and have no comorbid conditions requiring acute antihypertensive treatment, the benefit of initiating or reinitiating the treatment of hypertension within the first 48–72 h is uncertain. It might be reasonable to lower BP by 15% during the first 24 h after onset of stroke.

Seizures in stroke can occur during both early and late stages. There is a wide discrepancy in reporting seizure incidence after stroke, much due to differences in time frames. Early seizures occur in 2–33% of patients suffering from acute stroke (Camilo & Goldstein, 2004); the presence of subarachnoid hemorrhage and cortical location were common predictive factors for the appearance of early seizures. It is obvious that seizures in stroke need to be addressed; however there is no clear answer regarding which medications are most effective for the management of post-stroke epilepsy, how long a patient should be treated, and whether prophylactic treatment of seizures is necessary. The Cochrane Collaboration is expected to publish a review of antiepileptic drugs for the primary and secondary prevention of seizures after stroke, which hopefully will shed light on this issue and provide treating neurologists with statistical underpinnings for their clinical decisions.

Encephalopathies

Encephalopathy is a term that literally means "disease of brain," referring to an altered mental state. The differential diagnosis of encephalopathy is diverse and encompasses pathological conditions from every category of disease and is one of the most common in-patient reasons for neurological consultation. Encephalopathy may be caused by an infectious agent, increased intracranial pressure, metabolic or mitochondrial dysfunction, exposure to toxins (including alcohol, drugs, radiation, industrial chemicals, and heavy metals), vitamin deficiencies, hormonal abnormalities, or hypoxia/hypoperfusion of the brain. Common neurological symptoms associated with encephalopathy are progressive loss of memory and cognitive ability, personality changes, difficulty with concentration, lethargy, and progressive loss of consciousness. The patient's neurological exam can be significant for the presence of myoclonus, nystagmus, asterixis, tremor, dementia, seizures, or any other lateralizing signs. Routine blood tests, cerebrospinal fluid (CSF) examination by performing a lumbar puncture, imaging studies, and electroencephalograms are a few examples of diagnostic studies that may be useful in the differentiation of various causes of encephalopathy. Herpetic encephalopathy is the most common, treatable cause of encephalopathy. Another example of encephalopathy is anoxic/hypoxic encephalopathy. Anoxic and hypoxic brain injuries are located on the same continuum; hypoxic injury is defined as relatively diminished oxygenation of the brain, and anoxic injury is defined as absent oxygenation of the brain. In contrast to focal hypoxemic injury, such as one sustained due to a stroke, anoxic/hypoxic injury usually happens due to systemic causes and therefore preferentially affects watershed areas of the brain. Specific lobes of the brain, the mesial temporal lobes, are particularly sensitive to hypoxia, thereby making short-term memory loss the most common complication of anoxic/hypoxic brain injury (Gibson, Pulsinelli, Blass, & Duffy, 1981). The degree of overall damage is highly dependent on the period of time that oxygenation was compromised. While occasionally the cause of anoxic/hypoxic brain injury is apparent or is discovered during initial assessment,

neurologists are usually called in to evaluate the patient. In this case, the role of the neurologist is not to diagnose the condition but to prognosticate meaningful recovery. A poor neurological outcome is defined as death from neurological cause, persistent vegetative state, or severe neurological disability. The most commonly used indicators of severe hypoxic ischemic brain injury include bilateral absence of corneal and pupillary reflexes, bilateral absence of N_2O waves of short-latency somatosensory evoked potentials, high blood concentrations of neuron specific enolase, unfavorable patterns on electroencephalogram, and signs of diffuse HIBI on computed tomography or magnetic resonance imaging of the brain. Current guidelines recommend performing prognostication not earlier than 72 h after return of spontaneous circulation in all comatose patients with an absent or extensor motor response to pain, after having excluded confounders such as residual sedation that may interfere with clinical examination (Sandroni, D'Arrigo, & Nolan, 2018).

Pain and Headache

Pain is a complex multidimensional subjective experience mediated by emotion, attitude, and perception. Pain in ABI can appear early and resolve, or may linger, thereby becoming chronic. Early pain due to injury reflects underlying bodily injury as affecting discrete neuroanatomical pathways. It is thought to confer evolutionary advantage by alerting the organism to the need for recovery. Chronic pain is thought to be a result of maladaptation of the central nervous system to the injury, as no clear cause–effect relationships between severity of injury and severity of chronic pain exist, no particular survival benefits are conferred, and muscular activity is avoided, thereby inhibiting successful recovery and restoration of function.

Central Pain Syndrome

The International Association for the Study of Pain has defined central pain caused by a lesion or dysfunction in the central nervous system. The lesion or disease process can occur anywhere along the neuraxis. Typical locations include the cerebral hemispheres, brainstem, and spinal cord. The clinician must have a certain degree of suspicion in order to make this diagnosis, especially if there is no identifiable lesion. In TBI, one may find an infarct or a hemorrhage in a patient with central pain syndrome. However, there appears to be no difference between hemorrhages and infarcts in regard to the tendency to induce central pain.

Typically, these patients describe a "neuropathic-type" pain—an unfamiliar, odd, dysesthetic (painful numbness), burning, lancinating sensation, with the skin often sensitive to simple touch; this phenomenon is known as allodynia. Central pain syndrome often begins shortly after the causative injury or damage but may be delayed by months or even years, especially if it is related to post-stroke pain.

This condition is often refractory to standard analgesic medications and requires a combination of pharmacological agents including antiepileptics or antidepressants to achieve adequate pain control. Opioids should only be considered as a last resort treatment and may require a pain management evaluation. In addition to pharmacological therapies, patients should be seen in a comprehensive pain management center utilizing the services of a pain psychologist. If above measures are not effective, there has been some success with surgical interventions, such as deep brain stimulation and ablation procedures.

Headache

The patient is a 34-year-old female with a prior history of migraines who presents with worsening headache after she tripped and fell on the ground, sustaining a scalp laceration. Patient thinks she might have momentarily lost consciousness. Patient describes her headaches as unilateral and throbbing, typical of her usual migraines which were previously easily controlled with ibuprofen. However, they have become increasingly more difficult to control, and patient reports that ibuprofen no longer relieves the headache. Since the incident happened 2 months ago, patient has missed 7 work days due to headaches, and she is anxious, as she is afraid to lose her job. Her neurological examination is significant for painful neck spasms.

Post-traumatic headache (PTHA) has been classified as a secondary headache disorder by the International Headache Society (Lipton, Bigal, Steiner, Silberstein, & Olesen, 2004). Examples of primary headache disorders, on the other hand, include migraine, tension-type, and cluster headaches. Most people who report having a headache following a TBI have a pre-existing primary headache disorder or an immediate family member relative with a primary headache. Post-concussion headache is the most common sequelum of brain trauma. In the acute stage of head and neck injury, headache prevalence is estimated to be 90%; this percentage point falls only to 44% at 6 months after the injury and may persist in up to 20% of patients 4 years later (Ramadan & Keidel, 2000). Women have a 1.9-fold increased risk of developing post-traumatic headache compared to men. This may be secondary to the higher incidence of primary headache disorders among women.

Studies, as summarized by Martelli, Grayson, and Zasler (1999), support the conclusion that the presence of post-concussion headache is generally negatively correlated with scores on neuropsychological testing. Decrements in information processing speed and complex attention are most frequently observed, while reductions in cognitive flexibility and verbal associative fluency, as well as learning and memory, appear to represent secondary findings that may be mediated by decreases in information processing and complex attention. Investigations of the effect of general, chronic pain on neuropsychological test results have produced similar results.

Therefore, the presence of chronic headache and pain is very likely to hinder recovery process from ABI. However, patients who sustain a severe brain injury with marked cognitive deficits may never truly complain of headaches until the

cognitive deficits disappear. Hickling, Blanchard, Silverman, and Schwarz (1992) found that 15 of 20 consecutive patients referred to a psychological practice for post-traumatic headache had post-traumatic stress disorder. Anxiety and depression further contribute to the development of PTHA. The most frequently seen headaches following traumatic brain injury resemble a tension-type headache, characterized by a bilateral gripping, nonthrobbing sensation not typically associated with nausea, vomiting, or marked light or sound sensitivity; migraine headaches described by a unilateral throbbing sensation associated with nausea and/or vomiting or photo- and phonophobia usually interfering with an activity; occipital neuralgia-type headache frequently following a whiplash injury where the occipital nerve is irritated. These headaches are characterized by a unilateral headache originating in the cervical region extending to the forehead often described as a shooting, lancinating sensation triggered by neck movement. In addition, these may be accompanied by paresthesias (numbness) or dysesthesias. Less commonly, one may present with headaches triggered by marked position change known as low-pressure headaches or headaches secondary to intracranial hypotension. In these patients, the headaches are characteristically triggered by sitting up or standing and dramatically relieved upon lying down. This could result from a cerebrospinal fluid leak through a dural sleeve tear or a cribriform plate fracture. Myofascial pain disorders are often under-recognized. These usually involve the masseter, trapezius, or temporalis muscles and may occur following a traumatic brain injury. Workup of headaches in clients with ABI should include an imaging study of the brain and cervical spine, especially in patients with a TBI, as the presence of acute hemorrhage needs to be excluded. For this particular purpose, a CT scan is sufficient and is preferred over an MRI. Additionally, an X-ray or CT of the c-spine to rule out a cervical fracture should be performed. In the subacute or chronic stage, an MRI of the brain is the preferred test, as it provides a better, higher-resolution image of intracranial structures without subjecting patients to radiation. If the headaches persist or there is an apparent cervicogenic component, an MRI of the cervical spine should be considered. An electroencephalogram (EEG) is generally not recommended in any patient presenting with headache unless there is a strong suspicion of a seizure disorder. Blood testing is generally not useful in post-traumatic setting although obtaining a complete count to rule out anemia and a thyroid panel to rule out hypothyroidism may be helpful.

Patient has been referred for MRI of the brain, which was reportedly normal. As her headaches were now occurring more often than four times per month, a decision to start prophylactic treatment along with acute migraine management was made. Patient was started on nortriptyline (Pamelor) nightly, as well as sumatriptan (Imitrex) as needed for acute headaches, which she was able to tolerate well. She was additionally referred to physical therapy for the management of neck spasm. Patient reported improvement in her headaches over the subsequent 2 months.

The treatment of acute headache following trauma depends upon the underlying pathogenesis, that is, hemorrhage requiring immediate surgical intervention (see Chap. 2). There is no FDA-approved agent for the treatment of post-concussion headache. The management of post-concussion headaches requires a multidimen-

sional approach, combining pharmacotherapeutics with psychotherapy, physiotherapy, and relaxation techniques. The choice of medication should be guided by the characteristics of the headache. If the headache appears clinically to be migraine as per the International Headache Society criteria, then anti-migraine medications should be employed. As a general rule, if the patient's episodic headaches are less than 4 days per month, an abortive agent such as a triptan, ergotamine, or specific non-steroidal anti-inflammatory medication such as diclofenac powder (Cambia) should be employed, whereas if the headaches are more frequent (four or more headaches per month), a preventive therapy should be considered. Whether to consider prophylactic therapy for patients presenting with migraine headaches following trauma should be dealt with on an individual basis depending on severity, interference with activity, and duration (weeks to months) of the headache. The anticonvulsants sodium valproate (Depakote) and topiramate (Topamax) and the beta-blocker Inderal (propranolol) are FDA-approved for the prevention of episodic migraines. However, one must be aware of the potential side effects of these agents. Topiramate can cause cognitive slowing and memory difficulties which may prove to be quite problematic for the post-concussion/post-traumatic headache sufferer. Somnolence, tremor, weight gain, hair loss, and potential neural tube defects in the fetus are the few well-known side effects of sodium valproate; therefore caution must be exercised when prescribing these drugs in patients with TBI. These agents may also prove to be helpful in post-traumatic headache patients with an associated mood disorder, due to their mood-stabilizing properties. The beta-blockers, nadolol (Corgard) and propranolol (Inderal), can also be considered as a preventive agent for headache treatment. These agents are known to affect mood negatively and are therefore contraindicated in patients susceptible to depression. The choice of a preventive therapy is frequently influenced by associated co-morbidities, such as depression and insomnia. There are other non-FDA-approved agents that have been given a Level A classification due their overwhelming positive evidence for preventing migraines, such as amitriptyline (Elavil). Depressed patients who have insomnia may be appropriate candidates for tricyclic antidepressants such as amitriptyline (Elavil) or its secondary amine, nortriptyline (Pamelor). Unfortunately, these agents have many potential side effects which may interfere with recovery in a patient with brain injury, such as orthostatic hypotension, sedation, and cognitive slowing. Therefore, judicial use of these agents is advocated. Botulinum toxin (BOTOX) injections, FDA-approved for the prevention of chronic migraines has also been employed and can be potentially beneficial especially in cognitively impaired post-trauma headache patients. Newer migraine therapies, CGRP antagonists, may show promise in the post-concussion headache population. Headaches with a cervicogenic component may respond to physical therapy modalities, including myofascial techniques with or without trigger point injection therapy, relaxation strategies such as biofeedback/meditation, acupuncture, and local occipital nerve blocks. Electromyography (EMG)-guided biofeedback has been shown in the past to be beneficial in PTHA treatment when combined with cognitive behavioral therapy (CBT) and pharmacotherapeutics (Onorato & Tsushima, 1983). However, no strong evidence from clinical trials is available to direct the treatment of PTHA. Some guidelines are

offered for PTHA management based on primary headache categories and treatments (Watanabe, Bell, Walker, & Schomer, 2012).

In their retrospective biofeedback outcome study, Ham and Packard (1996) reported that the greatest treatment effects were obtained for general relaxation and for ability to cope with pain.

There are very few randomized clinical trials evaluating the efficacy of non-pharmacological interventions. Therefore, future research, which considers the noted biopsychosocial factors, is needed in the field to determine if these interventions reduce PTHA (Fraser, Matsuzawa, Lee, & Minen, 2017).

While never tested for PTHA, cognitive–behavioral therapy, relaxation treatment, and hypnosis were all shown to be of use in the treatment of chronic headaches (Holroyd & Andrasik, 1978; Tobin, Holroyd, Baker, Reynolds, & Holm, 1998). Acupuncture is an appealing alternative treatment for headache sufferers; however, its usefulness has not been measured until recently. Cochrane review recently suggested that existing evidence supports the use of acupuncture in idiopathic headache, although the quality of evidence is not fully convincing (Melchart et al., 2001). Most recently, the Journal of the American Medical Association (JAMA) published a randomized controlled trial of acupuncture in migraine sufferers, which compared acupuncture versus sham acupuncture versus waiting list in migraine sufferers. Results have shown that while acupuncture was significantly better in reducing headaches as compared to no intervention, it was not better than sham acupuncture, that is, placement of needles in nontherapeutic points (Linde et al., 2005). Similar results were observed in the study of acupuncture in tension-type headache, where the acupuncture group performed better than the no acupuncture group but similar to the group that received minimal acupuncture (Melchart et al., 2005). A more recent multicenter single-blinded randomized controlled trial suggested that both traditional acupuncture and sham acupuncture had effects on relieving pain and, traditional acupuncture was slightly better than sham acupuncture (Wang et al., 2012).

Further studies are necessary in order to determine if indeed there is a role of acupuncture in chronic headache treatment, particularly in PTHA. Additionally, dietary discretion, particularly avoidance of cheese, monosodium glutamate, caffeine, chocolate, and wine, is also recommended in chronic headache sufferers, as tyramine heightens sympathetic arousal.

Sleep Disorder

Sleep disturbance is common following traumatic brain injury (TBI), affecting 30–70% of individuals, many occurring after mild injuries. Insomnia, fatigue, and sleepiness are the most frequent post-TBI sleep complaints with narcolepsy (with or without cataplexy), sleep apnea (obstructive and/or central), periodic limb movement disorder, and parasomnias occurring less commonly. In addition, depression, anxiety, and pain are common TBI co-morbidities with substantial influence on sleep quality (Viola-Saltzman & Watson, 2012).

Additionally, it is well known that excessive somnolence is a major cause of motor vehicle accidents, resulting in 36% of highway fatalities and up to 54% of collisions. In a recent small-scale study of ten patients with TBI and sleep complaints, all were found to have treatable sleep disorders. Seven patients were suffering from sleep disorders on the obstructive spectrum, two had narcolepsy, and only one had post-traumatic hypersomnia (Castriotta & Lai, 2001). It has also been found that patients with obstructive sleep apnea may have significant impairment in daytime functioning, intellectual capacity, memory, and motor coordination, which could be exacerbating cognitive deficits sustained during ABI. Since the presence of sleep disorder itself appears to be significantly correlated with ABI, and the majority of sleep disorders are treatable, it is imperative that patients suffering from sleep disorders be identified and treated as they potentially present a hazard to themselves and public health. The Epworth Sleepiness Scale (Johns, 1991) still remains to be one of the most popular means of screening patients with daytime sleepiness for the presence of potential sleep disorder and can be performed by a general practitioner. Patients whose test results are suggestive of excessive daytime sleepiness should be evaluated by a neurologist or pulmonologist skilled in sleep evaluation. Further diagnostic workup usually involves polysomnography and multiple sleep latency tests. Patients diagnosed with obstructive sleep apnea should undergo titration of nasal airway pressure in the sleep laboratory, in order to determine the optimal pressure that prevents episodes of apnea, oxygen desaturation, and snoring. While severe TBI tends to be associated with disorders of hypersomnia, mild to moderate TBI is frequently associated with insomnia, which is a perception that sleep quality is inadequate or nonrestorative despite the adequate opportunity to sleep. Additionally, as patients recover from TBI, hypersomnia pattern appears to change to insomnia pattern. Curiously, it appeared that patients with severe TBI had fewer sleep complaints than patients with mild to moderate TBI, which could be related to the lack of awareness of limitation due to cognitive impairment. Additionally, patients with mild TBI are expected to resume their responsibilities much sooner than patients with moderate or severe TBI and are rarely afforded the time and resources necessary in order to fully reintegrate themselves into their daily routine. This appears to cause additional stress, which in turn triggers further insomnia. The treatment of sleep disorders in ABI depends on the nature of the disorder. Hypersomnia during the acute stage of TBI can be successfully treated using modafinil (Provigil) and methylphenidate (Ritalin). Modafinil is a medication that is commonly used to treat excessive daytime somnolence associated with narcolepsy. The precise mechanism of action is not completely understood, since it appears to be nonreactive with any major class of receptors. However, modafinil has been found to inhibit GABA release in the basal ganglia (Smith, 2003). Given that hypersomnolence is a common complaint among TBI patients, and current experience with modafinil also suggests its usefulness for the treatment of fatigue, this medication has become popular in the arsenal of pharmacological agents to augment functional recovery (see Chap. 6). Methylphenidate is used as a stimulant for excessive sleepiness and as a treatment of attention-deficit/hyperactivity disorder (ADHD). Frontal lobes are frequently damaged as a result of TBI; they are also implicated in ADHD. Both of those conditions have sleep

disturbance as a common comorbidity, which suggests that sleep disorder is closely related to frontal lobe functioning. A study by Flanagan, Kane, and Rhoades (2003) demonstrated that the use of methylphenidate has a beneficial effect on processing speed, distractibility, vigilance, and sustained attention in patients with TBI. Walker-Batson et al. (2001) suggested that dextroamphetamine administration results in a significant improvement in language skills in a group of patients with stroke-induced aphasia when paired with speech language therapy as compared to controls. Additionally, Cochrane review of amphetamines for improving recovery after stroke shows that there are trends toward benefit in language and motor recovery in stroke patients who were given amphetamines; however, further studies are required in order to statistically confirm its usefulness (Martinsson, Wahlgren, & Hardemark, 2003). Therefore, methylphenidate would be an acceptable choice of treating hypersomnolence in a patient with ABI and could potentially exert beneficial effects on other aspects of patient's functioning. The treatment of insomnia in ABI is much more involved because of comorbidity of insomnia with other psychiatric conditions, particularly depression. Therefore, the approach to the patient with insomnia should be very similar to the approach to a patient with pain disorder. It requires pharmacotherapeutic intervention coupled with appropriate rehabilitation techniques, proper counseling, and maintenance of sleep hygiene. Keeping a sleep log for 2–4 weeks helps to establish a pattern of insomnia, further clarifying which approach would work best. The current arsenal of medications active against insomnia includes eszopiclone (Lunesta), zaleplon (Sonata), and zolpidem (Ambien). As nonbenzodiazepine receptor–active medications, they offer an alternative to the use of short-acting benzodiazepine receptor agonists, which are commonly used in the treatment of insomnia but contraindicated in clients with ABI. Meta-analyses showed that zaleplon and eszopiclone were safe choices in the treatment of insomnia in the elderly, with improved profile of effect on psychomotor and cognitive performance (Glass, Lanctot, Herrmann, Sproule, & Busto, 2005). Cognitive-behavioral therapy, progressive relaxation, guided imagery, and biofeedback are useful as well (Smith, Huang, & Manber, 2005).

Conclusion

In conclusion, the neurologist plays an integral role in the evaluation and management of patients with acquired brain injury. The primary role of the neurologist is to make an accurate diagnosis or, at least, confirm the diagnosis and assess the extent of injury by obtaining a comprehensive history and performing a detailed neurological examination. A neurological treatment plan is then formulated to help prevent further neurological sequelae and promote restoration of function. As an educator and advisor, the neurologist has a unique opportunity to discuss prognosis and recommend possible nonsurgical as well as surgical alternatives to patients and their families. Lastly, a key role of the neurologist is to interface with the multidisciplinary brain injury team focusing on the primary goal of facilitating maximum recovery.

References

Aguilar, M., Hart, R., & Hart, R. M. (2005). Antiplatelet therapy for preventing stroke in patients with non-valvular atrial fibrillation and no previous history of stroke or transient ischemic attacks. *Cochrane Database of Systematic Reviews*, (4), CD001925.

Benjamin, E. J., Blaha, M. J., Chiuve, S. E., Cushman, M., Das, S. R., Deo, R., … Muntner, P. (2017). Heart disease and stroke statistics. American Heart Association Statistics Committee and Stroke Statistics Subcommittee. *Circulation, 135*, e146–e603.

Bennett, M. H., Weibel, S., Wasiak, J., Schnabel, A., French, C., & Kranke, P. (2014). Hyperbaric oxygen for acute ischaemic stroke. *Cochrane Database of Systematic Reviews*, (11), CD004954.

Binnie, C. D., & Stefan, H. (1999). Modern electroencephalography: Its role in epilepsy management (Review). *Clinical Neurophysiology, 110*(10), 1671–1697.

Camilo, O., & Goldstein, L. B. (2004). Seizures and epilepsy after ischemic stroke. *Stroke, 35*(7), 1769–1775.

Castriotta, R. J., & Lai, J. M. (2001). Sleep disorders associated with traumatic brain injury. *Archives of Physical Medicine and Rehabilitation, 82*(10), 1403–1406.

Chang, B. S., Lowenstein, D. H., & Quality Standards Subcommittee of the American Academy of Neurology. (2003). Practice parameter: Antiepileptic drug prophylaxis in severe traumatic brain injury: Report of the Quality Standards Subcommittee of the American Academy of Neurology. *Neurology, 60*(1), 10–16.

Chen, D. K., So, Y. T., & Fisher, R. S. (2005). Use of serum prolactin in diagnosing epileptic seizures: Report of the Therapeutics and Technology Assessment Subcommittee of the American Academy of Neurology (Review). *Neurology, 65*(5), 668–675.

Coello, A. F., Canals, A. G., Gonzalez, J. M., & Martin, J. J. (2010). Cranial nerve injury after minor head trauma. *Journal of Neurosurgery, 113*(3), 547–555.

Delorenzo, R. J., Sun, D. A., & Deshpande, L. S. (2004). Cellular mechanisms underlying acquired epilepsy: The calcium hypothesis of the induction and maintenance of epilepsy. *Pharmacology and Therapeutics, 105*(3), 229–266.

Ding, K., Gupta, P. K., & Diaz-Arrastia, R. (2016). Epilepsy after traumatic brain injury. In D. Laskowitz & G. Grant (Eds.), *Translational research in traumatic brain injury*. Boca Raton, FL: CRC Press/Taylor and Francis Group.

Flanagan, S. R., Kane, L., & Rhoades, D. (2003). Pharmacological modification of recovery following brain injury. *Journal of Neurologic Physical Therapy, 27*, 129–136.

Fraser, F., Matsuzawa, Y., Lee, Y. S. C., & Minen, M. (2017). Behavioral treatments for post-traumatic headache. *Current Pain and Headache Reports, 21*(5), 22.

Gibson, G. E., Pulsinelli, W., Blass, J. P., & Duffy, T. E. (1981). Brain dysfunction in mild to moderate hypoxia. *American Journal of Medicine, 70*(6), 1247–1254.

Glass, J., Lanctot, K. L., Herrmann, N., Sproule, B. A., & Busto, U. E. (2005). Sedative hypnotics in older people with insomnia: Meta-analysis of risks and benefits. *British Medical Journal, 331*(7526), 1162.

Granger, C. B., Alexander, J. H., McMurray, J. J., Lopes, R. D., Hylek, E. M., Hanna, M., … Wallentin, L. (2011). Apixaban versus warfarin in patients with atrial fibrillation. *N Engl J Med, 365*(11), 981–992.

Ham, L. P., & Packard, R. C. (1996). A retrospective, follow-up study of biofeedback-assisted relaxation therapy in patients with post-traumatic headache. *Biofeedback and Self-Regulation, 21*, 93–104.

Hickling, E. J., Blanchard, E. B., Silverman, D. J., & Schwarz, S. P. (1992). Motor vehicle accidents, headaches and post-traumatic stress disorder: Assessment findings in a consecutive series. *Headache, 32*(3), 147–151.

Holroyd, K. A., & Andrasik, F. (1978). Coping and the self-control of chronic tension headache. *Journal of Consulting and Clinical Psychology, 5*, 1036–1045.

Hong, K.-S. (2017). Blood pressure management for stroke prevention and in acute stroke. *Journal of Stroke, 19*(2), 152–165.

Johns, M. W. (1991). A new method for measuring daytime sleepiness: The Epworth sleepiness scale. *Sleep, 14*(6), 540–545.

Kammersgaard, L. P., & Olsen, T. S. (2005). Post stroke epilepsy in the Copenhagan stroke study: Incidence and predictors. *Journal of Stroke and Cerebrovascular Diseases, 14*(5), 210–214.

Lawrence, E. S., Coshall, C., Dundas, R., Stewart, J., Rudd, A. G., Howard, R., & Wolfe, C. D. (2001). Estimates of the prevalence of acute stroke impairments and disability in a multiethnic population. *Stroke, 32*(6), 1279–1284.

Linde, K., Streng, A., Jurgens, S., Hoppe, A., Brinkhaus, B., Witt, C., … Melchart, D. (2005). Acupuncture for patients with migraine: A randomized controlled trial. *Journal of American Medical Association, 293*(17), 2118–2125.

Linden, T., Samuelsson, H., Skoog, I., & Blomstrand, C. (2005). Visual neglect and cognitive impairment in elderly patients late after stroke. *Acta Neurologica Scandinavica, 111*(3), 163–168.

Linden, T., Skoog, I., Fagerberg, B., Steen, B., & Blomstrand, C. (2004). Cognitive impairment and dementia 20 months after stroke. *Neuroepidemiology, 23*(1–2), 45–52.

Lipton, R. B., Bigal, M. E., Steiner, T. J., Silberstein, S. D., & Olesen, J. (2004). Classification of primary headaches. *Neurology, 63*(3), 427–435.

Martelli, M. F., Grayson, R. L., & Zasler, N. D. (1999). Posttraumatic headache: Neuropsychological and psychological effects and treatment implications. *Journal of Head Trauma Rehabilitation, 14*(1), 49–69.

Martinsson, L., Wahlgren, N. G., & Hardemark, H. G. (2003). Amphetamines for improving recovery after stroke (Review). *Cochrane Database of Systematic Reviews*, (3), CD002090.

Melchart, D., Linde, K., Fischer, P., Berman, B., White, A., Vickers, A., & Allais, G. (2001). Acupuncture for idiopathic headache. *Cochrane Database of Systematic Reviews*, (1), CD001218.

Melchart, D., Streng, A., Hoppe, A., Brinkhaus, B., Witt, C., Wagenpfeil, S., … Linde, K. (2005). Acupuncture in patients with tension-type headache: Randomised controlled trial. *British Medical Journal, 331*(7513), 376–382.

Mitchell, A. J. (2017). The Mini-Mental State Examination (MMSE): Update on its diagnostic accuracy and clinical utility for cognitive disorders. In A. J. Larner (Ed.), *Cognitive screening instruments* (pp. 15–47). New York, NY: Springer.

Nguyen, R., & Tellez Zenteno, J. F. (2009). Injuries in epilepsy: A review of its prevalence, risk factors, type of injuries and prevention. *Neurology International, 1*(1), e20.

Onorato, V. A., & Tsushima, W. T. (1983). EMG, MMPI, and treatment outcome in the biofeedback therapy of tension headache and posttraumatic pain. *American Journal of Clinical Biofeedback, 6*, 71–81.

Ramadan, N. H., & Keidel, M. (2000). The headaches. In J. Olesen, P. Tfelt-Hansen, & P. Welch (Eds.), *The headaches* (2nd ed., pp. 771–780). Philadelphia, PA: Lippincott Williams & Wilkins.

Sandroni, C., D'Arrigo, S., & Nolan, J. P. (2018). Prognostication after cardiac arrest. *Critical Care, 22*(1), 150.

Smith, B. W. (2003). Modafinil for treatment of cognitive side effects of antiepileptic drugs in a patient with seizures and stroke. *Epilepsy and Behavior, 4*, 352–353.

Smith, M. T., Huang, M. I., & Manber, R. (2005). Cognitive behavior therapy for chronic insomnia occurring within the context of medical and psychiatric disorders. *Clinical Psychology Review, 25*(5), 59–92.

Szaflarski, J. P., Rackley, A. Y., Kleindorfer, D. O., Khoury, J., Woo, D., Miller, R., … Kissela, B. M. (2008). Incidence of seizures in the acute phase of stroke: A population-based study. *Epilepsia, 49*(6), 974–981.

Tobin, D. L., Holroyd, K. A., Baker, A., Reynolds, R. V. C., & Holm, J. E. (1998). Development in clinical trial of a minimal contact, cognitive–behavioral treatment for tension headache. *Cognitive Therapy and Research, 12*, 325–339.

Viola-Saltzman, M., & Watson, N. (2012). Traumatic brain injury and sleep disorders. *Neurologic Clinics, 30*(4), 1299–1312.

Walker-Batson, D., Curtis, S., Natarajan, R., Ford, J., Dronkers, N., Salmeron, E., … Unwin, D. H. (2001). A double-blind, placebo controlled study of the use of amphetamine in the treatment of aphasia. *Stroke, 32*, 2093–2098.

Wang, L. P., Zhang, X. Z., Guo, J., Liu, H. L., Zhang, Y., Liu, C. Z., … Li, S. S. (2012). Efficacy of acupuncture for acute migraine attack: A multicenter single blinded, randomized controlled trial. *Pain Medicine, 13*(5), 623–630.

Watanabe, T. K., Bell, R. B., Walker, W. C., & Schomer, K. (2012). Systemic review of interventions for post-traumatic headache. *PM&R, 4*, 129–140.

Wijdicks, E. F., Bamlet, W. R., Maramattom, B. V., Manno, E. M., & McClelland, R. L. (2005). New coma scale: The FOUR score. *Annals of Neurology, 58*, 585–593.

Chapter 4
Physiatry and Acquired Brain Injury

Sarah Khan, Komal Patel, and Gonzalo Vazquez-Cascals

Introduction

Physiatrists are specialists who focus not only on the disease process or injury that limits the patient's functioning but also on the secondary effects that may occur as a result of the disease process or injury. We utilize a biopsychosocial model that is unlike conventional medicine, which tends to focus on the diagnosis and treatment specifically geared toward the disease process (biomedical model) (Stiens, O'Young, & Young, 2002). The underlying principle is based on treating each patient as a "whole." Rehabilitation medicine takes physical, emotional, and social needs into account when formulating a treatment plan. The physiatrist utilizes therapeutic exercises and physical agents in addition to medications to treat patients. The role of the physiatrist is to restore a patient's overall quality of life. Our emphasis is on maximizing a patient's functional capabilities.

Role of the Physiatrist

The Americans with Disabilities Act defines a person with a disability as "a person who has a physical or mental impairment that substantially limits one or more major life activity" (ADA National Network, n.d.). It is the physiatrist's role to identify a

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patient's physical and cognitive deficits as well as the functional impact of these deficits in order to better highlight a patient's impairment, activity limitations, and participation restrictions.

The World Health Organization International Classification of Functioning (ICF) defines impairment as "a problem in body function or structure." Activity limitation is "a difficulty encountered by an individual in executing a task or action." Participation restriction is "a problem experienced by an individual in involvement in life situations." By identifying these three components of functional assessment, the physiatrist can construct a treatment plan that can help to minimize the impact of the impairments in day-to-day life (Health Topics, 2018) (Table 4.1).

This holistic way of treating patients requires an interdisciplinary approach. The multifaceted nature of the clinical consequences of acquired brain injuries makes the interdisciplinary team approach the most appropriate strategy for treatment (Roth & Harvey, 2000). Interdisciplinary team meetings allow the members of the rehabilitation team to establish goals in order to provide patients with a unified, coordinated treatment plan. The team will determine the most appropriate disposition, home versus an alternative living facility, establish a discharge date, and identify services the patient may need upon discharge. Good communication skills among the members of the team are required to construct a rehabilitation program that is individualized for each patient. The overall goal of comprehensive rehabilitation is to optimize a patient's quality of life and achieve maximal independent functioning.

In an outpatient setting, the physiatrist assesses patients for physical, occupational, and speech therapy needs to address functional deficits, provide appropriate referrals, and follow their progress. Patients with ABI are also assessed for visual deficits that occur as a result of their injury and are referred to a neuro-optometrist or neuro-ophthalmologist for further treatment. The physiatrist also determines if patients with ABI will benefit from pharmacological treatment for cognitive or behavioral or psychological symptoms. Referrals to neuropsychology for cognitive assessment and treatment as well as counseling for psychological symptoms are made as determined appropriate. The physiatrist is also often involved in determining when a patient has recovered adequately enough to return to work or driving. Often these decisions are made with input from members of the rehabilitation multidisciplinary team and, when necessary, other physicians. It is not uncommon for

Table 4.1 Examples of ICF classification for ABI

Impairment	Activity limitation	Participation restriction
Paralysis or weakness of lower extremities	Inability to or difficulty with ambulation	Unable to go shopping or to work
Paralysis or weakness of the upper extremity	Inability to manipulate objects	Unable to dress oneself or drive
Dysphagia	Inability or difficulty chewing and/or swallowing	Unable to enjoy social events like going to a restaurant or holiday dinners
Aphasia	Impairment of expression	Unable to call for help in an emergency

patients recovering from ABI to be referred to behind the wheel driving assessment or vocational training to achieve these goals.

Rehabilitation of the Patient with Acquired Brain Injury

Acquired brain injury (ABI) consists of a diverse set of conditions involving particular mechanisms of injury as well as consequences including physical, cognitive, behavioral, and emotional deficits. Such complex constellation of symptoms and deficits requires by necessity a comprehensive treatment approach. While many different approaches to intervention are reported in the scientific literature, a significant question remains whether interdisciplinary rehabilitation for brain injury can be considered an effective and cost-efficient method to help ABI patients to achieve their maximum recovery potential (Salazar, et al., 2000).

Systematic review of the effectiveness of multidisciplinary rehabilitation for adults with TBI (Turner-Stokes, Pick, Nair, Disler, & Wade, 2015) found that:

1. A majority of patients with mild TBI make a good recovery.
2. Most patients with PTA < 1 h, not admitted to hospital, do not need specific interventions.
3. Patients with PTA ≥ 1 h receive benefit from routine follow-up contact to obtain information and advice.
4. Some patients with moderate to severe injury benefit from a more intensive level of intervention and this may not be reachable unless routine follow-up is provided.

There is a limited number of studies assessing the effect of community-based rehabilitation programs in people with TBI that do suggest improvement in certain measures of functioning (Bowen, Tennant, Neumann, & Chamberlain, 2001; Powell, Heslin, & Greenwood, 2002). One high-quality randomized control trial found a significant improvement on measures of community integration, perceived quality of life, and self-efficacy (Cicerone et al., 2008). There has been more attention in the literature studying the effect of inpatient rehabilitation services for individuals with TBI. In general, these have shown earlier and improved functional outcomes and significantly decreased caregiver stress (Semlyen, Summers, & Barnes, 1998; Zhu, Poon, Chan, & Chan, 2007) and shorter lengths of stay (Shiel et al., 2001; Slade, Tennant, & Chamberlain, 2002).

Systematic review of the effectiveness of stroke rehabilitation (Turner-Stokes et al., 2015) found that there is moderate evidence that outpatient therapy improves outcomes of stroke rehabilitation.

Neuroplasticity

Recovery after acquired brain injury can occur spontaneously or through neuroplasticity. Natural spontaneous neurological recovery may lead to a decrease in the extent of neurological impairment via resolution of local edema, resorption of local toxins, improvement of local circulation, and recovery of partially damaged ischemic neurons (Roth & Harvey, 2000). Neuroplasticity is a concept that refers to the ability of the central nervous system (CNS) to modify its structural and functional organization to restore severed paths and/or create new neuronal paths to substitute those damaged in order to return the organism to a previous state of equilibrium. It is influenced by the environment and stimulation, repetition of tasks, and motivation. Neuroplasticity involves neuronal regeneration or collateral sprouting and the unmasking of previously latent functional pathways. Following cerebral injury, surviving neurons retain the ability to form new synapses.

Neuroplasticity can be experience-independent, experience-expectant, or experience-dependent. Groups of neurons that naturally tend to activate together in patterns in time form connections that are considered experience-independent plasticity. Experience-expectant plasticity occurs in early developmental learning and often occurs during certain "critical periods." An example of this is how a toddler's brain is optimally primed for language development, compared to learning a new language as an adult. Experience-dependent plasticity occurs throughout one's lifetime. Synaptic neural connections are established based on environmental experiences (Kolb, Harkar, & Gibb, 2017). After brain injury, a comprehensive rehabilitation program is important to promote recovery using activities and exercises that stimulate motor and cognitive learning by modifying dendritic connections within a neural network. Negative influences to the brain, such as major stress or alcohol, often diminish synaptic connectivity and impair neurorecovery (Kolb & Gibb, 2014).

Brain-derived neurotrophic factor (BDNF) is a molecular protein that plays an important role in neuroplasticity. According to Cotman and Bertold, "exercise induces the expression of genes associated with plasticity, such as that encoding (BDNF), and in addition promotes brain vascularization, neurogenesis, functional changes in neuronal structure and neuronal resistance to injury." Studies have shown that aerobic exercise increases BDNF in a number of brain regions, including the hippocampus, cerebellum, cerebral cortex, and spinal cord, and improves learning and memory (Cotman & Berchtold, 2002). Performance of aerobic exercise shortly before motor training has been shown to improve motor learning. Cognitive performance has also been demonstrated to benefit from aerobic exercise, particularly in executive functioning tasks (Colcombe & Kramer, 2003). This improvement is believed to be modulated by the increase in BDNF stimulated by aerobic exercise. There is some evidence that resistance exercise also increases BDNF and therefore promotes improvement in cognition and motor learning. Based on a literature review of neuroplasticity and cognition, it is recommended that an exercise program to enhance neuroplasticity should include a combination of both aerobic and

resistance exercises, at least 30 min of aerobic exercise at 70% maximum heart rate four times weekly (Mang, Campbell, Ross, & Boyd, 2013).

The function of BDNF may be influenced by genetic factors. About one-third to half the population have a single nucleotide polymorphism where the amino acid valine is replaced by methionine at position 66 on the BDNF gene (Shimizu, Hashimoto, & Iyo, 2004). There is limited and conflicting evidence in the literature regarding if people with this genetic morphology have slower or worse neurological recovery outcomes (Shimizu et al., 2004), and this requires further investigation.

Predictors of Outcome

With the growing rates of acquired brain injury, outcome prognostication in these individuals plays a crucial role in their recovery. Prognosis of outcome is an important tool in assessing the individual's eventual quality of life, healthcare practices, as well as clinical research. Much of what is known to this date about prognosis of individuals with brain injury are related to their Glasgow Coma Scale (GCS), length of post-traumatic amnesia (PTA), length of coma, age, presence of extracranial injuries, presence of pupil reactivity, and imaging findings. Post-traumatic amnesia is defined as the period of delirium after brain injury where there is an inability to encode and retain new information and experiences. Given all these different features to help predict prognosis and outcome, none of them should be taken as a single consideration; rather a multivariate method should be utilized in order to have a better understanding of a patient's recovery potential after a brain injury.

Currently, brain injuries are classified as mild, moderate, or severe based on a patient's initial GCS score (see Chap. 2). The motor component is the best predictor of functional recovery (Healey et al., 2003; Teasdale et al., 2014). The GCS was first described by Teasdale and Jennett in 1974 to assess an individual's level of consciousness. It contains three independently evaluated patient behaviors—eye opening, verbal response, and motor response (Teasdale et al., 2014). The GCS score is one of the most commonly used indicators of severity of brain injury with scores ranging from 3 to 15. The lower a patients' GCS, the more severe their brain injury is and hence a potentially worse outcome (Teasdale et al., 2014). Furthermore, the CRASH trial published in 2008 showed an increase in early mortality as a patient's GCS decreased from 14 to 4 (MRC CRASH Trial Collaborators 2008). A correlation was also noted between a patient's GCS and reduction in metabolic rate in the cortical gray matter (Wu, Huang, Hattori, et al., 2004) thalamus, brainstem, and cerebellum at various times after a brain injury (Hattori et al., 2003). With the GCS providing some understanding into an individual's degree of brain injury based on the measurement of a patient's level of consciousness, the length of a patient's coma as well as PTA plays a role in helping to determine the severity and outcome after a brain injury.

Coma is defined as the presence of three features: an individual's eyes being closed, the lack of a sleep/wake cycle, and no spontaneous purposeful movements.

A longer duration of coma is associated with a poorer outcome. If coma lasts less than 2 weeks, then a severe disability is unlikely; however if coma lasts greater than 4 weeks, then a good recovery is unlikely. Additionally, the length of PTA can help to evaluate how a patient with a brain injury will recover. PTA is defined as the duration of time from loss of consciousness to return of continuous memory for day-to-day events. Ritchie Russell, in 1932, first presented the duration of PTA as an indicator of severity of brain injury and of subsequent duration of disability (Furbringer e Silva & de Sousa, 2007). Nakase-Richardson et al. in (2011) then further reviewed duration of PTA and severity of brain injury in these individuals, examining approximately 4000 patients with brain injuries. Based on their research, they classified those with PTA for 0–14 days to have moderate TBIs, 15–28 days having moderately severe TBIs, and those with 29–70 days having severe TBIs. Outcomes have also been shown to be poorer in those with PTA for greater than 13 days (McMillan, Jongen, & Greenwood, 1996). An association between lower GCS and a longer duration of PTA has also been noted (McMillan et al., 1996), bringing together these two factors in helping to determine patient's prognosis and recovery course (Table 4.2).

Another important component which is an independent predictor of outcome is an individual's age, in which the risk of poor outcome following a brain injury increases with age (Gomez et al., 2000; Hukkelhoven et al., 2003; Luerssen, Klauber, & Marshall, 1988; Tsyben et al., 2017).

It is difficult to say at which specific age the risk of poor outcome significantly changes, but it has been shown that patients older than the age of 40 not only have worse functional outcome at any severity of brain injury but also significantly longer PTA as well (Katz & Alexander, 1994; Wu et al., 2004). Additionally, a progressive increase in adverse outcome rates in those with brain injuries has been noted to start at age 35, whereas patients under the age of 18 have significantly better outcomes (Berger, Pitts, Lovely, Edwards, & Bartkowski, 1985; Gomez et al., 2000).

Moreover, physical exam and imaging findings also serve as a tool to predict a patient's outcome. On head computed tomography (CT) scans, the Marshall CT classification system is considered class I evidence in terms of prognosis. Signs of raised intracranial pressure, such as the obliteration of the third ventricle, midline shift, compression/effacement of basal cisterns, as well as the presence of intraventricular blood and traumatic subarachnoid hemorrhage on imaging were shown to

Table 4.2 Classification of severity of brain injury based on GCS and length of PTA

GCS	Length of PTA	Severity of TBI
–	<5 min	Very mild
13–15	5–60 min	Mild
9–12	1–24 h	Moderate
3–8	1–7 days	Severe
–	1–4 weeks	Very severe
–	>4 weeks	Extremely severe

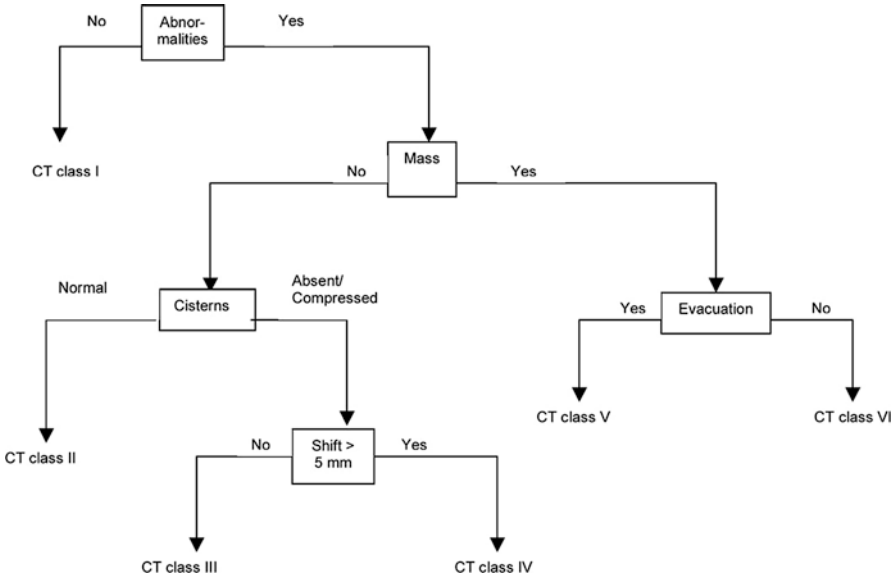

Fig. 4.1 Marshall CT classification (Maas et al., 2005)

be strong predictors of mortality (Gomez et al., 2000; Maas, Hukkelhoven, Marshall, & Steyerberg, 2005; MRC CRASH Trial Collaborators et al., 2008). Also, individuals with abnormal pupillary responses were found to be at increased risk for adverse outcomes (Gomez et al., 2000). Furthermore, it has also been shown that the presence of a non-evacuated hematoma has a strong predictor of unfavorable outcome at 6 months (MRC CRASH Trial Collaborators et al., 2008) (Fig. 4.1).

Given that trying to determine a patient's prognosis, especially early on in the course, can be difficult, there are many factors that are taken into consideration. These help to guide clinical course and treatment plans. As patients progress in their recovery, there are several assessment tools that can be used to help monitor their level of arousal, orientation, and level of function.

Assessment Scales

As mentioned previously, the GCS helps to determine the level of consciousness for a person, but there are additional tools that will help to determine an individual's recovery from coma, such as the JFK coma recovery scale (CRS). Both of these tools are used as diagnostic assessments for outcome prediction, predicting disposition needs, treatment planning, and monitoring effectiveness of treatments. However, the CRS was developed in order to provide a more standardized approach to assessing a patient's level of consciousness as compared to the GCS. The GCS tends to be

more relevant in the acute period after brain injury, and the CRS tends to be more useful in the subacute period or rehabilitation phase of brain injury.

Initially described in 1991, the CRS characterizes patient functioning in more detail, including 25 hierarchically arranged items that include six subscales monitoring auditory, visual, motor, oromotor, communication, and arousal processes (Giacino, Kalmar, & Whyte, 2004). The grading of the CRS is such that the lowest scores indicate reflexive responses, whereas the highest scores represent cognitively mediated processes. Additionally, studies evaluating the CRS have shown some utilization in outcome prognostication. For instance, a group of patients in vegetative state were shown to have a higher recovery of consciousness within the first 12 months post-injury if they visually tracked as compared to those individuals who did not track. Furthermore, CRS score change done during the initial 4 weeks of inpatient rehabilitation has a stronger correlation with functional outcome at 1 year than GCS score change (Giacino et al., 2004).

As a patient transitions out of minimally conscious state, orientation to the surrounding environment and situation becomes of crucial importance as it is usually the initial step in cognitive rehabilitation (Novack, Dowler, Bush, Glen, & Schneider, 2000). Scales used to evaluate and analyze a patient's orientation are the Galveston Orientation Assessment Tool (GOAT) and the Orientation Log (O-Log) and the Westmead Post-Traumatic Amnesia Scale. The GOAT, which was first published in 1979, aimed to prospectively evaluate cognition serially, measuring orientation to person, place, time, and memory of events preceding and following the injury and eventually establishing the duration of PTA (Levin, O'Donnell, & Grossman, 1979). The GOAT consists of a ten-item questionnaire, with scores ranging from 0 to 100. In order for a patient to be considered out of PTA, the individuals must score greater than 75 on 2 consecutive days at least 24 h apart (Furbringer e Silva & de Sousa, 2007; Novack et al., 2000). The GOAT is a useful tool to measure orientation but can be sometimes difficult to administer and limited in patients that are non-verbal.

Given these limitations, the O-log was developed as a brief measure of orientation that is easy to administer to rehabilitation populations as an alternative to the GOAT (Novack et al., 2000). It also has the advantage of being able to be used in patients with speech impairments. It consists of ten items related to orientation to place, time, and situation, with each item being scored based on the patient's response and a maximum total of 30 points (Novack et al., 2000) (Table 4.3). The cutoff for the end of PTA as determined by the O-log has been correlated to the

Table 4.3 Scoring of O-log based on patient response and amount of cueing required

Response	O-log score
Correct spontaneous without cueing response	3
Correct response upon logical cueing	2
Correct response upon multiple choice or phonemic cueing	1
Incorrect response despite multiple cueing	0

GOAT, and a score greater than or equal to 25 has been shown to have the best sensitivity (Novack et al., 2000).

The Westmead Post-Traumatic Amnesia Scale, developed in the 1980s, was originally an extension of the Oxford Scale. It not only examined orientation to person, place, and time but also investigates an individual's ability to consistently recall new information from one day to the next. It consists of 12 questions and is administered once a day. PTA is considered to be cleared the first day an individual scores a perfect score on 3 consecutive days (Tate et al., 2006).

Assessment and Management of Spasticity

Spasticity is defined as "a motor disorder characterized by a velocity-dependent increase in tonic stretch reflexes with exaggerated tendon jerks, resulting from hyperexcitability of the stretch reflex, as one component of the upper motor neuron syndrome" (Lance, 1980).

Spasticity is commonly seen in patients with acquired brain injury, spinal cord injury, multiple sclerosis, and cerebral palsy. It is clinically assessed by passively ranging a patient's limb and measuring the increase in resistance of the muscle or muscle tone at that joint. The Modified Ashworth Scale (Table 4.4) is the most common clinical scale used to grade spasticity. Common complications secondary to spasticity include contractures, heterotopic ossification, pressure ulcers, and respiratory infections.

When considering treatment for spasticity, it is important to determine if the spasticity is causing functional impairment. This should be done by taking a thorough history from the patient and/or caregiver regarding the patient's limitations in daily living and setting realistic functional goals. The clinician should also observe the patient performing functional activities, including gait and synergy patterns of movement. Functional goals of treatment are to improve hygiene, decrease pain and spasms, decrease deformity, improve orthotic fit, improve gait, decrease energy

Table 4.4 Modified Ashworth Scale

Grade on Modified Ashworth Scale	Definition
0	No increase in resistance
1	Slight increase in resistance, catch at the very end of range of motion (ROM)
1+	Catch followed by resistance felt in less than half of the ROM
2	Resistance felt throughout most of the ROM, but the limb is easily moved
3	Difficult to passively range the limb, often unable to complete full ROM
4	Rigid, unable to range the limb at all

expenditure of gait, and facilitate motor control (Boake, Francisco, Ivanhoe, & Kothari, 2000). For some patients, their spasticity may improve their overall functional ability. For example, extensor spasticity in the lower extremities may stabilize a patient's limbs and help them stand or ambulate.

The first step in treating spasticity is to avoid exposure to noxious stimuli (such as pressure ulcers, urinary tract infections, constipation, ingrown toenails). Non-pharmacological treatments include proper positioning, stretching, range of motion, modalities, serial casting, and dynamic splinting. These methods are primarily implemented by occupational and physical therapists. Stretching, positioning, and range of motion are basic and essential components of the treatment process.

Stretching involves elongation of the muscle. In addition to manual stretching, splinting is another option to apply sustained stretch to a spastic limb. Dynamic splints are devices that can apply an active, active assistive, or passive stretch to a muscle. Saebo splints, like the SaeboFlex, are custom fabricated and utilize springs and pulleys providing a dynamic stretch. Dynasplints provide adjustable, continuous low-tension, long-duration stretch to any limb joint in the body. Lycra garments have fabric that are sewn in particular directions to provide sustained stretch when worn (Shingleton, Kinzinger, & Elovic, 2011). Serial casting involves placing casts on the spastic limb to increase joint range of motion progressively. The skin should be monitored when placing and removing splints or serial casts. Skin irritation or breakdown can be a noxious stimulus and worsen the spasticity. Serial casting can be applied between 1 and 7 days. Once there is no improvement in range of motion after two sequential cast periods, serial casting should be stopped (Shingleton et al., 2011).

Cryotherapy, which refers to cold and hot applications, has been shown to decrease muscle stretch reflex excitability and increase range of motion (Katz, Dewald, & Schmit, 2000). The application of cold modalities has been theorized to improve spasticity by slowing nerve conduction, desensitization of muscle spindle activity and cutaneous receptors, and altering central nervous system excitability (Shingleton et al., 2011). Other modalities that can be used are biofeedback and electrical stimulation. Functional electrical stimulation and neuromuscular stimulation have been shown to improve spasticity, range of motion, hand function, and ambulation in hemiplegic and quadriplegic patients (Katz et al., 2000; Shingleton et al., 2011).

Oral medications can also be used in conjunction with these methods. There is a long list of anti-spasticity medications. Unfortunately many of them cause sedation and cognitive impairment which limits their use, especially in patients with brain injury who are particularly sensitive to central side effects of medications. The four most commonly used oral medication for treating spasticity in the United States are baclofen, diazepam, dantrolene, and tizanidine.

Baclofen is a structural analog of GABA and binds to presynaptic GABA-B receptors in the CNS. This medication acts by inhibiting gamma motor neuron activity to muscle spindle intrafusal fibers which decreases monosynaptic and polysynaptic reflexes. A limited amount of orally ingested baclofen crosses into the cerebral spinal fluid. Given that often high doses of baclofen are needed to achieve meaningful clinical improvement of spasticity, patients with brain injury are unable to tolerate this medication due to central side effects that occur at these doses.

Side effects of baclofen include drowsiness, confusion, sedation, memory and attention impairment, headache, dizziness, weakness, coordination impairment, hallucinations, and seizures (Epocrates Inc., 2016; Meythaler & Kowalski, 2011).

Diazepam is a benzodiazepine that treats spasticity by facilitating sodium conductance which enhances the release of the GABA-A neurotransmitter. Benzodiazepines are CNS depressants and are the least likely oral medications to effectively treat spasticity in patients with brain injury due to prominent side effects of lethargy, sedation, and cognitive impairment. Its side effect profile also includes ataxia, headache, tremor, dystonia, dizziness, irritability, amnesia, seizures, urinary retention, and nausea (Epocrates Inc., 2016; Meythaler & Kowalski, 2011).

Dantrolene is a muscle relaxant that works peripherally by inhibiting calcium release from the sarcoplasmic reticulum which reduces contraction force produced by intrafusal and extrafusal muscle fibers. Although in theory it has been thought to be the best oral medication to treat spasticity in patients with acquired brain injury given its mechanism of action is peripheral rather than central acting, in actual clinical practice, its use is limited due to its high side effect profile. Side effect profile includes sedation, dizziness, hallucinations, fatigue, weakness, and seizures. It also increases risk of hepatotoxicity, and liver enzymes must be carefully monitored while on this medication.

Tizanidine is a centrally acting, selective alpha-2 adrenergic agonist and works presynaptically to inhibit excitatory neurotransmitter effect on the motor neuron and inhibits polysynaptic reflexes. Unlike baclofen and dantrolene, this medication does not cause weakness as it does not directly affect skeletal muscle fibers (Epocrates Inc., 2016). Side effects include sedation, dizziness, dry mouth, hypotension, anesthesia, and constipation. Clinically, tizanidine tends to be the most practical of spasticity medications for use in patients with acquired brain injury, as its side effect profile, although limiting, is usually more tolerable compared to the previously discussed oral spasticity medications.

Other treatment options include phenol or ethanol chemoneurolysis and botulinum toxin injections for localized treatment and intrathecal baclofen pump placement for generalized spasticity management. Chemical neurolysis injections with phenol or ethanol functions to damage a specific area of a nerve in order to disrupt nerve conduction to control spasticity. Typically, the effects of this type of injection last 3–6 months but may last close to a year. The most concerning side effect is dysesthesias that occur as a result of blocking sensory or mixed sensory/motor nerves. The pain is neuropathic in nature and usually starts a few days to a couple weeks after administration of the injection. Electrical stimulation guidance should be used to selectively target the motor branch of the nerve to avoid risk of dysesthesia.

Botulinum toxin is administered directly into muscles via injections allowing the toxin to be targeted to specific muscles to maximize clinical effect. It prevents acetylcholine vesicles from binding with proteins needed for fusion to surface membranes. This decreases the number of presynaptic transmitter vesicles preventing neuromuscular transmission. The onset of botulinum toxin is usually 3–7 days; the effect peaks at 3–4 weeks and lasts 3–4 months. The most common side effects are

excessive weakness, which occasionally may spread to other adjacent muscles, and injection site pain or irritation. Rarely, nausea, headache, fatigue, and upper respiratory symptoms have been reported. If improperly injected, serious effects from systemic toxin spread, such as respiratory compromise, may occur. Botulinum toxin A is available as onobotulinumtoxin A (Botox) or abobotulinumtoxin A (Dysport). Botox and Dysport are FDA approved for upper and lower limb spasticity and cervical dystonia (Epocrates Inc., 2016). Botulinum toxin B available as rimabotulinumtoxin B (Myobloc) is FDA approved only for cervical dystonia.

The intrathecal baclofen pump allows baclofen to be administered directly into the intrathecal space. Intrathecal administration significantly increases the dose of baclofen that can be delivered directly to the CNS GABA-B receptors, with considerably less side effects of sedation and cognitive deficits. It has been shown to be more efficacious for lower extremity spasticity. The system consists of a pump reservoir, which is surgically placed in the abdominal subcutaneous tissue and attached to a catheter that delivers baclofen to the CSF. Patients need to follow up regularly for baclofen pump refills. This can be considered for a patient who has failed other less invasive methods of spasticity management and has more generalized rather than focal spasticity. Proper patient selection and education prior to implanting an intrathecal baclofen pump are essential as maintenance is a long-term commitment and critical responsibility on the part of the patient or their caregiver. A patient who is considering the baclofen pump can undergo a trial in which a bolus dose of baclofen is injected intrathecally via lumbar puncture and the patient's response is monitored. A common criterion used to signify a successful trial is a reduction of spasticity by 2 points on the Modified Ashworth Scale. It is important to recognize risks and symptoms of baclofen withdrawal or overdose. Baclofen withdrawal initially presents with rebound spasticity, pruritus, tremors, or irritability. If the condition progresses, symptoms worsen to include hallucinations, altered mental status, seizures, fever, hemodynamic instability, and death. Symptoms of overdose include significant hypotonia, hyporeflexia, respiratory depression, seizures, autonomic instability, hypothermia, hallucinations, coma, and possibly death (Saulino, Yablon, Moberg-Wolff, Chow, Stokic 2011).

Surgical procedures for spasticity are permanent interventions and less frequently used. These include tendon lengthening, transfers, and rhizotomies. Surgery may be considered for patients with very severe spasticity that has or is developing contractures or if all other spasticity interventions have been exhausted. Like intrathecal baclofen, patient selection is critical.

Medical Complications in Neurorehabilitation

With the current healthcare trend of mobilizing patients earlier on and limiting individual's length of stay on acute medical floors, vigilance for acute medical complications on the rehabilitation floors must be increased.

Deep Venous Thrombosis (DVTs)

The diagnosis of DVT requires a high index of suspicion, especially in the brain-injured population who may not necessarily be able to provide the symptomatic complaints that other hospitalized patients would be able to. Venous thromboembolic (VTE) disease constitutes a spectrum of disorders, including DVT, pulmonary embolism (PE), and superficial thrombophlebitis, all of which patients are still prone to despite prophylaxis. Prompt diagnosis and treatment are of upmost importance, as progression of a DVT can be fatal. The triad described by Virchow in 1856 including venous stasis, hypercoagulability, and endothelial injury helps to explain the increased risk of DVTs in the brain-injured population, as they are less mobile than most other individuals admitted to the hospital. Despite mechanical and pharmacological prophylaxis, patients with brain injuries that are in an intensive care unit are at an increased risk of developing VTE disease, especially those who are older and have a greater weight or severity of brain injury (Huijben et al., 2017; Skrifvars et al., 2017). Given this increased susceptibility, noninvasive techniques to diagnose DVTs, primarily ultrasonography of the lower extremities, are commonly used. Other techniques include D-dimer assays, which are non-specific, impedance plethysmography, and contrast venography. Furthermore, pulmonary angiography remains the gold standard for the diagnosis of PE.

Commonly utilized prophylactic measures include low-dose unfractionated heparin (5000 units every 8 h), low molecular weight heparin (Enoxaparin 40 mg once a day), and/or mechanical compression devices. Additionally, an inferior vena cava filter can also be utilized in those who may have a contraindication to chemical and/or mechanical DVT prophylaxis. If a DVT or PE is detected, treatment with full-dose anticoagulation is typical for 3–6 months. The presence of intracranial hemorrhage usually causes some hesitation in initiating chemical VTE prophylaxis; however, initial studies have shown that the use of chemical VTE prophylaxis in patients with brain injuries who have stable head cat scans for at least 24 h is safe and actually decreases the rate of DVTs (Farooqui, Hiser, Barnes, & Litofsky, 2013). Further meta-reviews revealed that chemical DVT prophylaxis should not be given within 3 days of moderate- to high-risk intracranial hemorrhages, but in low-risk patients, 2 days is a reasonable time frame (Abdel-Aziz, Dunham, Malik, & Hileman, 2015). Furthermore, in patients with diffuse axonal injury who have not developed intracranial hemorrhage, it is reasonable to start chemical prophylaxis after 3 days. DVT risk significantly increases after chemical prophylaxis is withheld for greater than 7 days (Abdel-Aziz et al., 2015).

Heterotrophic Ossification (HO)

HO is the formation of mature lamellar bone in abnormal, ectopic, extraskeletal soft tissue, primarily occurring in the proximal joints of the upper and lower extremities (Edwards & Clasper, 2015; Gil, Waryasz, Klyce, & Daniels, 2015;

Ranganathan et al., 2015). Patients with brain injuries who have prolonged coma greater than 2 weeks, spasticity, long-bone fractures, and decreased range of motion are at increased risk of developing HO. Despite the incidence of HO going down secondary to patients being mobilized earlier, including range of motion to the joints, and spasticity being better controlled, it can still pose a problem with rehabilitating and functional progress in a patient with a brain injury. Most frequently in those with brain injury, HO affects the hips most commonly, followed by the elbows, shoulders, and then knees. Symptoms of HO may include joint and muscle pain, warmth, swelling, limited range of motion, and contracture formation, which may also represent other ailments (Ellerin, Helfet, Parikh, et al., 1999; Ranganathan et al., 2015). To detect HO in the earliest phases, a triple-phase Technetium-99 bone scan should be completed (Ranganathan et al., 2015). HO may be seen within the first 2–4 weeks in phase I and phase II (blood-flow and blood-pool phases) of a triple-phase bone scan. Phase III (static phase) will detect HO after 4–8 weeks. The use of X-rays is limited to detecting HO that has been present anywhere from 3 weeks to 2 months, as it requires some maturation of the bone. Laboratory testing, such as checking serum alkaline phosphatase, calcium, and phosphorous levels, is fairly non-specific and may be abnormal in a number of conditions (Ranganathan et al., 2015). Current therapies to treat HO include medications including anti-inflammatories, in particular indomethacin, bisphosphonates, opioid painkillers, physical therapy and therapeutic modalities, radiation therapy, and, as a last resort, surgery. This may require further imaging to preoperatively plan the surgery (Ellerin et al., 1999; Ranganathan et al., 2015).

Paroxysmal Sympathetic Hyperactivity (PSH)

Patients with acquired brain injuries may suffer from paroxysmal sympathetic hyperactivity, the official term that has replaced many other descriptions of this clinical syndrome, such as dysautonomia, diencephalic seizures, paroxysmal autonomic instability with dystonia, and paroxysmal sympathetic storming. This constellation of symptoms may present with alterations in blood pressure, pulse, temperature, and posturing (Meyfroidt, Baguley, & Menon, 2017). Typically, individuals have elevated blood pressures with tachycardia, fevers, tachypnea, and diaphoresis, believed to be related to the lack of inhibition from the parasympathetic system and a hyperadrenergic state. It is typically a diagnosis of exclusion in which other more detectable ailments, such as infection/sepsis, neuroleptic malignant syndrome, malignant hyperthermia, clotting, and drug withdrawal, must be first ruled out. Furthermore, these symptoms are self-limiting, but they must be controlled as they can lead to other end organ damage, including additional acquired brain injuries, ultimately affecting a patient's prognosis.

The presence of PSH has also been shown to have an effect on outcomes, in which its presence is associated with a worse outcome (Meyfroidt et al., 2017). Furthermore, a retrospective case-controlled study of subjects with and without

PSH showed that subjects with PSH had a significantly worse Glasgow Outcome Scale, Functional Independence Measure, duration of PTA, and hospital length of stay (Baguley et al., 1999, 2007; Perkes, Menon, Nott, & Baguley, 2011). Additionally, these individuals are believed to be at a higher risk to develop HO as well (Hendricks, Geurts, van Ginneken, Heeren, & Vos, 2007; Perkes et al., 2011).

PSH is most commonly seen in individuals in which anoxic brain injury was the primary pathophysiology of the brain injury. Traumatic brain injuries are the second most common cause, followed by strokes as the third leading cause in which they are commonly associated with hemorrhagic strokes as compared to ischemic strokes (Perkes et al., 2011).

Treatment options for some of these symptoms include a cooling blanket, ice packs, and infusion of cold saline for hyperthermia. First-line oral medications to help control symptoms include beta-blockers, most opioids (i.e., morphine), gabapentin, benzodiazepines, centrally acting alpha-agonists (i.e., clonidine), and beta-antagonists (Baguley et al., 2007). In regard to beta-blockers, propranolol in particular is used to control heart rate, blood pressure, as well as temperature. Moreover, dopamine agonists such as bromocriptine and datrolene have some efficacy in symptom management but are typically considered second-line medications.

Hydrocephalus

With a widely reported incidence, anywhere from 30% (Gudeman, Kishore, Becker, et al., 1981) all the way to 86%, this complication of an acquired brain injury is difficult to predict. Patients with acquired brain injuries that were noted to have hydrocephalus were those who had more severe brain injuries and were more frequently in vegetative state and those who needed longer rehabilitation stays (Mazzini et al., 2003). With this in mind, though, it is crucial to be weary and mindful of the symptoms that would lead one to believe this may be a future complication. Typically speaking, those with hydrocephalus tend to start with vague symptoms, such as nausea, headache, and fatigue, and may progress to more obvious signs, like gait instability, poor arousal, vomiting, and newfound weakness. Being mindful of the cerebrospinal fluid (CSF) tract and individuals that would be at higher risk can help to screen these patients.

CSF is produced by the choroid plexus, with most of it being secreted in the lateral ventricles (Kartal & Algin, 2014). It travels through the ventricles of the brain as well as the subarachnoid space, only to be reabsorbed by the arachnoid villa/granulations (Kartal & Algin, 2014). Expansion of the ventricles by the CSF is what leads to hydrocephalus and can be classified as either obstructive or non-obstructive hydrocephalus. In the patient population with acquired brain injuries, those with subarachnoid hemorrhages are at the greatest risk for developing non-obstructive hydrocephalus, in that the blood products prevent the arachnoid villa from resorbing the CSF. Other patient populations that would be at risk are those with intracranial tumors, which may compress on the pathway of CSF, leading to a blockage of the CSF.

Typically, intracranial pressures are monitored with extra-ventricular drains, and prior to these drains being removed, a clamping trial is undertaken. This trial helps to determine whether or not a CSF shunting is necessary. If necessary, the ventriculoperitoneal shunt (VPS) is the predominant mode of shunting (Reddy, Bollam, & Caldito, 2014). With this shunt, CSF is redirected into the peritoneal space relieving the increased pressure in the ventricles.

Seizures

Having an acquired brain injury places individuals at a high risk for seizures related to their brain injury (see Chap. 3). Seizures themselves have risks in terms of subsequent complications such as secondary brain injury, higher neuronal metabolic demand, aspiration pneumonia, further physical trauma, or even death (Zafar, Khan, Ghauri, & Shamim, 2012). For these reasons, it is crucial that patients with brain injuries receive proper prophylaxis in order to prevent seizures. Post-traumatic seizures are classified based on their timing in relation to time of brain injury. Immediate seizures are defined as seizures that occur within the first 24 h since injury, early seizures occur between 1 and 7 days of injury, and late seizures are those that occur any time after 7 days post-injury. Length of prophylaxis or treatment is dependent on when the seizure occurs.

Anti-epileptic treatment after an acquired brain injury has been shown to be beneficial in this population. The drug phenytoin was studied in 1990 and was shown to be significantly more effective in preventing post-traumatic brain injury seizure as compared to placebo within the first 7 days of injury (Temkin et al., 1990). That being said, phenytoin has a greater side effect profile than some of the newer anti-epileptic medications, such as levetiracetam. The general guidelines at this time is that seizure prophylaxis is no longer required if a patient with brain injury has completed 7 days of prophylaxis without any seizure like activity within days 1–7 post-injury. This is important to remember as anti-epileptic medications do not come without potential side effects, particularly lethargy, that may prolong rehabilitation and recover.

Aspiration Pneumonia

Individuals with brain injuries must be evaluated for dysphagia early in their course, as many studies have found that infections from aspiration pneumonia, pneumonia, and septicemia cause a higher than expected number of deaths in those with brain injuries (Harrison-Felix et al., 2015). Given the intricacies and necessary coordination for chewing and swallowing, improper swallowing and potential for aspiration are high in those with brain injuries. Aspiration may be occurring silently, or the

patient may even be actively coughing while attempting to swallow certain consistencies of food or liquid. A bedside swallowing exam may be completed initially, but a modified barium swallow study or fiber-optic endoscopic evaluation should truly be done to fully evaluate a patient's swallowing capabilities in detail and rule out silent aspiration. Penetration differs from aspiration in that oral material that is being attempted to be swallowed does not pass the true vocal cords, whereas with aspiration this food content does pass through the vocal cords (Ozaki et al., 2010). Some individuals may be safe to be placed on an oral diet if they are penetrating; however those who are at higher risk for aspirating food or liquid may require special techniques while eating or even a percutaneous endoscopic gastrostomy (PEG) tube placement to provide nutrition. These modified techniques of eating may include head tilt/rotation, chin tucks, supervised meals, alternating solids and liquids, or discouraging the use of straws. The risk of aspiration, providing nutrition, and modes of providing medications are all aspects that need consideration when evaluating swallowing and the risk of aspiration.

Neuroendocrine Dysfunctions

The primary source of neuroendocrine dysfunction in those with acquired brain injuries is the pituitary gland. This central area of concern is a very small organ, located at the base of the brain, within the sella turcica. It consists of both an anterior and a posterior portion, each releasing different hormones (Table 4.5).

The first reported case of anterior pituitary dysfunction as a result of head trauma was described in 1918 (Ghigo, Masel, Aimaretti, et al., 2005). Subsequent studies in patients with brain injuries have shown an increased risk of neuroendocrine dysfunction in this population, since the pituitary and thalamus are prone to insult (Ghigo et al., 2005). The pituitary has a fragile vascular supply, lending itself to increased injuries (Kelly et al., 2000). In particular, it has been seen that patients are at a higher risk of growth hormone deficiency which is released by the anterior pituitary. More specifically, the cells that release growth hormone are thought to be located more laterally at the wings of the pituitary gland, a location vulnerable to damage (Ghigo et al., 2005). In contrast, cells that are located more ventrally, such as cells that release adrenocorticotropic hormone and thyroid-stimulating hormone, are generally better protected from damage.

Table 4.5 Hormones released by the anterior vs. posterior pituitary

Anterior pituitary	Posterior pituitary
Growth hormone	Antidiuretic hormone
Adrenocorticotropic hormone	Oxytocin
Thyroid-stimulating hormone	
Luteinizing hormone	
Follicle-stimulating hormone	
Prolactin	

Testing for neuroendocrine dysfunction is important as deficiencies in these hormones may influence the rehabilitation process. Hormones such as growth hormone may improve muscle mass, and gonadal hormones may influence energy levels, both of which play a crucial role in how patients recover (Ghigo et al., 2005). Moreover, hormone replacement for diabetes insipidus, adrenal insufficiency, and thyroid deficits should be immediate to avoid further medical complications (Ghigo et al., 2005).

Stroke Recovery

Constraint-Induced Movement Therapy

The practice of constraint-induced movement therapy was first investigated in studies by Edward Taub and Wolf, most notably the EXCITE trial. The concept was designed to counteract the idea that the paralyzed upper extremity of an individual with stroke develops "learned nonuse" based on observations of differentiated limbs studied in primates (Taub, 1980). It was found that restricting the intact limb for a certain period of time and inducing the primates to use the affected limb promoted neuroplastic changes that allowed the ability to have functional use of the impaired limb.

The original CIMT protocol (Taub et al., 2006) involves:

1. Constraining the unaffected limb with a mitt or sling for 90% of waking hours.
2. Engagement in 6 h of task-specific therapy a day of gradually increasing intensity with the affected upper extremity for 2 weeks.
3. Frequent biofeedback regarding the speed and quality of movement.

Qualifying patients have to demonstrate at least 10° of wrist extension, at least 10° of active thumb abduction/extension, and at least 10° of extension in at least two additional digits. Movement must be performed at least three times in 1 min. They must also demonstrate adequate balance wearing the restraint during transfers (Wolf et al., 2006).

Given studies showing challenges with adherence to CIMT treatment protocol (Daniel, Howard, Braun, & Page, 2012; Page, Levine, Sisto, Bond, & Johnston, 2002; Viana & Teasell, 2012), a less-intense constraint therapy regimen of *modified constraint-induced movement therapy* (mCIMT) was introduced. Similar to the original CIMT, mCIMT includes restraint, a repetitive task practice period, and behavioral techniques to promote use. There is no one defined protocol for mCIMT, and period of restraint and task practice period vary. The wearing schedule of the restraint has varied in studies from 5 to 12 h/day, and the period of task-specific therapy with the affected extremity has varied from 30 min to 6 h/day for 2–12 weeks (Kwakkel, Veerbeek, van Wegen, & Wolf, 2015; Page, Levine, & Leonard, 2005; Smania et al., 2012).

Recent meta-analysis of CIMT and mCIMT showed significant improvement in measures of upper extremity function (Etoom et al., 2016) and, specifically, movement, activities, and quality of movement (Kwakkel et al., 2015). No significant improvement was observed in grip strength, ADLs, IADLs, or quality of life. It has been noted that the quality of evidence for CIMT and mCIMT was weak due to small sample sizes (Etoom et al., 2016; Kwakkel et al., 2015).

A Cochrane review published in 2017 demonstrated significant benefit of CIMT including mCIMT on motor function and dexterity, but no significant benefit on disability or quality of life (Corbetta, Sirtori, Castellini, Moja, & Gatti, 2015). This updated a 2009 Cochrane review (Sirtori, Corbetta, Moja, & Gatti, 2009) that concluded "modest evidence that CIMT improves disability."

The above studies compared outcomes of CIMT protocols to conventional rehabilitation or no intervention. As per current review of the literature, studies examining the effect of CIMT combined with therapy are lacking. Currently there is inadequate evidence in the literature to determine the optimal dose or timing to start CIMT (Etoom et al., 2016).

Virtual Reality

Virtual reality has been defined as "the use of interactive simulations created with computer hardware and software to present users with opportunities to engage in environments that appear and feel similar to real-world objects and events" (Weiss, Kizony, Feintuch, & Katz, 2006). Interventions can vary in the degree of "immersion" that the user experiences. Immersion refers to the degree the user perceives their experience as in a virtual environment versus in real life. Low immersion technology is mainly screen-based computer tasks or games. A partially immersive experience would involve a person having a sensation that they are surrounded by a three-dimensional world, such as in an IMAX theater or flight simulator. Fully immersive virtual reality is more involved and utilizes head-mounted devices that when worn the user perceives their body inside and interacting in a virtual setting.

The concept behind why virtual reality may help improve stroke recovery is based on the hypothesis that motor plasticity occurs through mirror neurons or mental imagery. It is also felt that virtual reality provides an enriching and exciting environment that is motivating for patients to engage in therapeutic activities. Another benefit is it allows for patients to practice tasks that may be dangerous in the real world, like driving or crossing the street.

A good handful of systematic reviews have analyzed the effect of virtual reality for stroke rehabilitation. A 2017 Cochrane review found that there was no significant difference in upper extremity function or grip strength when virtual reality was compared to conventional therapy, but there was a significant improvement in upper extremity function when virtual reality was adjunctive to traditional therapy. Also, adjunctive treatment with virtual reality demonstrated a significant improvement in ADL functioning. No significant improvement in gait speed or balance was

observed. Overall the quality of evidence was noted to be low due to small sample sizes and the degree of heterogeneity of interventions (Laver et al., 2017). This updated a 2015 Cochrane review which concluded that there is low-quality evidence showing significant benefit of VR either compared to or adjunctive to a traditional rehabilitation program for upper limb motor function and ADLs. No significant benefit was found for improvement in grip strength, gait speed, or global motor function (Laver, George, Thomas, Deutsch, & Crotty, 2015).

Another area of focus in the stroke rehabilitation literature involving virtual reality has been on the effect on lower extremity function: gait speed, balance, and mobility. Literature has been mixed, but in general most systematic reviews suggest that virtual reality interventions show benefit in improving gait speed, mobility, and balance. Combining conventional therapy and virtual reality appears to be more effective (De Rooij, van de Port, & Meijer, 2016; Luque-Moreno et al., 2015). One meta-analysis comparing virtual reality interventions to traditional therapy found substituting virtual reality for any period of time of a traditional therapy program showed significant improvement on measures of gait speed, mobility, and balance. Combining virtual reality-based rehabilitation and a traditional rehabilitation program showed significant improvement in measures of mobility but lack of evidence for walking speed or balance (Corbetta, Imeri, & Gatti, 2015). Chen found that virtual reality significantly improved static and dynamic balance in chronic stroke, when combined with traditional rehabilitation therapy, but this was not noted for virtual reality alone (Chen et al., 2016). Reviews by Li and Darekar also found improvement in balance for patients treated with virtual reality compared to patients treated with conventional rehabilitation (Li, Han, Sheng, Ma et al., 2016; Darekar, McFadyen, Lamontagne, & Fung, 2015). Some variability in the above studies may likely be due to differences in the included studies and number of studies in the above reviews. Much of the literature on virtual reality for lower extremity stroke recovery is limited by heterogeneity of interventions and small sample sizes. Most of the studies involved chronic stroke patients, so there is inadequate evidence to determine the effect of virtual reality on patients with acute or subacute stroke. Currently, there is insufficient evidence to determine the timing, dosing, and VR interventions that are best for stroke rehabilitation (Corbetta, Imeri, & Gatti, 2015).

Brain Computer Interface

Brain-computer interface (BCI) is a computer-based system that receives, analyzes, and translates brain signals into commands that are then transmitted to an output device in order to carry out an intended action. As such, BCIs provide the opportunity to interact with the environment by using signals instead of muscles. Certainly, this technology offers great potential for rehabilitation applications, some of which will be briefly presented below. Also, the main brain imaging and signals presently used in BCI will also be mentioned.

In order for BCIs to collect signals informative of the user's intention, brain activity is recorded and translated into electrical signals. Two of these signals can be monitored: electrophysiological and hemodynamic. The former consists of electrophysiological activity generated by electrochemical transmitters that exchange information between the neurons, whereas the latter results from the process of glucose being released by the bloodstream to activate neurons. Different neuroimaging approaches have been used to capture brain activity information that may be later processed by BCIs, including:

(a) Electroencephalography (EEG)
(b) Magnetoencephalography (MEG)
(c) Electrocorticography (ECoG)
(d) Intracortical neuron recording
(e) Functional magnetic resonance imaging (fMRI)
(f) Near-infrared spectroscopy (NIRS)

By far, EEG is the more frequently used neuroimaging modality due to its high temporal resolution, low cost compared to other modalities, high portability, and low-risk profile (Nicolas-Alonso & Gomez-Gil, 2012).

Once the brain activity has been captured and analyzed, BCIs need to interpret the user's intentions by keeping track of it. While there is a large number of brain signals available, only a few signals are used in current BCI systems. These include:

(a) Visual evoked potential (VEP) which is a brain activity occurring in the visual cortical areas after a visual stimulus. Examples include transient VEPs (TVEPs) and steady-state VEPs (SSVEPs).
(b) Slow cortical potentials (SCPs) or slow voltage changes lasting from one to several seconds.
(c) P300 evoked potentials, which are positive peaks in the EEG resulting from infrequent auditory, visual, or somatosensory stimuli.
(d) Sensorimotor rhythms, which include mu and beta rhythms. Both oscillations in brain activity taking place in the mu or rolandic band and the beta band. Most current rehabilitation uses of BCIs make use of VEPs and P300 EPs.

Given the ability to pick up different kinds of brain signals that can be translated into commands which may be used in real time to execute the individual's intentions by means of hardware, BCI has a significant potential to variously assist individuals with communication or motor deficits to overcome to some extent their deficits. The interested reader is referred to the articles by Carelli et al. (2017) and Shih, Krusienski, and Wolpaw (2012), where a review of current applications in rehabilitation is provided. Patients that suffer from severe motor disabilities, including locked-in syndrome caused by stroke or amyotrophic lateral sclerosis (ALS), may be among the groups who could gain more benefit from such technology (De Massari et al., 2013). A common application for individuals with communications involves training in the use of a virtual keyboard on screen where letter selection can be done by means of voluntarily controlled SCPs (Hinterberger et al., 2004) or P300 event-related brain potentials (Farwell & Donchin, 1988).

Another area with tremendous potential for physical rehabilitation is that of motor restoration, where typical applications include functional electrical stimulation (FES). FES helps to compensate for the loss of voluntary movement by eliciting artificial muscle contraction by using EEG control signals, which can substantially help individuals who are rehabilitating from strokes, spinal cord injury, and other neurological diseases affecting sensory and motor functions. Alternatives to FES have also been explored to assist those who lack the residual movements needed for its use, e.g., using rolandic oscillations or ERPs to control devices such as hand orthosis (Pfurtscheller, Guger, Müller, Krausz, & Neuper, 2000) or a neuroprosthetic device to restore the grasp function for people with SCI. Another significant area of application in rehabilitation settings concerns locomotion, which includes the use of EEG-based BCI for controlling powered wheelchairs (Tanaka, Matsunaga, & Wang, 2005). Applications for environmental control and entertainment have further been described in the comprehensive article by Nicolas-Alonso and Gomez-Gil.

Stem Cell Therapy for Brain Injury

Current therapies available after a patient suffers a brain injury focus on preventing and reducing the extent of secondary insult rather than repairing the primary source of damage. Injury to the brain is a complex process that could be diffuse or focal consisting of a combination of both white matter loss and neuronal damage resulting in functional impairments (Rolfe & Sun, 2015). To date no effective treatment for the structural repair of brain parenchyma after an individual suffers a brain injury has been developed (Rolfe & Sun, 2015). Stem cell therapy has been utilized in alternative diagnoses to help restore damaged tissue, which brings into question its role in a brain that has acquired damaged.

Recent studies indicate that multipotent neural stem cells, which are only present in selected brain regions, render the brain capable of generating new neural connections (Gage, Kempermann, Palmer, Peterson, & Ray, 1998; Lois & Alvarez-Buylla, 1993; Rolfe & Sun, 2015; Sun, 2014). These neural progenitor cells may play a reparative role in response to central nervous system damage. It is believed that the brain has a natural ability to repair tissue with cell proliferation and neurogenesis (Rolfe & Sun, 2015; Sun, 2014). Additionally, elevated levels of neurogenesis have been observed in response to brain injury which is suggestive of the brain natural ability to restore damaged neurons (Sun, 2014). This brings hope that transplantation of progenitor cells will allow for the potential of these cells to differentiate into region-specific cell lines and integrate themselves into the natural host tissue, in particular the specific brain region they are implanted in.

A crucial area that is of particular focus with stem cell therapy after a brain injury is the hippocampus, an area that is vulnerable to second injury and has been noted

to have decreased levels of neurogenesis after a pathological event (Rolfe & Sun, 2015; Sun, 2014). This area of the brain is associated with learning and memory, which plays a vital role in functional recovery after a brain injury. There are currently many clinical trials currently underway to see if certain medications or clinical circumstances will enhance neurogenesis, including erythropoietin, statins, progesterone, anti-depressants such as imipramine, and hypothermia.

There is still a lot of knowledge that is to be gained about this potential recovery mechanism. Ideally, embryonic stem cells are the best source of neuronal implantation as they are pluripotent and have the potential for increased plasticity (Rolfe & Sun, 2015). This stem cell type has been shown to differentiate, migrate, and make neuronal innervations when transplanted into normal and damaged central nervous system tissue (Hentze, Graichen, & Colman, 2006). With the current research underway in rodent brain injury models, there is hope that one day this therapy will be made available for therapeutic use to those who suffer an acquired brain injury.

Conclusion

Many individuals are left with a combination of physical, cognitive, and psychosocial impairments following an acquired brain injury. The challenge for physiatrists specializing in neuro-rehabilitation is in applying the knowledge gathered from evidence-based medicine to the management of each patient's unique pattern of deficits, as well as delivering quality and cost-effective care in a system where resources may often be limited.

The pattern of impairments varies greatly from patient to patient. Participation restrictions may be affected by differences in the social and physical environments to which the brain injury survivor returns providing further treatment challenges for the physiatrist specializing in brain injury rehabilitation (Whyte, Hart, Laborde, & Rosenthal, 1998). Therefore an interdisciplinary treatment approach that considers the complex and unique pattern of deficits seen in an individual patient is the best approach to treatment. Physiatrists must be able to communicate effectively with patients, families, and staff and provide assessments of prognosis based on literature, prognostic parameters, and clinical experience. At the same time, we must never take away hope.

Brain injury survivors face many challenges to community living everyday of their lives. These individuals experience the unique challenge of "walking the sometimes conflicting paths of who they were, who they are, and who they want to be" (Gordon, Hibbard, Brown, Flanagan, & Korves, 1999). Rehabilitation and community integration focus on helping the person achieve a new sense of "self." While we are often unable to "cure" those who have suffered a devastating acquired brain injury, we are capable of ameliorating the impact of physical and cognitive impairments on the individual's functional status. Physiatrists and the rest of the rehabilitation team provide

individuals with the tools they require to adapt and adjust to their new circumstances. Helping to meet the complex, ongoing needs of this population is one of the key challenges for all involved in neuro-rehabilitation, including clinicians, survivors, as well as family and friends.

References

Abdel-Aziz, H., Dunham, C. M., Malik, R. J., & Hileman, B. M. (2015). Timing for deep vein thrombosis chemoprophylaxis in traumatic brain injury: An evidence-based review. *Critical Care, 19*, 96.

ADA National Network. (n.d.). *What is the definition of disability under the ADA?* Retrieved January, 2018, from https://adata.org/faq/what-definition-disability-under-ada

Baguley, I. J., Nicholls, J. L., Felmingham, K. L., Crooks, J., Gurka, J. A., & Wade, L. D. (1999). Dysautonomia after traumatic brain injury: A forgotten syndrome? *Journal of Neurology, Neurosurgery, and Psychiatry, 67*(1), 39–43.

Baguley, I. J., Slewa-Younan, S., Heriseanu, R. E., Nott, M. T., Mudaliar, Y., & Nayyar, V. (2007). The incidence of dysautonomia and its relationship with autonomic arousal following traumatic brain injury. *Brain Injury, 21*(11), 1175–1181.

Berger, M. S., Pitts, L. H., Lovely, M., Edwards, M. S., & Bartkowski, H. M. (1985). Outcome from severe head injury in children and adolescents. *Journal of Neurosurgery, 62*(2), 194–199.

Boake, C., Francisco, G. E., Ivanhoe, C. B., & Kothari, S. (2000). Brain injury rehabilitation. In R. L. Braddom (Ed.), *Physical medicine and rehabilitation* (pp. 1073–1116). Philadelphia, PA: WB Saunders.

Bowen, A., Tennant, A., Neumann, V., & Chamberlain, M. A. (2001). Neuropsychological rehabilitation for traumatic brain injury: Do carers benefit? *Brain Injury, 15*(1), 29–38.

Carelli, L., Solca, F., Faini, A., Meriggi, P., Sangalli, D., Cipresso, P., … Poletti, B. (2017). Brain-computer interface for clinical purposes: Cognitive assessment and rehabilitation. *BioMed Research International, 2017*, 1–11.

Chen, L., Ambrose Lo, W. L., Mao, Y. R., Ding, M. H., Lin, Q., Li, H., … Huang, D. F. (2016). Effect of virtual reality on postural and balance control in patients with stroke: A systematic literature review. *BioMed Research International, 2016*, 1–8.

Cicerone, K. D., Mott, T., Azulay, J., Sharlow-Galella, M. A., Ellmo, W. J., Paradise, S., & Friel, J. C. (2008). A randomized controlled trial of holistic neuropsychologic rehabilitation after traumatic brain injury. *Archives of Physical Medicine and Rehabilitation, 89*(12), 2239–2249.

Colcombe, S., & Kramer, A. F. (2003). Fitness effects on the cognitive function of older adults: A meta-analytic study. *Psychological Science, 14*, 125–130.

Corbetta, D., Imeri, F., & Gatti, R. (2015). Rehabilitation that incorporates virtual reality is more effective than standard rehabilitation for improving walking speed, balance and mobility after stroke: A systematic review. *Journal of Physiotherapy, 61*, 117–124.

Corbetta, D., Sirtori, V., Castellini, G., Moja, L., & Gatti, R. (2015). Constraint-induced Movement therapy for upper extremities in people with stroke. *Cochrane Database of Systematic Reviews, 10*, 1–100.

Cotman, C. W., & Berchtold, N. C. (2002). Exercise: A behavioral intervention to enhance brain health and plasticity. *Trends in Neurosciences, 25*, 295–301.

Daniel, L., Howard, W., Braun, D., & Page, S. J. (2012). Opinions of constraint-induced movement therapy among therapists in southwest Ohio. *Topics in Stroke Rehabilitation, 19*(3), 268–275.

Darekar, A., McFadyen, B. J., Lamontagne, A., & Fung, J. (2015). Efficacy of virtual reality based intervention on balance and mobility disorders post-stroke: A scoping review. *Journal of Neuroengineering and Rehabilitation, 12*, 46.

De Massari, D., Ruf, C. A., Furdea, A., Matuz, T., Van Der Heiden, L., Halder, S., … Birbaumer, N. (2013). Brain communication in the locked-in state. *Brain, 136*(6), 1989–2000.

De Rooij, I. J. M., van de Port, I. G. L., & Meijer, J. G. (2016). Effect of virtual reality training on balance and gait ability in patients with stroke: Systematic review and meta-analysis. *Physical Therapy, 96*, 1905–1918.

Edwards, D. S., & Clasper, J. C. (2015). Heterotopic ossification: A systematic review. *Journal of the Royal Army Medical Corps, 161*(4), 315–321.

Ellerin, B. E., Helfet, D., Parikh, S., Hotchkiss, R. N., Levin, N., Nisce, L., Moni, J. (1999). Current therapy in the management of heterotopic ossification of the elbow: A review with case studies. *American Journal of Physical Medicine & Rehabilitation, 78*(3), 259–271.

Epocrates Inc. (2016). *Epocrates RX version 18.4*. New York, NY: Epocrates.

Etoom, M., Hawamdeh, M., Hawamdeh, Z., Alwardat, M., Giordani, L, Bacciu,S,...Foti,C. (2016). Constraint-induced movement therapy as a rehabilitation intervention for upper extremity in stroke patients: Systematic review and metaanalysis. *International Journal of Rehabilitation Research, 39*, 197–210.

Farooqui, A., Hiser, B., Barnes, S. L., & Litofsky, N. S. (2013). Safety and efficacy of early thromboembolism chemoprophylaxis after intracranial hemorrhage from traumatic brain injury. *Journal of Neurosurgery, 119*(6), 1576–1582.

Farwell, L. A., & Donchin, E. (1988). Talking off the top of your head: Toward a mental prosthesis utilizing event-related brain potentials. *Electroencephalography and Clinical Neurophysiology, 70*(6), 510–523.

Furbringer e Silva, S. C., & de Sousa, R. M. (2007). Galveston Orientation and Amnesia Test: Applicability and relation with the Glasgow Coma Scale. *Revista Latino-Americana de Enfermagem, 15*(4), 651–657.

Gage, F. H., Kempermann, G., Palmer, T. D., Peterson, D. A., & Ray, J. (1998). Multipotent progenitor cells in the adult dentate gyrus. *Neurobiology, 36*, 249–266.

Ghigo, E., Masel, B., Aimaretti, G., et al. (2005). Consensus guidelines on screening for hypopituitarism following traumatic brain injury. *Brain Injury, 19*(9), 711–724.

Giacino, J. T., Kalmar, K., & Whyte, J. (2004). The JFK Coma Recovery Scale-Revised: Measurement characteristics and diagnostic utility. *Archives of Physical Medicine and Rehabilitation, 85*(12), 2020–2029.

Gil, J. A., Waryasz, G. R., Klyce, W., & Daniels, A. H. (2015). Heterotopic ossification in neurorehabilitation. *Rhode Island Medical Journal, 98*(12), 32–34.

Gomez, P. A., Lobato, R. D., Boto, G. R., De la Lama, A., Gonzalez, P. J., & de la Cruz, J. (2000). Age and outcome after severe head injury. *Acta Neurochirurgica (Wien), 142*(4), 373–380; discussion 380–381.

Gordon, W. A., Hibbard, M. R., Brown, M., Flanagan, S., & Korves, M. C. (1999). Community integration and quality of life of individuals with traumatic brain injury. In M. Rosenthal, E. R. Griffith, J. S. Kreuztzer, & B. Pentland (Eds.), *Rehabilitation of the adult and child with traumatic brain injury* (pp. 312–325). Philadelphia, PA: F.A. Davis Company.

Gudeman, S. K., Kishore, P. R., Becker, D. P., et al. (1981). Computed tomography in the evaluation of incidence and significance of post-traumatic hydrocephalus. *Radiology, 141*(2), 397–402.

Harrison-Felix, C., Pretz, C., Hammond, F. M., Cuthbert, J.P., Bell, J, Corrigan J,...Haarbauer-Krupa, J. (2015). Life expectancy after inpatient rehabilitation for traumatic brain injury in the United States. *Journal of Neurotrauma, 32*(23), 1893–1901.

Hattori, N., Huang, S. C., Wu, H. M., Yeh, E., Glenn, T.C., Vespa, P.M.,...Bergsneider, M. (2003). Correlation of regional metabolic rates of glucose with Glasgow Coma Scale after traumatic brain injury. *Journal of Nuclear Medicine, 44*(11), 1709–1716.

Healey, C., Osler, T. M., Rogers, F. B., Healey, M.A., Glance, L.G., Kilgo, P.D.,...Meredith, J.W. (2003). Improving the Glasgow Coma Scale score: Motor score alone is a better predictor. *The Journal of Trauma, 54*(4), 671–678.

Health Topics. (2018). *Disabilities*. World Health Organization. Retrieved January, 2018, from http://www.who.int/topics/disabilities/en/

Hendricks, H. T., Geurts, A. C., van Ginneken, B. C., Heeren, A. J., & Vos, P. E. (2007). Brain injury severity and autonomic dysregulation accurately predict heterotopic ossification in patients with traumatic brain injury. *Clinical Rehabilitation, 21*(6), 545–553.

Hentze, H., Graichen, R., & Colman, A. (2006). Cell therapy and the safety of embroyic stem cell-derived grafts. *Trends in Biotechnology, 25*(1), 24–32.

Hinterberger, T., Schmidt, S., Neumann, N., Mellinger, J., Blankertz, B., Curio, G., & Birbaumer, N. (2004). Brain-computer communication and slow cortical potentials. *IEEE Transactions on Biomedical Engineering, 51*(6), 1011–1018.

Huijben, J. A., van der Jagt, M., Cnossen, M. C., Kruip, M.J.H.A, Haitsma, I.K., Stocchetti, N.,... Lingsma, H.F. (2017). Variation in blood transfusion and coagulation management in traumatic brain injury at the intensive care unit: A survey in 66 neurotrauma centers participating in the Collaborative European NeuroTrauma Effectiveness Research in Traumatic Brain Injury Study. *Journal of Neurotrauma*. https://doi.org/10.1089/neu.2017.5194

Hukkelhoven, C. W., Steyerberg, E. W., Rampen, A. J., Farace, E., Habbema, J.D., Marshall, L.F.,...Maas, A.I. (2003). Patient age and outcome following severe traumatic brain injury: An analysis of 5600 patients. *Journal of Neurosurgery, 99*(4), 666–673.

Kartal, M. G., & Algin, O. (2014). Evaluation of hydrocephalus and other cerebrospinal fluid disorders with MRI: An update. *Insights Imaging, 5*(4), 531–541.

Katz, D. I., & Alexander, M. P. (1994). Traumatic brain injury. Predicting course of recovery and outcome for patients admitted to rehabilitation. *Archives of Neurology, 51*(7), 661–670.

Katz, R. T., Dewald, J. P. A., & Schmit, B. D. (2000). Spasticity. In R. L. Braddom (Ed.), *Physical medicine and rehabilitation* (pp. 592–615). Philadelphia, PA: WB Saunders.

Kelly, D. F., Gonzalo, I. T., Cohan, P., Berman, N., Swerdloff, R., & Wang, C. (2000). Hypopituitarism following traumatic brain injury and aneurysmal subarachnoid hemorrhage: A preliminary report. *Journal of Neurosurgery, 93*(5), 743–752.

Kolb, B., & Gibb, R. (2014). Searching for the principles of brain plasticity and behavior. *Cortex, 58*, 251–260.

Kolb, B., Harkar, A., & Gibb, R. (2017). Principles of plasticity in the developing brain. *Developmental Medicine & Child Neurology, 59*, 1218–1223.

Kwakkel, G., Veerbeek, J. M., van Wegen, E. E. H., & Wolf, S. (2015). Constraint-induced movement therapy after stroke. *Lancet Neurology, 14*(2), 224–234.

Lance, J. (1980). Symposium synopsis in spasticity. In R. G. Feldman, R. Young, & W. P. Koella (Eds.), *Disordered motor control* (pp. 485–495). Chicago, IL: Year Book Medical Publishers.

Laver, K. E., George, S., Thomas, S., Deutsch, J. E., & Crotty, M. (2015). Virtual reality for stroke rehabilitation. *Cochrane Database of Systematic Reviews*, (2), CD008349.

Laver, K. E., Lange, B., George, S., Deutsch, J. E., Saposnik, G., & Crotty, M. (2017). Virtual reality for stroke rehabilitation (Review). *Cochrane Database of Systematic Reviews*, (11), CD008349.

Levin, H. S., O'Donnell, V. M., & Grossman, R. G. (1979). The Galveston Orientation and Amnesia Test. A practical scale to assess cognition after head injury. *The Journal of Nervous and Mental Disease, 167*(11), 675–684.

Li, Z., Han, X., Sheng, J., Ma, S. (2016). Virtual reality for improving balance in patients after stroke: A systematic review and meta-analysis. *Clinical Rehabilitation, 30*(5), 432–440.

Lois, C., & Alvarez-Buylla, A. (1993). Proliferating subventricular zone cells in the adult mammalian forebrain can differentiate into neurons and glia. *Proceedings of the National Academy of Sciences of United States of America, 90*, 2074–2077.

Luerssen, T. G., Klauber, M. R., & Marshall, L. F. (1988). Outcome from head injury related to patient's age. A longitudinal prospective study of adult and pediatric head injury. *Journal of Neurosurgery, 68*(3), 409–416.

Luque-Moreno, C., Ferragut-Garcias, A., Rodriguez-Blanco, C., Heredia-Rizo, A. M., Olivia-Pascual-Vaca, J., Kiper, P., & Oliva-Pascual-Vaca, A. (2015). A Decade of Progress using Virtual Reality for Poststroke Lower Extremity Rehabilitation: Systematic Review of Intervention Methods. *BioMed Research International, 2015*, 1–7.

Maas, A. I., Hukkelhoven, C. W., Marshall, L. F., & Steyerberg, E. W. (2005). Prediction of outcome in traumatic brain injury with computed tomographic characteristics: A comparison between the computed tomographic classification and combinations of computed tomographic predictors. *Neurosurgery, 57*(6), 1173–1182; discussion 1173–1182.

Mang, C. S., Campbell, K. L., Ross, C. J. D., & Boyd, L. A. (2013). Promoting neuroplasticity for motor rehabilitation after stroke: Considering the effects of aerobic exercise and genetic variation on brain-derived neurotrophic factor. *Physical Therapy, 93*, 1707–1726.

Mazzini, L., Campini, R., Angelino, E., Rognone, F., Pastore, I., & Oliveri, G. (2003). Posttraumatic hydrocephalus: A clinical, neuroradiologic, and neuropsychologic assessment of long-term outcome. *Archives of Physical Medicine and Rehabilitation, 84*(11), 1637–1641.

McMillan, T. M., Jongen, E. L., & Greenwood, R. J. (1996). Assessment of post-traumatic amnesia after severe closed head injury: Retrospective or prospective? *Journal of Neurology, Neurosurgery, and Psychiatry, 60*(4), 422–427.

Meyfroidt, G., Baguley, I. J., & Menon, D. K. (2017). Paroxysmal sympathetic hyperactivity: The storm after acute brain injury. *Lancet Neurology, 16*(9), 721–729.

Meythaler, J. M., & Kowalski, S. (2011). Pharmacologic management of spasticity: Oral medications. In A. Brashear & E. Elovic (Eds.), *Spasticity diagnosis and management* (pp. 199–227). New York, NY: Demosmedical.

MRC CRASH Trial Collaborators, Perel, P., Arango, M., Clayton, T., Edwards, P., Komolafe, E.,...Yutthakasemsunt, S. (2008). Predicting outcome after traumatic brain injury: Practical prognostic models based on large cohort of international patients. *BMJ, 336*(7641), 425–429.

Nakase-Richardson, R., Sherer, M., Seel, R. T., Hart, T., Hanks, R., Arango-Lasprilla, J.C.,... Hammond, F. (2011). Utility of post-traumatic amnesia in predicting 1-year productivity following traumatic brain injury: Comparison of the Russell and Mississippi PTA classification intervals. *Journal of Neurology, Neurosurgery, and Psychiatry, 82*(5), 494–499.

Nicolas-Alonso, L. F., & Gomez-Gil, J. (2012). Brain computer interfaces, a review. *Sensors, 12*(2), 1211–1279.

Novack, T. A., Dowler, R. N., Bush, B. A., Glen, T., & Schneider, J. J. (2000). Validity of the Orientation Log, relative to the Galveston Orientation and Amnesia Test. *The Journal of Head Trauma Rehabilitation, 15*(3), 957–961.

Ozaki, K., Kagaya, H., Yokoyama, M., Saitoh, E., Okada, S., Gonzalez-Fernandez, M.,...Uematsu, A.H. (2010). The risk of penetration or aspiration during videofluoroscopic examination of swallowing varies depending on food types. *The Tohoku Journal of Experimental Medicine, 220*(1), 41–46.

Page, S. J., Levine, P., & Leonard, A. C. (2005). Modified constraint-induced therapy in acute stroke: A randomized controlled pilot study. *Neurorehabilitation and Neural Repair, 19*, 27–32.

Page, S. J., Levine, P., Sisto, S., Bond, Q., & Johnston, M. V. (2002). Stroke patients' and therapists' opinions of constraint-induced movement therapy. *Clinical Rehabilitation, 16*(1), 55–60.

Perkes, I. E., Menon, D. K., Nott, M. T., & Baguley, I. J. (2011). Paroxysmal sympathetic hyperactivity after acquired brain injury: A review of diagnostic criteria. *Brain Injury, 25*(10), 925–932.

Pfurtscheller, G., Guger, C., Müller, G., Krausz, G., & Neuper, C. (2000). Brain oscillations control hand orthosis in a tetraplegic. *Neuroscience Letters, 292*(3), 211–214.

Powell, J., Heslin, J., & Greenwood, R. (2002). Community based rehabilitation after severe traumatic brain injury: A randomised controlled trial. *Journal of Neurology, Neurosurgery, and Psychiatry, 72*(2), 193–202.

Ranganathan, K., Loder, S., Agarwal, S., Wong, V. W., Forsberg, J., Davis, T. A., Levi, B. (2015). Heterotopic ossification: Basic-science principles and clinical correlates. *The Journal of Bone and Joint Surgery. American Volume, 97*(13), 1101–1111.

Reddy, G. K., Bollam, P., & Caldito, G. (2014). Long-term outcomes of ventriculoperitoneal shunt surgery in patients with hydrocephalus. *World Neurosurgery, 81*(2), 404–410.

Rolfe, A., & Sun, D. (2015). Stem cell therapy in brain trauma, implications for repair and regeneration of injured brain in experimental TBI models; Chapter 42. In F. H. Kobeissy (Ed.), *Brain neurotrauma: Molecular, neuropsychological, and rehabilitation aspects*. Boca Raton, FL: CRC Press/Taylor & Francis.

Roth, E. J., & Harvey, R. L. (2000). Rehabilitation of stroke syndromes. In R. L. Braddom (Ed.), *Physical medicine and rehabilitation* (pp. 1117–1160). Philadelphia, PA: WB Saunders.

Salazar, A. M., Warden, D. L., Schwab, K., Spector, J., Braverman, S., Walter, J.,...Ellenbogen, R.G. (2000). Cognitive rehabilitation for traumatic brain injury: A randomized trial. Defense and Veterans Head Injury Program (DVHIP) Study Group. *JAMA, 283*(23), 3075–3081.

Saulino, M., Yablon, S. A., Moberg-Wolff, E., Chow, J. W., Stokic, D. S. (2011). Intrathecal baclofen for spasticity. In A. Brashear & E. Elovic (Eds.), *Spasticity diagnosis and management* (pp. 229–239). New York, NY: Demosmedical.

Semlyen, J. K., Summers, S. J., & Barnes, M. P. (1998). Traumatic brain injury: Efficacy of multidisciplinary rehabilitation. *Archieves of Physical Medicine and Rehabilitation, 79*(6), 678–683.

Shiel, A., Burn, J. P., Henry, D., Clark, J., Wilson, B. A., Burnett, M. E., & McLellan, D. L. (2001). The effects of increased rehabilitation therapy after brain injury: Results of a prospective controlled trial. *Clinical Rehabilitation, 15*(5), 501–514.

Shih, J. J., Krusienski, D. J., & Wolpaw, J. R. (2012). Brain-computer interfaces in medicine. *Mayo Clinic Proceedings, 87*(3), 268–279.

Shimizu, E., Hashimoto, K., & Iyo, M. (2004). Ethnic difference of the BDNF 196G/A (val66met) polymorphism frequencies: The possibility to explain ethnic mental traits. *American Journal of Medical Genetics. Part B, Neuropsychiatric Genetics, 126*, 122–123.

Shingleton, R., Kinzinger, J. H., & Elovic, E. (2011). The role of physical and occupational therapy in the evaluation and management of spasticity. In A. Brashear & E. Elovic (Eds.), *Spasticity diagnosis and management* (pp. 155–182). New York, NY: Demosmedical.

Sirtori, V., Corbetta, D., Moja, L., & Gatti, R. (2009). Constraint-induced movement therapy for upper extremities in stroke patients. *Cochrane Database of Systematic Reviews*, (4), CD004433.

Skrifvars, M. B., Bailey, M., Presneill, J., French, C., Nichol, A., Little, L.,...Bellomo, R. (2017). Venous thromboembolic events in critically ill traumatic brain injury patients. *Intensive Care Medicine, 43*(3), 419–428.

Slade, A., Tennant, A., & Chamberlain, M. A. (2002). A randomised controlled trial to determine the effect of intensity of therapy upon length of stay in a neurological rehabilitation setting. *Journal of Rehabilitation Medicine, 34*(6), 260–266.

Smania, N., Gandolfi, M., Paolucci, S., Iosa, M., Ianes, P., Recchia, S.,...Farina, S. (2012). Reduced-intensity modified constraint induced movement therapy versus conventional therapy for upper extremity rehabilitation after stroke: A multicenter trial. *Neurorehabilitation and Neural Repair, 26*(9), 1035–1045.

Stiens, S. A., O'Young, B., & Young, M. A., II. (2002). Person-centered rehabilitation: Interdisciplinary intervention to enhance patient enablement. In B. J. O'Young, M. A. Young, & S. A. Stiens (Eds.), *Physical medicine and rehabilitation secrets* (pp. 4–9). Philadelphia, PA: Hanley & Belfus.

Sun, D. (2014). The potential of endogenous neurogenesis for brain repair and regeneration following traumatic brain injury. *Neural Regeneration Research, 9*(7), 688–692.

Tanaka, K., Matsunaga, K., & Wang, H. O. (2005). Electroencephalogram-based control of an electric wheelchair. *IEEE Transactions on Robotics, 21*(4), 762–766.

Tate, R. L., Pfaff, A., Baguley, I. J., Marosszeky, J.E., Gurka, J.A., Hodgkinson, A.E.,...Hanna, J. (2006). A multicentre, randomised trial examining the effect of test procedures measuring emergence from post-traumatic amnesia. *Journal of Neurology, Neurosurgery, and Psychiatry, 77*(7), 841–849.

Taub, E. (1980). Somatosensory deafferentation research with monkeys: Implications for rehabilitation medicine. In L. P. Ince (Ed.), *Behavioral psychology in rehabilitation medicine: Clinical applications* (pp. 371–401). New York, NY: Williams & Wilkins.

Taub, E., Uswatte, G., King, D. K., Morris, D., Crago, J. E., & Chatterjee, A. (2006). A placebo-controlled trial of constraint-induced movement therapy for upper extremity after stroke. *Stroke, 37*, 1045–1049.

Teasdale, G., Maas, A., Lecky, F., Manley, G., Stocchetti, N., & Murray, G. (2014). The Glasgow Coma Scale at 40 years: Standing the test of time. *Lancet Neurology, 13*(8), 844–854.

Temkin, N. R., Dikmen, S. S., Wilensky, A. J., Keihm, J., Chabal, S., & Winn, H. R. (1990). A randomized, double-blind study of phenytoin for the prevention of post-traumatic seizures. *The New England Journal of Medicine, 323*(8), 497–502.

Tsyben, A., Guilfoyle, M., Timofeev, I., Anwar, F., Allanson, J., Outtrim, J.,...Helmy, A. (2017). Spectrum of outcomes following traumatic brain injury-relationship between functional impairment and health-related quality of life. *Acta Neurochirurgica (Wien), 160*(1), 107–115.

Turner-Stokes, L., Pick, A., Nair, A., Disler, P. B., & Wade, D. T. (2015). Multi-disciplinary rehabilitation for acquired brain injury in adults of working age. *Cochrane Database of Systematic Reviews*, (12), CD004170.

Viana, R., & Teasell, R. (2012). Barriers to the implementation of constraint-induced movement therapy into practice. *Topics in Stroke Rehabilitation, 19*(2), 104–114.

Weiss, P., Kizony, R., Feintuch, U., & Katz, N. (2006). Virtual reality in neurorehabilitation. In M. Selzer, L. Cohen, F. Gage, S. Clarke, & P. Duncan (Eds.), *Textbook of neural repair and rehabilitation* (pp. 182–197). Cambridge: Cambridge University Press.

Whyte, J., Hart, T., Laborde, A., & Rosenthal, M. (1998). Rehabilitation of the patient with traumatic brain injury. In J. A. Delisa & B. M. Gans (Eds.), *Rehabilitation medicine: Principles and practice* (pp. 1191–1239). Philadelphia, PA: Lippincott-Raven.

Wolf, S. L., Winstein, C. J., Miller, J. P., Taub, E., Uswatte, G., Morris, D., … Nichols-Larsen, D. (2006). Effect of Constraint-Induced movement therapy on upper extremity function 3 to 9 months after stroke. The EXCITE Randomized clinical trial. *JAMA, 296*(17), 2095–2104.

Wu, H. M., Huang, S. C., Hattori, N., et al. (2004). Selective metabolic reduction in gray matter acutely following human traumatic brain injury. *Journal of Neurotrauma, 21*(2), 149–161.

Zafar, S. N., Khan, A. A., Ghauri, A. A., & Shamim, M. S. (2012). Phenytoin versus Leviteracetam for seizure prophylaxis after brain injury - A meta analysis. *BMC Neurology, 12*, 30.

Zhu, X. L., Poon, W. S., Chan, C. C. H., & Chan, S. S. H. (2007). Does intensive rehabilitation improve the functional outcome of patients with traumatic brain injury (TBI)? A randomized controlled trial. *Brain Injury, 21*(7), 681–690.

Chapter 5
Practical Review of Robotics in the Treatment of Chronic Impairment After Acquired Brain Injury

Johanna L. Chang, Maira Saul, and Bruce T. Volpe

Why Robots? Basic Principles and Benefits of Robotic Interventions

In 1996, Nudo, Wise, SiFuentes, and Miliken published a pivotal preclinical neurological recovery study, which demonstrated that activity-dependent plasticity underlies motor recovery following brain injury. Two separate groups of primates underwent intracortical microstimulation (ICMS) to confirm the region of the motor cortex that was responsible for fine motor control. Both groups were then subjected to cortical injury, causing specific unilateral hemiparesis of the wrist and the hand. Following the neurological insult, the first group was allowed to recover without any further intervention. They exhibited minimal recovery of function, and repeat ICMS mapping revealed an extension of the brain lesion into previously undamaged, adjacent motor areas. The second group was provided intensive fine motor training involving retrieval of food pellets from small wells. Not only did the trained group exhibit recovery of function, but repeat ICMS mapping revealed that they had no further loss of neural tissue and, in some cases, had increased representations for hand movements expanding into regions of the motor cortex, which were originally responsible for shoulder/elbow control (Nudo et al., 1996). This study provided important evidence for activity-dependent neuroplasticity: the idea that intensive motor training can improve motor function and correspondingly reorganize cortical motor representations following ABI.

Results from early motor learning research highlighted key tenets of successful motor rehabilitation and drove the development of interactive robotic therapy devices. Effective robotic devices share several important characteristics; they are

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impairment focused, and they deliver ***reproducible***, ***objective***, and most importantly ***intensive*** and ***adaptive*** motor training (Krebs et al., 2004, 2007). Robotic therapy devices typically operate through a series of *interactive* motors with low impedance, which are ***adaptive***: the robot moves a patient's limb when the patient cannot move and progressively intervenes less as the patient improves and regains more voluntary movement. The assistance provided is no different from active-assistive range of motion exercises performed in physical or occupational therapy (Volpe et al., 2008). However, a robot can typically provide more ***intensive*** active-assistive intervention than a therapist, as the robot does not get tired or need a rest or break, it becomes a durable tool for therapists in any restorative clinic. For example, a single robotic therapy session will deliver over a thousand movement repetitions in an hour. In comparison, a recent study of 312 inpatient and outpatient sessions across seven rehabilitation sites demonstrated that a standard OT and PT treatment for individuals with stroke averaged only 32 repetitions of a given movement per session (Lang et al., 2009). Intensity of repetitions both within and across sessions has been shown to improve overall motor outcomes following acute and chronic brain injuries (Cramer, 2018).

Another key element of robotic therapy is that it is ***impairment focused***: each device trains a specific limb segment or segments in isolation, and allows the patient to focus on regaining motor control for a singular area of weakness such as the wrist, hand, shoulder/elbow, or ankle. The advantage of isolated joint(s) training is that it is safe, because a patient can typically sit during the intervention, and it is also easily ***reproducible***, allowing for quick setup, reliable dosing, and more ***objective*** measurement of improvements. Subjective clinical scales tend capture broad functional changes, whereas objective robotic measures are consistently precise and sensitive to small improvements. Nearly all commercially available devices today incorporate an online or intermittent "report card," which automatically tracks patient progress, providing the clinician efficient and unbiased data for justification of services, and providing the patient motivation to "beat" his or her previous score.

Overall, the most significant benefit of robotic intervention is its efficiency. A robot can efficiently deliver intensive repetitions of motor training at patient-specific areas of impairment, enabling the PT or OT to then incorporate those motor improvements into functional training for ADLs.

Efficacy: A Current Evidence Base for Robotic Intervention

The critical multicenter study providing an evidence base for upper limb robotic rehabilitation was executed by Lo and Colleagues (2010). A total of 127 individuals with chronic moderate-to-severe upper limb hemiparesis following stroke were randomized to three treatment groups: intensive robot-assisted therapy ($n = 49$), intensive comparison therapy ($n = 50$), and usual care ($n = 28$). Both the robot-assisted and intensive comparison therapy group received thirty-six 1-h sessions (3×/week for 12 weeks) of intensity-matched intervention, with the latter group performing all movements with the active assistance of a clinician instead of the robot. This active,

intensive therapist delivered treatment was verified as nearly comparable to the number of movements executed by the robot in a previous study (Volpe et al., 2008). The usual care group received standard medical and/or outpatient rehabilitation services, which were not intensity-matched. At 6-month follow-up (week 36 of study), the robot group and intensive comparison therapy group both improved significantly compared with usual care (average of ~3 points on the Fugl Meyer Upper Extremity Scale). Moreover, younger participants (<55 years) and participants who were a shorter time post-stroke (<12 months) exhibited the greatest improvements (average of 7–16 points on the Fugl Meyer) (Wu, Guarino, Lo, Peduzzi, & Wininger, 2016). Again, this study demonstrated that robotic devices do not inherently provide an intervention that a clinician cannot, but the robot exceeds at delivering intensive motor repetitions at a level that is impractical for a clinician to perform regularly. Overall, the take-home message was clear: intensive active-assistive motor intervention significantly boosts long-term functional recovery.

Subsequent cost–benefit analysis following this multicenter VA study found that at 36 weeks (6 month follow up appointment), individuals who received robotic intervention had actually cost the VA health system significantly less (over $1000 per patient) than usual care or intensive comparison therapy (Wagner et al., 2011). Even though robotic therapy had added costs to administer, the patients who received it ultimately used less resources throughout the health system following the intervention, thereby reducing the overall financial burden. Following the Lo et al. study, the American Heart Association recommended in favor of robotic therapy (level IIa) as a "reasonable" form of upper extremity stroke rehabilitation (Winstein et al., 2016).

There has been less rigorous investigation of robotics for the lower extremity, though a recent meta-analysis across 36 studies of electromechanical gait rehabilitation found that patients who received physical therapy in combination with these devices were more likely to walk independently, with the most benefit for clients who were nonambulatory and in the acute phase after stroke (Mehrholz et al., 2017). Comparatively, the LEAPS multicenter study employed locomotor training exercise and exoskeletal body-weight-supported treadmill training and found this intervention was comparable but not superior to progressive exercise at home managed by an expert therapist (Duncan et al., 2011). At present, the American Heart Association has assigned lower extremity robotic therapy with the slightly lower level IIB designation (Winstein et al., 2016). This means lower extremity robotics can be considered as an intervention, though patient candidacy and careful documentation of improvement should be tracked.

Rehabilitation Devices on the Marketplace

Over the past 20 years, there has been an explosion of commercially available rehabilitation devices in the marketplace for the treatment of hemiparesis following ABI. While this is an exciting time in rehabilitation medicine where technology is integrated to improve clinical care, it is critical to remember that not all

interventions are created equally. It is important that the clinician trial any new device in order to understand the type and level of assistance it provides, carefully review its evidence base, and develop an impairment-focused treatment rationale.

For the upper extremity, there are a multitude of rehabilitation devices, which vary widely based upon the types and levels of assistance they offer. The four main categories of interventions provided by rehabilitation devices are robotic, mechanical, electrical stimulation (e-stim), and biofeedback. While the primary focus of this chapter is on *robotic* rehabilitation devices and their evidence base, it is also important to briefly review mechanical, electrical stimulation, and biofeedback devices, as these are many of the commercially available interventional solutions in the market at present. Robotic devices such as the InMotion Arm™ provide motorized assistance to the limb as it engages in a movement, whereas mechanical devices such as the Saeboflex™ provide spring-loaded support. E-stim devices such as the Bioness™ deliver an electrical current to engage the muscle during the movement. In contrast, biofeedback devices such as MusicGlove™ provide only visual feedback to engage the patient to increase his or her own movement without direct assistance (Table 5.1). These devices also vary based on the level of assistance they offer from total passive ROM where the limb is taken through the movement without any required engagement of the patient, to active-assistive, which requires the patient to attempt to move and the device provides assistance-as-needed, to fully active ROM, where the patient is provided visual feedback of performance and encouraged to move independently. Many devices can provide a range of assistance, which is adjusted by the clinician or the patient with some training.

Generally, active-assistive devices employing either robotic support or electrical stimulation are superior for motor training, because they require continued attention from the patient to attempt to execute the motor task, while delivering interventional support as needed to complete the movement with the targeted muscle complex. Active attention and task-engagement are two components that are critical to motor learning (Antelis, Montesano, Giralt, Casals, & Minguez, 2012). In contrast, passive range of motion settings are beneficial to initially stretch a targeted muscle group or promote overall joint health, but do not directly assist in motor training (Hogan et al., 2006). The InMotion Arm™ and InMotion Wrist™ robots were some of the earliest robotic active-assistive devices to the marketplace and have the largest published evidence base across all upper extremity devices (Table 5.1). One of the important features that differentiates these devices from others is that they are "backdrivable": the device allows the patient to make an attempt *before* it provides any assistance (Hogan et al., 2006). Many other active-assistive range of motion devices provide a programmable percentage of assistance throughout the movement, which must be adjusted by the clinician, and they do not pause for the patient to attempt to move totally independently before providing support. At this time, there is not definitive clinical evidence in favor of one or the other approach. Nonetheless, there are many new and promising upper extremity device companies with emerging efficacy evidence.

One important gap in rehabilitation technology is the development of a consistent solution for the recovery of hand function, which is one of the most coveted

Table 5.1 Summary table of commercially available upper extremity rehabilitation devices (Lo, Stephenson, & Lockwood, 2017; Maciejasz, Eschweiler, Gerlach-Hahn, Jansen-Troy, & Leonhardt, 2014; McConnell et al., 2017; Poli, Morone, Rosati, & Masiero, 2013; Yue, Zhang, & Wang, 2017)

Device	Assistive technology	Mechanical design	Training modalities	Treatment target	Task description	Manufacturer
InMotion Arm	Robotic	End-effector	Passive/ active-assistive/ active/resistive	Shoulder and elbow	Desk-mounted, seated shoulder/elbow training device with back-drivable active-assistance integrated into a video game; for clinical use only	Bionik
InMotion Hand	Robotic	End-effector	Passive/ active-assistive/ active/resistive	Hand	Desk-mounted, seated, shoulder/elbow/grip training device with back-drivable active assistance integrated into a video game; for clinical use only	Bionik
InMotion Wrist	Robotic	End-effector	Passive/ active-assistive/ active/resistive	Wrist	Seated wrist training device with back-drivable active-assistance integrated into a video game; for clinical use only	Bionik
Hand of Hope	Robotic + biofeedback	Exoskeleton	Passive/ active-assistive/ active	Hand and fingers	Wearable surface EMG-driven robotic hand therapy device for isolated training or integrated with video games; for clinical use	Rehab-Robotics
Amadeo	Robotic	End-effector	Passive/ active-assistive/ active	Hand and fingers	Desk-mounted, seated finger movement and dexterity training device with active-assistance integrated into a video game; for clinical use only with higher functioning patients	Tyromotion
Diego	Robotic	End-effector	Passive/ active-assistive/ active	Arm and shoulder	Seated shoulder/elbow gravity compensation training device integrated into video game; for clinical use only	Tyromotion
Pablo	Biofeedback	End-effector	Active	Hand and arm	Wrist/hand/arm therapy and assessment device that facilitates independent movement with biofeedback; for clinical and home use	Tyromotion
Hand Mentor Pro	Robotic	Exoskeleton	Active-assistive	Hand	Wearable robotic hand device with guided and controlled interactive game based-training; for clinical use	Motus Nova

(continued)

Table 5.1 (continued)

Device	Assistive technology	Mechanical design	Training modalities	Treatment target	Task description	Manufacturer
Saebo Glove	Mechanical	Exoskeleton	Active-assistive/ active	Hand and fingers	Wearable, bungee-loaded hand device promoting active finger extension and training functional grasp; for clinical and home use	Saebo
Hand Tutor	Biofeedback	Exoskeleton	Active	Hand and wrist	Wearable hand device with integrated video display, facilitating independent hand function with biofeedback; for clinical and home use	MediTouch
SaeboFlex	Mechanical	Exoskeleton	Active-assistive/ active/resistive	Hand and fingers	Wearable, spring-loaded hand device promoting active finger extension and training functional grasp; for clinical and home use	Saebo
ExoHand	Robotic	Exoskeleton	Active-assistive/ active	Hand and fingers	Wearable hand device used to perform computer-based tasks and training of gross motor ADL; for clinical and home use	Festo
H200 Wireless Hand	Electrical stimulation	Exoskeleton	Passive/ active-assisted	Hand and wrist	Wearable hand orthosis providing Functional Electrical Stimulation to activate muscles of wrist and hand to facilitate hand function and prevent contractures; for clinical and home use	Bioness
Music Glove	Biofeedback	Exoskeleton	Active	Hand and fingers	Wearable hand device for independent movement with biofeedback during interactive music guided task; for clinical and home use	Flint Rehab
Armeo	Robotic	End-effector	Passive/ active-assistive/ active	Upper extremity	Seated shoulder/elbow training device with range of assistance during video game tasks; for clinical use only	Hocoma
MyoPro Orthosis	Electrical stimulation + robotic	Exoskeleton	Active-assistive/ active	Elbow, wrist and hand	Myoelectric elbow/wrist/hand orthosis that supports and enables controlled function of impaired hand and arm; for clinical and home use during ADLs	Myomo

Rapael Smart Glove	Biofeedback	Exoskeleton	Active	Hand and fingers	Wearable glove with biofeedback via integrated video display for customized hand rehabilitation; for clinical and home use	Neofact
Neomanus	Robotic	Exoskeleton	Active-assistive/ active	Hand and fingers	Wearable hand robot designed to assist with ADL training and integration of function; for clinical and home use	Neofact
Gloreha Sinfonia	Robotic	Exoskeleton	Passive/ active-assistive/ active	Hand, fingers and wrist	Robotic glove enriched by a multisensory stimulation and 3D animation that supports finger joints motion, while detecting voluntary active motion. Allows ADL training; for clinical use	REHA Technology
Armotion	Robotic	End-effector	Passive/ active-assistive/ active	Upper extremity	Portable Arm therapy device with computer-based gaming with visual feedback; for clinical and home use	REHA Technology

goals for patients after ABI. Part of the challenge is that the hand is made up of an inherently complex set of muscles with many degrees of freedom, allowing for the execution of specified, dexterous movements. The other challenge is that following a stroke, the hand typically presents with the most severe muscle spasticity. Consequently, it is difficult to create an interventional device with enough structure to prevent the hand from assuming a flexed posture, and enough fluidity to allow the degrees of movement freedom required for the articulation of the digits or apposition of the thumb during functional tasks.

Shoulder and elbow improvements from robotic training are mostly achieved by active-assistive and resistance-based tasks, but weakness and high tone of the hand impose challenges to the selection of patients that could benefit from any one product currently available in the market. Presently, clinicians should carefully review all available hand rehabilitation devices and select a device that fits with the impairment level of the patient. Generally, hand rehabilitation devices should not be introduced until the patient has enough proximal control of the shoulder and elbow for reaching tasks. At that time, patients with more severe difficulties might try a device that focuses on gross finger extension with a more fixed, structured spine to keep the hand in place during movement, such as Saeboflex™. For higher functioning patients with mild residual spasticity and partial finger extension, consider a device that focuses on finer finger movements with a less restrictive orthosis such as the RAPAEL Smart Glove™, Gloreha Sinfonia™, or the AMADEO™ (Table 5.1). Research and premarket testing of newer, improved hand devices remains ongoing, promising better solutions for functional hand use across the continuum of recovery.

For the lower extremity, the majority of the technology-based rehab interventions are known as electromechanical gait devices. These large, body weight-supported gait training devices can be divided into two categories: exoskeletal and end-effector. Exoskeletal devices, such as the EKSO™, ReWalk™, and Lokomat™, look like wearable robotic legs and function through programmable drives that move the knees and hips during gait phases. In contrast, end-effector devices, such as the G-EO System™ and the LokoHelp™, provide harness support while holding the feet on foot-plates that move in trajectories mimicking the stance and swing of gait (Table 5.2). Nearly all electromechanical gait devices can be programmed to provide more or less device assistance as needed in order to tailor therapy to the level of impairment. Other lower extremity devices in the market include the InMotion ANKLE™, which is an active-assistive robotic ankle therapy device used in a seated position, and programmable electrical stimulation gait devices such as the WalkAide™ and the Bioness™ L300™, which are worn in place of an orthosis to remediate foot drop during everyday gait function (Table 5.2). There is less overall data on lower extremity devices as a whole, and the largest single study of an exoskeletal device involving comparison of the Lokomat™ training to a home exercise program managed by a physical therapist found no additional benefit from device use (Duncan et al., 2011). However, as mentioned previously, a recent meta-analyses of electromechanical gait intervention across 36 trials with a total of 1472

Table 5.2 Summary table of commercially available lower extremity rehabilitation devices (Lo et al., 2017; Poli et al., 2013)

Device	Assistive technology	Mechanical design	Training modalities	Treatment target	Task description	Manufacturer
GEO System	Robotic	End-effector	Passive/ active-assistive/ active	Lower extremity	Body weight-supported gait trainer for simulated overground walking and step climbing prompted by video display	REHA Technology
NexStep	Robotic	End-effector	Passive/ active-assistive/ active	Lower extremity	Body weight-supported gait trainer for simulated overground walking	REHA Technology
Lokomat	Robotic	Exoskeleton	Passive/ active-assist/ active	Lower extremity	Treadmill-based body weight support gait trainer with assist-as-needed properties	Hocoma
LOPES	Robotic	Exoskeleton	Active-assistive/active	Lower extremity	Treadmill-based body weight support gait trainer with assist-as-needed properties	University of Twente
InMotion Ankle	Robotic	Exoskeleton	Active-assistive/active	Ankle	Seated isolated ankle-training device prompted by video display	Bionik
Gait Trainer GT 1	Robotic	End-effector	Active-assistive	Lower extremity	Body weight-supported robotic gait trainer system. Allows FES integration	Reha-stim
LokoHelp	Robotic	End-effector	Active-assistive/active	Lower extremity	Body weight-supported gait trainer and postural control device	Woodway
WalkAide	Electrical stimulation	Wearable	Active-assistive	Ankle	FES-triggered device targeting muscles involved in gait as an alternative to an AFO	Innovative Neurotronics
L300 Go	Electrical stimulation	Wearable	Active-assistive	Ankle, ankle + knee	FES-triggered device targeting muscles involved in gait as an alternative to an AFO	Bioness
Ekso GT	Robotic	Exoskeleton	Active-assistive/active	Lower extremity	Overground wearable dynamic robotic gait trainer for use in the clinic	Bionic
ReWalk	Robotic	Exoskeleton	Active-assistive/active	Lower extremity	Overground wearable dynamic robotic gait trainer for use in the clinic and out in the community	ReWalk Robotics

patients found that these devices in combination with physical therapy significantly improved functional ambulation status for individuals in the acute phase after stroke who were nonambulatory (Winstein et al., 2016).

Client Selection: Tips for Treating Clinicians and the Interdisciplinary Team

As a whole, robotic therapy is suitable for most patients seeking motor rehabilitation following ABI. As with all rehabilitation interventions, the client should be medically cleared for exercise, and may be excluded for complications such as cardiac failure, severe pain management issues, or recent orthopedic injury/surgery to the targeted joint. Additionally, clients who are unable to remain engaged in a robotic task for at least 45 min to 1 h or who present with total flaccid paralysis, often accompanied by near-complete sensory loss, or fixed contractures of the affected muscle complex should be excluded. More specific candidacy requirements are device dependent.

If there is a question of client candidacy and the intervention is relatively safe, such as a device used in a seated position, it is often worthwhile to trial the treatment, but with vigilant monitoring by the clinician. For example, we have noticed anecdotally that clients who present with receptive language deficits, neglect, and/or inattention (such as global aphasia and hemiparesis) may excel with seated, active-assistive robotic intervention involving a video game/visual display, because it provides a repetitive, goal-directed, nonverbal task, which can be intuited by the patient over time. It may require 2–3 sessions for the patient to initially engage in the task, but generally after this period, the patient becomes increasingly alert, more oriented to field of neglect, and actively participates using the hemiparetic limb. Similarly, for patients with significant shoulder dislocation, the OT may opt to trial a proximal upper extremity robotic device in a seated position, but should cautiously monitor shoulder positioning, provide adequate support for glenohumeral stability during exercise, and carefully assess the patient for report of pain or increasing edema. When carefully controlled, such shoulder intervention can successfully reduce shoulder subluxation, a problem which is often persistent, painful, and challenging for the clinician to address. In one such study with a robotic device, clients with chronic shoulder subluxation underwent 18 robotic sessions of targeted active-assistive training of the shoulder complex, and demonstrated an average reduction of glenohumeral instability of 36.4 mm, or about two finger breadths, at study follow-up (Dohle, Rykman, Chang, & Volpe, 2013).

Another important consideration when weighing the particular costs and benefits of a robotic intervention is setup time. Ideally, setup should not take more than 5–10 min to allow for a rigorous training session for the patient within what is often a tight clinical schedule for the therapist. In general, seated upper extremity robotic devices require limited setup and are well-suited for a fast-moving clinical context.

Some devices such as the MyoPro™ and the EKSO™ require a longer initial fitting/training, but save and retrieve individualized patient settings to be used for future treatments (Tables 5.1 and 5.2). Electromechanical devices involving bodyweight supported harnesses require longer setup and, based upon the severity level of the client, may necessitate the assistance of two clinicians to operate. Consequently, when a treatment team is testing these devices, there is a need to demonstrate improved efficiency of motor recovery to account for the time commitment and clinical resources. Since these devices have the most evidence for use in nonambulatory patients in the acute phase, the PT may consider trialing electromechanical gait intervention with this population, and plan for the commitment of at least two therapists and for lengthier treatment sessions initially (Mehrholz, Pohl, Platz, Kugler, & Elsner, 2015). If used with higher functioning clients that can ambulate, the PT should use his or her clinical judgment to select potential candidates and carefully track progress with repeated gait measures, as there is presently less clinical evidence for treatment with this group as a whole.

The great potential for robotics is to use them as a clinical tool, which augments the benefits of traditional rehabilitation and increases the efficiency of functional recovery. Training with rehabilitation devices can reduce pain and improve active range of motion at specific joints, which accordingly allows the clinician to reduce assistive devices/braces, train the patient/caregiver on correspondingly progressive stretch protocols for home, and, most importantly, integrate motor improvements to increasingly functional tasks. For chronic ABI populations where progress in standard OT/PT programs may plateau, there are evidence-based studies that robotic therapy may still provide additional functional benefits (Lo et al., 2010; Volpe et al., 2009). There is thus tremendous potential for the development of robotic outpatient gyms to be used in restorative care with some clinical oversight. Additionally, a recent uptick in lighter, more affordable home-use devices in the marketplace offers the opportunity to better integrate clinical goals into a home-exercise program both during rehabilitation and after discharge. Training intensity drives motor recovery, but insurance-covered rehabilitation sessions can be limited. Home use devices such as the HandTutor™, Bioness™, and RAPAEL Smart Glove™ enable the therapist to provide the patient with increased opportunities for intensive motor training beyond the clinical schedule at home, and also provide virtual report cards to track patient adherence and progress (Table 5.1). However, it is critical with such devices that the OT train the client and, ideally, the caregiver to monitor for correct device use, as it can be easy to cheat to complete the task, by, for example, hiking in the elbow to move a hand device downward instead of pronating at the wrist.

Overall, the integration of robotic therapy works best as an interdisciplinary team approach. Insights regarding patient medical history, cognitive and communication status, visual perception, sensation, balance, affect, recovery goals, and caregiver support, all contribute to selecting the best interventions for motor recovery. Moreover, input from across the rehabilitation team is needed to monitor functional carryover and best characterize real-world outcomes. The following case report illustrates this point.

Clinical Case Example

CT is a 62 year old woman who sustained a right subcortical CVA resulting in left hemiparesis and dysarthria. Her hospital course included 4 days at a medical center followed by 3 weeks of acute rehabilitation, with subsequent discharge home with 3 additional months of outpatient PT, OT, and ST services. Therapy was complicated by the development of upper extremity neuropathic pain, which reduced her tolerance and engagement in OT.

She enrolled in an upper limb robotic therapy research trial approximately 15 months after her stroke, and nearly a year after discharge from rehabilitation services. At the time of enrollment, her dysarthria had resolved and she walked with a quad cane and an ankle foot orthosis (AFO). She had glenohumeral stability and demonstrated normal shoulder range of motion (ROM), but only partial active ROM at the elbow, wrist, and hand, which was limited by weakness and spasticity. She was able to dress herself, but required assistance with buttons and zippers. She also required minimal assistance with bathing her unaffected side due to pain and weakness in her affected wrist and hand. She complained that her hand movement was not functional, and displayed only partial finger extension/flexion and limited pincer grasp.

After 3 months in the study, consisting of 36 sessions of intensive active-assistive shoulder/elbow and wrist training, she demonstrated a 4-point improvement on the Upper Extremity Fugl Meyer scale, moving from a score of 34 to 38 points. At her 3-month study follow-up, she had another 2-point improvement to a score of 40, for total increase of 6 points. Her improvements were characterized by an increase from partial to full active elbow and finger flexion and increased wrist/finger extension, such that she was able to perform a cylinder grasp for the first time. She reported that she could eat with her affected hand more easily and bathe more independently as she was now able to extend her fingers around a long handled sponge or built-up fork, hold with improved grasp, and flex her elbow enough to bring the fork to her mouth. She also reported that her upper extremity pain was improving with exercise, though still present. Following study discharge, she was referred back to outpatient OT for integration of more functional hand use with the Saeboflex or Bioness. She was also referred to Physiatry for pain management where she received a low dose of gabapentin and reported improving symptoms.

Challenges with Integration of Robotics in Rehab Settings

Despite continued emerging evidence confirming the benefits of robotic rehabilitation interventions, there remain several challenges with the integration of these devices as a standard of care. One challenge is that robotic interventions generally lack specialized billing codes. Nonetheless, many of the top rehabilitation hospitals in the United States do offer robotic therapy, and typically bill for those services under alternative

codes related to the rehabilitation approach rather than the specific device, such as neuromuscular reeducation, attention, and therapeutic activities. Another significant challenge is that many of the most successful clinical trials provide therapy at a higher intensity than is typically covered by insurance. For example, in our own outpatient stroke rehabilitation studies, a typical robotic therapy dosage is eighteen 1-h sessions, 3×/week for 6 weeks (Chang et al., 2017; Dohle et al., 2013). This intensity of intervention is usually only offered in either a research or fee-for-service outpatient setting, as reimbursement does not generally comply. In a standard inpatient or outpatient setting, it is difficult to provide anywhere near this intensity of intervention while still addressing patient's other recovery needs. In practice, when robotic interventions are used as a billable service, they are typically used with less intensity (e.g., fewer repetitions, less visits), and with diminished positive outcome.

It may be that intensity is as important as timing (acute versus chronic) of the intervention. To investigate this hypothesis, we treated 248 functionally diverse inpatients and outpatients who had a Fugl Meyer Upper Extremity Scale score that ranged from 0 to 54 points (out of 66) and who were time post stroke = 5 days–11.3 years from their acute event. Each patient received at least 18 sessions of robotic training for the treatment of upper extremity hemiparesis poststroke. All clients on average had significant changes in upper extremity Fugl Meyer scores; however, we found that baseline severity level bore a strong relationship to predicted dosage of intervention required to achieve maximal response. Clients with moderate-to-severe hemiparesis (average admission FM = 24) generally plateaued after nine treatment sessions, while clients with more mild-to-moderate hemiparesis (average admission FM = 35) improved across eighteen treatment sessions and, in some cases, continued to spontaneously recover hand function during the 3-month follow-up window (Volpe et al., 2009).

As a broad rule of thumb, these data suggest the clinician might expect clinically significant functional improvements (MCID for UE FM = 4 points) after nine sessions of intensive upper extremity robotic therapy, but may continue to document recovery beyond nine sessions, based upon patient severity level and treatment targets (Lundquist & Maribo, 2017). However, if there is no demonstrable benefit by the ninth session, the device may not be beneficial for the client.

Perhaps the greatest challenge for the integration of robotic interventions into rehabilitation medicine is that the efficacy evidence for robotic rehabilitation is based upon a body of clinical research with varied patient characteristics, types of intervention, and intensity/duration information across trials, rendering it difficult to determine which candidate, device, and dosage is needed for best results (Mehrholz et al., 2015). Further motor recovery research is needed to continue to provide more specified device recommendations to clinicians and better service justification evidence to insurers. Rehabilitation research of acquired brain injury is inherently difficult due to the varied nature of the illness. Moreover, many of the accepted measures for capturing improvement are derived from subjective clinical scales that can be unreliable, unless the therapists are formally and rigorously trained. The overwhelming challenge in clinical research is to design a study with a subject pool, which is narrow enough to capture reliable functional improvements,

but diverse enough to be able to generalize the results to the treatment population as a whole.

We attempted to address these challenges in a recent gait study involving 18 sessions of active-assistive ankle training in a seated position to improve functional ambulation status (Chang et al., 2017). Twenty-nine clients with gait speed impairments following stroke were categorized into three functional groups based upon admission comfortable gait speed on the 10-meter walk test, using established gait speed impairment levels with the low function group walking at speed of <0.4 m/s, the moderate function group at 0.4–0.8 m/s, and the high function group at >0.8 m/s (Fritz & Lusardi, 2009). The results indicated three distinct and significant between-group patterns of gait speed recovery. Clients who were at the ceiling for balance measures experienced an impairment level shift in gait speed moving from "community ambulators" (0.8–1.2 m/s) to "normal walking speed" (>1.2 m/s). They also exhibited gait speed improvements that approached significance ($p = 0.051$) from discharge to follow-up on the 10-meter walk fast-paced condition, indicating generalization of functional gains and the development of a fast-paced breakaway speed without further intervention. This suggests that for high functioning patients, isolated ankle training may open a new therapeutic window for further PT intervention to focus on more aggressive functional goals such as running. Clients with moderate-to-severe impairments also had improvements in gait speed and balance, but did not experience an impairment level shift. Comparatively, these patients (e.g., home or limited community ambulators) routinely demonstrate flexor synergy control patterns of the entire lower extremity (hip hike, knee flexion, and foot drop), and may benefit from isolated training of the ankle to target foot drop along with treadmill training of the entire lower limb.

Thus, by using well-established gait speed parameters, we were able to examine patient performance more specifically as a function of baseline severity, and isolate an important subgroup of patients who are able to make an impairment level shift in gait speed from 18 sessions of seated robotic ankle training alone. As demonstrated in this study, intensive robotic interventions may more effectively focus on individualized medicine, which tailors dosage and type of intervention to severity level of the patient. Real-world outcomes are important. By separating patients by severity of impairment, a study will avoid overall failure and specify real-world outcomes that are attainable. Additionally, given the current financial strictures in rehabilitation medicine, future research should also investigate the integration of home-use devices into clinical care in an attempt to increase the intensity of the intervention beyond the limited number of approved treatment sessions.

A typical case history from one of the clients in the study above follows:

Clinical Case Example

KM is a 46-year-old male who sustained a right frontotemporal intracerebral hemorrhage for which he underwent a decompressive craniotomy and subsequent cranioplasty. His hospital course included 2 weeks at an acute medical center;

followed by a month of acute rehabilitation, another 5 weeks in subacute rehabilitation; and finally discharge to home with outpatient PT, OT, and ST services for approximately 3 months. Prior to his stroke, he was a manager at a company and an active tennis player and musician. As per the client and caregiver report, he was initially wheelchair-bound after the stroke, but had progressed to walking with a single-point cane. He had limited recovery of his upper extremity function characterized by persistent weakness and pattern of flexor synergy. He had received ST services to address impairments of planning/judgment and short-term recall. Though these deficits had improved, he had been unable to return to work, but maintained a busy social and family life.

Approximately 4 years after his stroke, he enrolled in a research study involving 6 weeks (18 sessions) of intensive seated anklebot training. At that time, he walked with a single point cane and no AFO, but exhibited mild residual foot drop. Following anklebot training, he had a 4-point improvement on the Berg Balance Scale (moving from a score of 47 to 51 points). He also had an impairment level shift in his gait speed on the 10-meter walk test, moving from a limited to full community ambulator, 0.75–1.0 m/s. He had mild residual foot drop while walking, though he reported improved confidence and gait stability. He consequently gave up the use of his cane and began walking a half mile on his local track several times per week.

Innovation: Noninvasive Electrical Stimulation Techniques

In the past 20 years, there has been a growing interest across the scientific community to explore noninvasive electrical stimulation techniques for the treatment of different neurological conditions. While none of these interventions are currently approved for clinical use in ABI rehabilitation, it is important for the clinician to be aware of these interventions as they are being extensively tested in clinical trials worldwide and have shown some promising efficacy in the treatment of certain conditions. Moreover, there has been a breadth of recent rehabilitation researches in ABI combining noninvasive electrical stimulation with robotic devices and other rehabilitation interventions with the goal of augmenting motor learning/function during a therapy task to improve outcome.

Noninvasive brain stimulation (NIBS) has been shown to modulate brain activity and promote transient improvements in learning and motor function through inhibition or excitation of brain pathways (Ahmed, 2014; Dayan, Censor, Buch, Sandrini, & Cohen, 2013; Hays, 2016; Rossi et al., 2009). Presently, the most investigated noninvasive stimulation devices include Transcranial Magnetic Stimulation (TMS), Transcranial Direct Current Stimulation (tDCS), Transspinal Direct Current Stimulation (tsDCS), and Vagus Nerve Stimulation (VNS). Safety and feasibility studies have demonstrated that NIBS are largely low risk (Hays, 2016; Rossi et al., 2009). TMS has established efficacy data and received FDA approval for the treatment of refractory depression and migraines. However, to date, clinical trials pairing robotics and NIBS have shown mixed and inconsistent results, and comprehensive efficacy reviews of these studies have thus far not established superior

motor recovery benefit (Di Pino et al., 2014). In our experience, we have investigated noninvasive stimulation techniques for use in motor rehabilitation and demonstrated some significant individual treatment responses as referenced in the case example below. Despite this, more research is needed to specify treatment (e.g., target population, timing, dosage) and establish efficacy prior to the introduction of these interventions into clinical practice.

Clinical Case Example: AR

AR was a healthy and thriving 26-year-old college graduate starting his professional life in IT and keeping an important role as a brother and son to a close-knit family. In the spring of 2017, AR awoke with acute-onset right hemiparesis and expressive aphasia. He was taken to an emergency room where an acute cerebrovascular event was diagnosed. After being considered clinically stable, AR was admitted for 3 weeks of acute rehab care where he received ST, OT, and PT services. His lower hemiparesis subsequently resolved, and he was discharged home with outpatient OT and ST services, which targeted improved upper extremity function and verbal expression. Still aiming for an alternative to reduce stiffness and improve hand function, AR consented to participate in a clinical trial involving transspinal direct current stimulation (tsDCS), an experimental device being investigated to reduce the spasticity of the wrist and hand.

After completing the study, AR was encouraged to keep a home-based exercise regimen and integrate bimanual activities into his activities of daily living. He was also enrolled in a 6-week intensive wrist robot program to take advantage of an apparent treatment window opened by the decrease of spasticity achieved post study discharge. Following the study and subsequent robotic intervention, he underwent the same clinical and objective assessments with an exciting outcome: AR had maintained a significant reduction in spasticity below his baseline study scores and showed improvements in active range of motion during the robotic wrist evaluation. Most importantly, he demonstrated increased functional use of hand his hand in everyday activities, including self-feeding, opening doors, and carrying dishes with the affected hand.

Conclusion

Restorative treatments for those with ABI require a multidisciplinary effort, and now, with the addition of bioengineering tools, outcomes over the long term are likely to improve. The goal of restoring the optimum sensorimotor recovery after ABI should be a challenge picked up by the medical community at large, not just the therapists. This will likely require rule changes so that the payment structure can support treatment during the chronic phase of the recovery. Clearly, the evidence basis for these

treatments 6 months or more after the injury supports the rationale for treatment. There is a wealth of information appearing with the use of several of the NIBS devices, and there is great interest and enthusiasm for testing combinations of devices in a new push to help the client express an optimum motor outcome.

References

Ahmed, Z. (2014). Trans-spinal direct current stimulation alters muscle tone in mice with and without spinal cord injury with spasticity. *Journal of Neuroscience, 34*(5), 1701–1709.

Antelis, J. M., Montesano, L., Giralt, X., Casals, A., & Minguez, J. (2012). Detection of movements with attention or distraction to the motor task during robot-assisted passive movements of the upper limb. *IEEE Engineering in Medicine and Biology Society, 2012*, 6410–6413.

Chang, J. L., Lin, R. Y., Saul, M., Koch, P. J., Krebs, H. I., & Volpe, B. T. (2017). Intensive seated robotic training of the ankle in patients with chronic stroke differentially improves gait. *NeuroRehabilitation, 41*(1), 61–68.

Cramer, S. C. (2018). Treatments to promote neural repair after stroke. *Journal of Stroke, 20*(1), 5–70.

Dayan, E., Censor, N., Buch, E. R., Sandrini, M., & Cohen, L. G. (2013). Noninvasive brain stimulation: From physiology to network dynamics and back. *Nature Neuroscience, 16*(7), 838–844.

Di Pino, G., Pellegrino, G., Assenza, G., Capone, F., Ferreri, F., Formica, D., … Di Lazzaro, V. (2014). Modulation of brain plasticity in stroke: A novel model for neurorehabilitation. *Nature Reviews. Neurology, 10*(10), 597–608.

Dohle, C. I., Rykman, A., Chang, J., & Volpe, B. T. (2013). Pilot study of a robotic protocol to treat shoulder subluxation in patients with chronic stroke. *Journal of Neuroengineering and Rehabilitation, 10*, 88.

Duncan, P. W., Sullivan, K. J., Behrman, A. L., Azen, S. P., Wu, S. S., Nadeau, S. E., … LEAPS Investigative Team. (2011). Body-weight-supported treadmill rehabilitation after stroke. *The New England Journal of Medicine, 364*(21), 2026–2036.

Fritz, S., & Lusardi, M. (2009). White paper: Walking speed: The sixth vital sign. *Journal of Geriatric Physical Therapy, 32*(2), 46–49.

Hays, S. A. (2016). Enhancing rehabilitative therapies with vagus nerve stimulation. *Neurotherapeutics, 13*(2), 382–394.

Hogan, N., Krebs, H. I., Rohrer, B., Palazzolo, J. J., Dipietro, L., Fasoli, S. E., … Volpe, B. T. (2006). Motions or muscles? Some behavioral factors underlying robotic assistance of motor recovery. *Journal of Rehabilitation Research and Development, 43*(5), 605–618.

Krebs, H. I., Ferraro, M., Buerger, S. P., Newbery, M. J., Makiyama, A., Sandmann, M., … Hogan, N. (2004). Rehabilitation robotics: Pilot trial of a spatial extension for MIT-Manus. *Journal of Neuroengineering and Rehabilitation, 1*(1), 5.

Krebs, H. I., Volpe, B. T., Williams, D., Celestino, J., Charles, S. K., Lynch, D., & Hogan, N. (2007). Robot-aided neurorehabilitation: A robot for wrist rehabilitation. *IEEE Engineering in Medicine and Biology Society, 15*(3), 327–335.

Lang, C. E., Macdonald, J. R., Reisman, D. S., Boyd, L., Jacobson Kimberley, T., Schindler-Ivens, S. M., … Scheets, P. L. (2009). Observation of amounts of movement practice provided during stroke rehabilitation. *Archives of Physical Medicine and Rehabilitation, 90*(10), 1692–1698.

Lo, A. C., Guarino, P. D., Richards, L. G., Haselkorn, J. K., Wittenberg, G. F., Federman, D. G., … Peduzzi, P. (2010). Robot-assisted therapy for long-term upper-limb impairment after stroke. *New England Journal of Medicine, 362*(19), 1772–1783.

Lo, K., Stephenson, M., & Lockwood, C. (2017). Effectiveness of robotic assisted rehabilitation for mobility and functional ability in adult stroke patients: A systematic review. *JBI Database of Systemic Reviews and Implementation Reports, 15*(12), 3049–3091.

Lundquist, C. B., & Maribo, T. (2017). The Fugl-Meyer assessment of the upper extremity: Reliability, responsiveness and validity of the Danish version. *Disability and Rehabilitation, 39*(9), 934–939.

Maciejasz, P., Eschweiler, J., Gerlach-Hahn, K., Jansen-Troy, A., & Leonhardt, S. (2014). A survey on robotic devices for upper limb rehabilitation. *Journal of Neuroengineering and Rehabilitation, 11*, 3.

McConnell, A., Moioli, R., Brasil, F., Vallejo, M., Corne, D., Vargas, P., & Stokes, A. (2017). Robotic devices and brain-machine interfaces for hand rehabilitation post-stroke. *Journal of Rehabilitation Medicine, 49*(6), 449–460.

Mehrholz, J., Pohl, M., Platz, T., Kugler, J., & Elsner, B. (2015). Electromechanical and robot-assisted arm training for improving activities of daily living, arm function, and arm muscle strength after stroke. *The Cochrane Datbase of Systematic Reviews*, (11), CD006876.

Mehrholz, J., Thomas, S., Werner, C., Kugler, J., Pohl, M., & Elsner, B. (2017). Electromechanical-assisted training for walking after stroke. *The Cochrane Database of Systematic Reviews*, (5), CD006185.

Nudo, R. J., Wise, B. M., SiFuentes, F., & Miliken, G. W. (1996). Neural substrates for the effects of rehabilitative training on motor recovery after ischemic infarct. *Science, 272*(5269), 1791–1794.

Poli, P., Morone, G., Rosati, G., & Masiero, S. (2013). Robotic technologies and rehabilitation: New tools for stroke patients' therapy. *Biomedical Research International, 2013*, 153872.

Rossi, S., Hallett, M., Rossini, P. M., Pascual-Leone, A., & Safety of TMS Consensus Group. (2009). Safety, ethical considerations, and application guidelines for the use of transcranial magnetic stimulation in clinical practice and research. *Clinical Neurophysiology, 120*(12), 2008–2039.

Volpe, B. T., Huerta, P. T., Zipse, J. L., Rykman, A., Edwards, D., Dipietro, L., … Krebs, H. I. (2009). Robotic devices as therapeutic and diagnostic tools for stroke recovery. *Archives of Neurology, 66*(9), 1086–1090.

Volpe, B. T., Lynch, D., Rykman-Berland, A., Ferraro, M., Galgano, M., Hogan, N., & Krebs, H. I. (2008). Intensive sensorimotor arm training mediated by therapist or robot improves hemiparesis in patients with chronic stroke. *Neurorehabilitation and Neural Repair, 22*(3), 305–310.

Wagner, T. H., Lo, A. C., Peduzzi, P., Bravata, D. M., Huang, G. D., Krebs, H. I., … Guarino, P. D. (2011). An economic analysis of robot-assisted therapy for long-term upper-limb impairment after stroke. *Stroke, 42*(9), 2630–2632.

Winstein, C. J., Stein, J., Arena, R., Bates, B., Cherney, L. R., Cramer, S. C., … Zorowitz, R. D. (2016). Guidelines for adult stroke rehabilitation and recovery: A guideline for healthcare professionals from the American Heart Association/American Stroke Association. *Stroke, 47*(6), e98–e169.

Wu, X., Guarino, P., Lo, A. C., Peduzzi, P., & Wininger, M. (2016). Long term effectiveness of intensive therapy in chronic stroke. *Neurorehabilitation and Neural Repair, 30*(6), 583–590.

Yue, Z., Zhang, X., & Wang, J. (2017). Hand rehabilitation robotics on poststroke motor recovery. *Behavioural Neurology, 2017*, 3908135.

Chapter 6
The Role of Neuro-Optometric Rehabilitation

M. H. Esther Han

Introduction

Neuro-optometric rehabilitation (NOR) is founded on the concept that the visual pathways are intricately integrated to other neuro-sensory pathways in the brain. In fact, approximately 70% of the afferent sensory input to the brain is vision related, and visual information is processed either directly or indirectly in every lobe of the brain (Han, 2007; Kaufman, 1992). The visual sequela resulting from an acquired brain injury (ABI) negatively impacts activities of daily living and overall functioning. This is especially true after a concussion or mild traumatic brain injury (mTBI), where the effects of diffuse axonal injury results in a significant number of visual symptoms (Craig et al., 2008; Master et al., 2016; Tannen, Darner, Ciuffreda, Shelley-Tremblay, & Rogers, 2015). Therefore, a patient with an ABI should be referred to a neuro-optometrist for the evaluation and management of their vision deficits. In addition, patients with visual-vestibular dysfunction, as well as patients with autoimmune conditions, may also present with similar vision symptoms and a referral may be indicated.

Visual Pathways

There are two streams of cortical visual information processing in the brain, the ventral and dorsal streams (Goodale, 2010; Helvie, 2011). The ventral stream is considered the "what is it?" stream, and is also called the parvocellular (P) pathway. This P pathway can also be referred to as the focal processing pathway or central processing pathway (Rosen, Cohen, & Trebling, 2001). The ventral stream mediates the recognition of objects and faces. The dorsal stream is considered the

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"where is it?" stream and is also called the magnocellular (M) pathway. This M pathway can also be referred to as the ambient processing pathway or the peripheral processing pathway (Rosen et al., 2001). It is involved in visual motion and visual spatial processing.

Patients with ABI typically report problems with visual motion and with visual spatial processing. There is a mismatch between visual information and other sensorimotor systems, which leads to poor peripheral awareness and poor spatial organization (Padula, 1988b). An individual with dorsal stream deficits will pay more attention to visual details rather than the visual gestalt, resulting in variable visual abilities. For example, some patients report stationary objects appearing to move, and overstimulating settings, such as supermarkets, airports, and shopping malls, to be visually unsettling. A patient with an ABI in the supermarket reports being overwhelmed by the visual details of all the items on the shelf, rather than seeing the aisle as a visual gestalt. This individual should not be processing all the specific visual details at this moment but should just be first looking for the correct aisle. This inability to ignore unimportant visual information leads to their symptoms of visual stimulation.

A multimodal, multisensory integrative approach is utilized in the evaluation and the management of patients with ABI. This will facilitate the rehabilitation process when the visual sequela negatively impacts progress in physical, occupational, cognitive, and vestibular rehabilitation therapy programs. It is important for the neuro-optometrist to understand how vision affects function and be able to clearly communicate how these vision deficits can affect activities of daily living. The purpose of this chapter is to present the elements of the evaluation and to describe the various treatment options available to the patient. Cases are presented to review the outcomes resulting from the neuro-optometric management of vision deficits secondary to an ABI.

Evaluation

The neuro-optometric rehabilitation (NOR) evaluation includes the following elements: an extensive case history, refractive status, sensorimotor status, and visual field status. Evaluation and management of the following will also occur if indicated: visual-vestibular status, visual perceptual status, light sensitivity, contrast sensitivity status, and colored filter and/or syntonic phototherapy considerations. For some cases, colored filters in the form of prescription tinted lenses or as a formal syntonics phototherapy program may be indicated. A typical evaluation would also include an ocular health assessment, which will not be discussed in this chapter. For an extensive description of ocular sequelae associated with ABI, see Gerner (1993), Suchoff, Kapoor, Waxman, and Ference (1999), Vogel (1992), and Sabates, Gonce, and Farris (1991).

A NOR evaluation can typically be completed over two to three appointments. The first examination entails the comprehensive eye examination, and would include

an in-depth case history, refractive status determination, screening for sensorimotor (version/oculomotor, vergence/binocular, and/or accommodation—see Glossary for definitions of terms) deficits, and the ocular health evaluation. Depending on how fatigued or uncomfortable the patient may be, the ocular health evaluation and automated visual field evaluation may require an additional appointment. If urgent visual symptoms are noted during the case history suggesting that the ocular health assessment with dilation is indicated, then that procedure must take priority. The second evaluation involves the sensorimotor evaluation to determine the severity of the conditions screened for during the first exam. The third evaluation is typically for the completion of additional tests not necessarily indicated for all patients.

Generally, compensatory strategies are recommended after each evaluation to help minimize the effects of their vision deficits while they are receiving rehabilitation services and may include the need to limit exposure to visually stimulating environments. This is especially the case in crowded public settings. For instance, any future appointments may need to be scheduled after or before commuter rush hours. When a patient must be in a visually overstimulating environment (i.e., watching a movie or a theater performance), he or she must be educated to take visual breaks, since they will be physically and cognitively drained, even up to 1–2 days after the event.

The evaluation results will drive how neuro-optometric rehabilitation therapy (NORT) is administered, especially in the early stages of therapy. For instance, the patient must be careful to avoid performing visual tasks in cluttered or visually overstimulating situations. Fatigue will occur sooner, when a patient attempts to multitask vision activities while performing other multimodal processing (verbal, motor, or auditory) tasks. For such patients, the goals of NORT may be to maximize their ability to process sensory (visual, auditory, verbal, or motor) information simultaneously, or to visually attend to two or more things at once, or to rapidly switch visual attention from one object to another.

Case History

Symptom surveys help the clinician to have a repeatable clinical tool to assess and quantify initial symptoms and then evaluate the efficacy of treatment when the survey is re-administered at a progress evaluation. There are several symptom surveys that are utilized for this purpose. The Convergence Insufficiency Symptom Survey (CISS) is a validated survey (see Appendix 1) that can be used effectively for post-concussion adolescent patients (Master et al., 2016). This is the recommended symptom survey to be used when *referring* a patient for a NOR evaluation. For an adult greater than 21 years, a score of 21 and greater is indicative of significant vision symptoms warranting a vision evaluation. For those under 21 years, a score of 16 and higher is indicative of a significant vision problem. This symptom survey is appropriate, since convergence insufficiency is highly prevalent in the ABI population, particularly in post-concussive patients. The Post Trauma Vision

Symptoms (PTVS) survey (see Appendix 2) is more comprehensive and relevant for the NOR evaluation, as it includes symptoms related to visual field and visual-vestibular deficits. A third symptom survey (see Appendix 3), the Brain Injury Vision Symptom Survey (BIVSS), is in the process of being further validated and is also specific for the NOR evaluation (Laukkanen, Scheiman, & Hayes, 2017).

The NOR case history (see Table 6.1 for a list of the common elements of the vision rehabilitation case history) involves a comprehensive investigation of the nature of the brain injury (including a loss of consciousness), referral source, types of rehabilitation services received, visual problems affecting activities of daily living, personal and family medical history, and personal and family ocular history. The case history is often the most revealing part of the examination for both the patient and the neuro-optometrist. For instance, the patient can experience relief to finally discover that an undiagnosed vision problem may be contributing to his or her difficulties. The case history is also a crucial point to gain a better picture of the patient's visual needs, and how he/she typically responds to vision challenges, e.g., avoidance, trying harder, or frustration.

The nature of the injury reveals the location of the brain injury and the possible areas of function that may be affected. In addition, vision deficits will arise from damage to areas of the brain other than the occipital lobe, within which the primary visual cortex resides (Gianutsos et al., 1988). The number of years post injury provides an indication of the acute versus chronic nature of the vision symptoms. A patient evaluated in the first 6 months after a brain injury may experience a sudden decrease in the frequency and severity of his or her visual symptoms within the first year of the injury (Suter, 1995). However, there are some patients who chronically experience visual symptoms several years after the incident. Clinically, it is known that ABI patients are prone to suffer from multiple head injuries and each incident is highly likely to exacerbate an existing vision dysfunction. Therefore, it is important to know whether a patient has suffered from multiple head injuries.

The source and the specific reason for the referral are significant. For instance, a vestibular therapist may refer a patient experiencing intermittent diplopia, or double vision, during a gaze stabilization task. The patient's diplopia now becomes an obstacle in his/her progress in the vestibular therapy. Communication is necessary in such cases between the referral source and the treating optometrist to evaluate

Table 6.1 Elements of the NOR evaluation case history

1. Nature of the injury, including the type of injury (with or without loss of consciousness), date of injury, number of injuries, length of hospitalization
2. Referral source
3. Rehabilitation history, including in-patient or outpatient rehabilitation services received to date
4. Current vision problems or complaints that affect overall performance and/or the progress of other types of rehabilitation
5. Personal and family medical history, including medications and allergies
6. Personal and family ocular history
7. Social history, which includes occupation, mobility, driving history, and support resources

progress in therapy. This is particularly the case for patients with cognitive deficits, who may not be able to adequately judge their progress in therapy.

The chief goal of the NOR evaluation is to determine the current visual needs of the patient. This requires a lengthy discussion of the functional difficulties a patient may be experiencing in daily life. This may range from bumping into people when walking, shaving only one side of the face, driving challenges, or poor multitasking abilities in the work setting. The neuro-optometrist will incorporate the case history and the vision findings to determine if a vision deficit is contributing to the patient's reported difficulties.

The personal and familial medical and ocular history is important to ensure that the patient is being routinely managed for other systemic conditions. Patients are often inundated with medical appointments and will benefit from being reminded to follow up with their other medical providers. Patients with ABI are also prescribed numerous medications; some have vision side effects that may affect the patient more than in a non-brain-injured patient. In such cases, the neuro-optometrist attempts to decrease the severity of the symptoms maximally, using visual hygiene strategies, lenses, and/or NORT.

The social history provides an idea of how the patient functioned in the pre-injury state. The difficulty of coping with functional losses because of the injury is often greater in higher-functioning individuals. Mobility and driving needs also are extensively reviewed to determine necessary recommendations.

Patients, with traumatic brain injury (TBI) specifically, report the following vision-related symptoms with high frequency (45–50%): eyestrain with near vision tasks, light sensitivity, headaches, near vision blur, loss of balance, and dizziness (Craig et al., 2008; Tannen et al., 2015). The following symptoms and their frequencies are most commonly reported by post-stroke patients: near vision blur (40%), eyestrain with near vision tasks (38.3%), loss of place when reading (33.3%), and distance vision blur (31.7%) (Craig et al., 2008). The vision symptoms listed in Table 6.2 are the common ones that must be investigated when evaluating a patient with an ABI.

Table 6.2 Common vision symptoms following ABI

Intermittent blurred vision (distance, near, or both)
Dulling of vision
Eyestrain
Double vision
Headaches
Light sensitivity
Depth perception problems
Reading efficiency and reading comprehension problems
Dizziness, loss of balance, or vertigo
Visual neglect
Loss of peripheral vision

Refractive Status

One of the main elements of the NOR examination is the determination of the refractive error. The types of refractive errors include emmetropia, myopia, hyperopia, astigmatism, or presbyopia (see Glossary for definitions of terms). These conditions are typically corrected using spectacles or contact lenses. Refractive surgery is also used for the correction of refractive error. Recommendations regarding this treatment option should be done on a case-by-case basis, as poor fixation, poor binocular status, and accommodative fluctuations may negatively affect surgical outcomes (Godts, Tassignon, & Gobin, 2004).

Myopia is corrected by minus or concave lenses, and hyperopia with plus or convex lenses (see Fig. 6.1). Astigmatism exists when refractive error differs in orthogonal meridians of the eye. Presbyopia is characterized by the need for additional plus lenses for near-point tasks due to age-related changes in the lens of the eye, and it generally manifests in patients older than 40 years (see Fig. 6.2) (Rosenfield, 1997). Clinically, the symptoms related to presbyopia may occur earlier in patients with a brain injury.

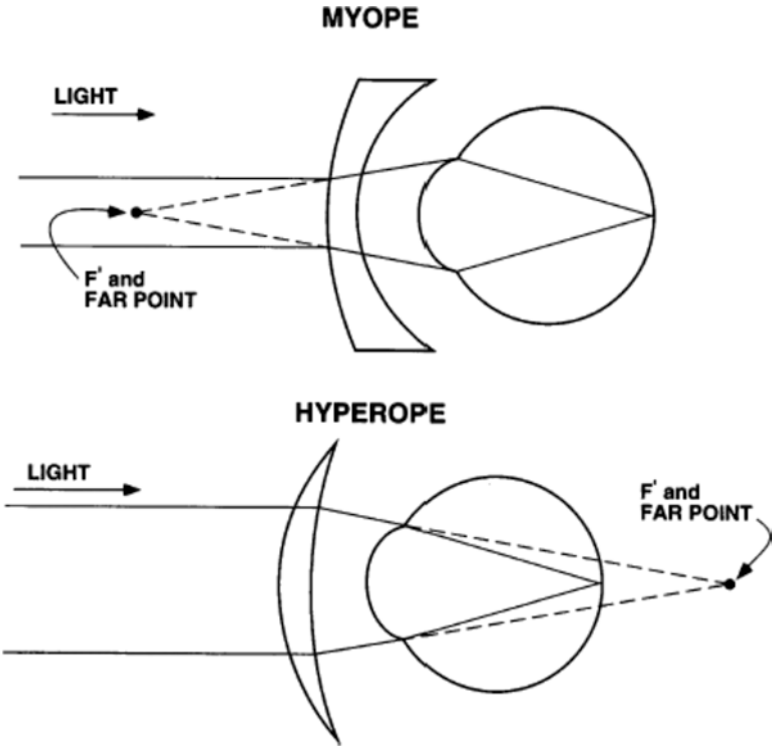


Fig. 6.1 Correction of myopia and hyperopia with lenses (Cho & Benjamin, 1998)

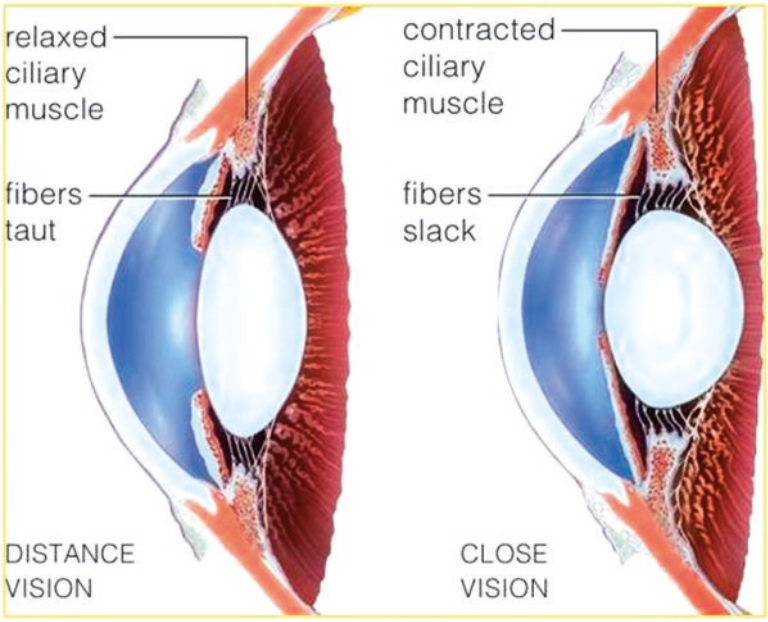


Fig. 6.2 Intraocular lens changes that occur when accommodating for close vision (Starr, Evers, & Starr, 2011). From Starr/Evers. Cengage Advantage Books: Biology, 8E. © 2011 Brooks/Cole, a part of Cengage, Inc. Reproduced by permission. www.cengage.com/permissions

Symptoms related to uncorrected refractive error include blurred vision at distance or near, shadows or halos around images, and eyestrain under sustained viewing conditions. One study indicated that 46% of patients with closed-head injury report some form of blurred or decreased vision (Sabates et al., 1991). Decreases in contrast sensitivity may also contribute to the quality of their vision and not significantly affect their visual acuity. This is particularly true in cerebrovascular accident (CVA) patients. Contrast sensitivity deficits were reported in 62% of patients with ischemic events affecting the posterior visual pathways (Warren, 1993). This is further exacerbated in elderly patients, who often have cataracts that will also contribute to decreased contrast sensitivity and visual acuity levels. Poor contrast sensitivity is also reported in patients with visual field defects (Warren, 1993). Deficits in contrast sensitivity will affect how a patient functions under foggy and dark conditions (Suter, 1995). Treatment options may involve contrast-enhancing colored tints or filters, high contrast reading material, and task specific lighting (Suter, 1995).

Suchoff et al. (1999) found that 50% of ABI patients in their study required a prescription for spectacles. These patients included those who needed glasses for the first time, those who needed a replacement for lost glasses, and those requiring a change in their prescription. In a study by Sabates et al. (1991), 88% of patient's eyes were correctable to 20/20. This reinforces the fact that most of the patients' vision complaints can be resolved with the appropriate refractive correction. This is particularly true, since changes in refractive status are observed post-injury and will

typically include increases in myopia, fluctuating prescriptions in hyperopic patients, and/or decreases in the degree of hyperopia (Padula, 1988a).

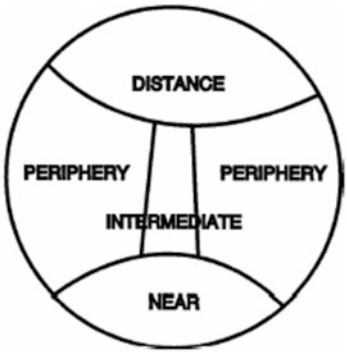
A consideration when determining the refractive status in a patient with ABI is that some patients experience a significant degree of vision fluctuation. During the examination, these fluctuations will be observed when measuring acuity and when determining the patient's prescription. Careful monitoring of visual acuities is necessary within the first 1–2 months of wearing the new prescription. Upon follow-up, it is possible that a change in the prescription will be indicated as vision continues to stabilize. In addition, some patients report significant improvement in clarity and comfort, with very small refractive changes that would typically not be noticeable by the non-brain-injured individual.

Spectacle Recommendations

Recommendations regarding choice of lens design are very important, and patients may require several pairs of glasses for different needs. Mobility/ambulation is one of the main issues that must be considered when writing the spectacle prescription. Progressive (no-line multifocal lenses) and bifocals are NOT recommended for patients with poor mobility. Single-vision lenses made of a polycarbonate (high impact resistance) material are recommended for distance use when walking (Brooks & Borish, 1996). Progressive lenses have defocus and distortion at the periphery of the lenses, which may contribute to the patient's unsteadiness (see Fig. 6.3). The patient, wearing either progressive or bifocal lenses, often views through the reading portion and will experience blurred vision when looking down at their feet while walking. This may occur when going up or down steps, or during physical therapy sessions when patients will inevitably look at their feet. This is especially critical during the first few months after the injury, when the patient is still adjusting to their visual sequela and has not developed or learned the necessary compensatory strategies. Single-vision near or single-vision intermediate glasses are typically advised for sustained visual activities such as reading or computer use.

Patients who have not worn glasses prior to the injury have clinically been noted to be poorly compliant with spectacle wear despite a significant increase in acuity because the patient may prefer "soft" vision that is not crisp. The improved clarity increases the amount of visual stimulation they experience, particularly in busy and crowded environments. Another obstacle to compliance with spectacle wear is the presence of cognitive deficits. These patients may consistently forget or lose their glasses and may not remember or understand which pair of glasses to use for a specific task. A multifocal (bifocal or progressive) lens design would be optimal for such patients who do not have mobility issues.

Prescribing for the dizzy patient is challenging because of aberrations inherent to any type of spectacle lenses. Patients with high refractive errors may not be able to wear spectacles frequently and, therefore, contact lenses may be a more comfortable option. When viewing through spectacle lenses, rotational magnification can



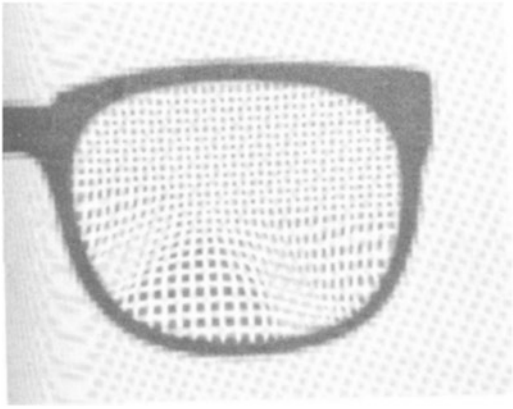


Fig. 6.3 Peripheral distortion seen with progressive lenses (Cho & Benjamin, 1998)

magnify or minify the effect of head movement depending on the power of the lens (Weiss, 2002). Sometimes slightly under-correcting the myope may stabilize vision enough to improve peripheral awareness and posture (Weiss, 2002).

The prevalence of light sensitivity in the TBI population is as high as 45–50%; this is especially true in post-concussive patients (Craig et al., 2008). As will be further discussed later in this chapter, tinted lenses are recommended for these patients. The most common tints that should be trialed are blue, brown, and gray tints. Clinically, the post-concussed patient will choose the blue tint with higher frequency. The tints are often recommended to relieve light sensitivity symptoms when indoors under the fluorescent lighting or when using electronic devices. The ranges of tint density for indoors that is prescribed is 10–15% for mild cases, and 20–35% for more moderate cases. Specifically, for visual-vestibular patients, the tint densities are higher for indoor use with a 10–15% blue tint or a 30–40% gray or brown tint (Iskander, Cohen, & Kapoor, 2010). Clinically, patients with significant visual-vestibular symptoms also report that polarizing sunglasses are very comfortable outdoors because reflected light is restricted to moving in only the vertical direction and thereby reduces glare intensity (Brooks & Borish, 1996).

Patients with ABI can also be prescribed prism into their spectacle lenses. The two most common types of prism are fusional and yoked prism. Fusional prism will

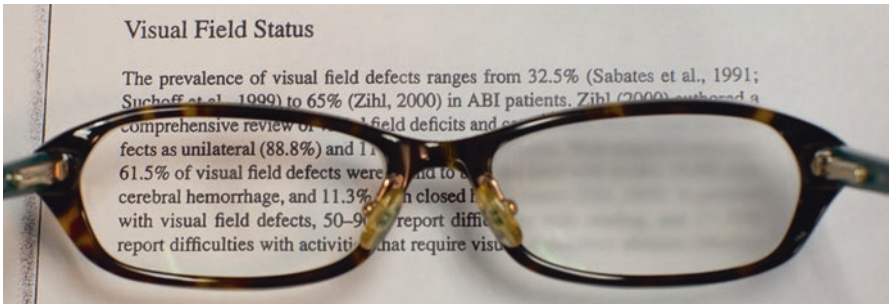


Fig. 6.4 Example of frosted occlusion when a patient has diplopia

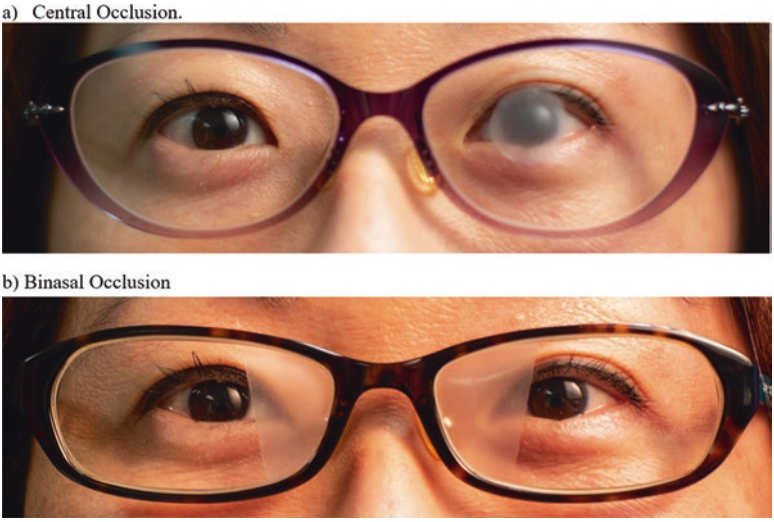


Fig. 6.5 Examples of partial occlusion. (**a**) Central occlusion. (**b**) Binasal occlusion

be used for patients who see diplopia (horizontal, vertical, or diagonal) but do not have the compensatory fusion abilities to make the image single on their own. Fusional prism is prescribed to move the double images, so they become single. Yoked prisms have the bases in the same direction over each eye and are typically *not* prescribed for patients with diplopia. Rather they are prescribed for patients with a visual field related deficit or for a patient with abnormal egocentric localization (AEL). These conditions will be further described later in this chapter.

Treatment options for the patient with ABI can also include different types of occlusion. Frosted occlusion (see Fig. 6.4) or a partial central occlusion (see Fig. 6.5a) with a Bangerter foil will be recommended for a patient with constant diplopia (Sanet & Press, 2011). This is very useful in a patient with variable diplopia and/or when there is noncomitant diplopia, that is, the separation of the double images changes in magnitude depending on the viewing direction. The Bangerter

foil occlusion is more advantageous than a black opaque occluder because the patient benefits from light entering the eye. There is greater benefit to using partial or central occlusion because the patient can still utilize their peripheral vision to help them maintain fusion.

Binasal occlusion (see Fig. 6.5b) is to address symptoms of visual motion sensitivity (VMS) in patients with ABI or with visual-vestibular deficits. According to Ciuffreda, Yadav, and Thiagarajan (2016), approximately 40% of ABI patients experience VMS that is associated with peripheral retinal-image motion related to a phenomenon called Gibsonian optic flow. Patients reporting VMS avoid visually crowded areas, such as a busy street or a busy shopping area. This behavior is often referred to as "supermarket syndrome." Patients feel unsteady and fear falling or bumping into others, and they can also report nausea. Binasal occlusion reduces the effect of the peripheral retinal-image motion, and the positive effects of this type of occlusion has been objectively demonstrated with visual-evoked potential (VEP) testing (Ciuffreda et al., 2016; Ciuffreda, Yadav, & Ludlam, 2017).

Sensorimotor Status

An evaluation of sensorimotor function includes the assessment of oculomotor, binocular, and accommodative functions. Oculomotor or versional function includes fixation, pursuit, and saccadic eye movement abilities (see Glossary for definitions). This involves horizontal and vertical eye movements. Evaluation of vergence or binocular function includes the assessment of ocular alignment, measurement of stereopsis, and determination of fusion (convergence and divergence) ranges at distance and near. Binocular tasks involve eye movements related to depth perception. Accommodative testing is typically performed in pre-presbyopic patients (younger than 45 years), and includes the measurement of amplitude and facility (monocularly and binocularly).

The neural network contributing to these areas of function encompasses the frontal, parietal, midbrain, and cerebellar regions of the brain (Ciuffreda, Han, Kapoor, & Suchoff, 2001; Ciuffreda & Tannen, 1995). A single locus of damage or diffuse damage from a TBI can lead to multiple levels of deficits in visual function. The midbrain is the control center for eye movements, so damage to this area leads to significant visual sequela after a TBI. In the brainstem lies the oculomotor, abducens, and trochlear cranial nuclei, which innervate the extraocular muscles (Ciuffreda & Tannen, 1995). Cortical structures involved in generating eye movements include the frontal eye fields and the parietal lobes. The parietal lobes are primarily involved in the localization of a target, while the frontal eye fields are involved in attention and target selection processes (Ciuffreda & Tannen, 1995; Helvie, 2011; Sanet & Press, 2011). Suter (1995) stated that 71% of TBI patients in an acute rehabilitation center were diagnosed with either an oculomotor or binocular dysfunction, strongly suggesting that a NOR evaluation is strongly indicated post-injury.

Oculomotor, binocular, and accommodative functions are directly responsible for the efficient input of visual information to the brain. When these systems are not functioning adequately, excess cognitive effort is exerted in an attempt to keep vision clear, and single enough to take in relevant visual information. This excess effort takes away from the cognitive processes of attention and comprehension during a task, such as reading. In ABI patients with cognitive deficits and vision deficits, performing near-vision tasks can be physically and mentally exhausting.

Oculomotor (Version) Function

Deficits of oculomotor/version function in the ABI population include deficits of fixation, pursuits, and saccades. Abnormal findings in oculomotor function will affect the following: initiation of an eye movement, latency, accuracy or gain, and visual attention for the required eye movement task (Ciuffreda & Tannen, 1995; Warren, 1993). Deficits in oculomotor function can significantly affect the patient's activities of daily living (ADLs). Table 6.3 lists the typical symptoms of patients with deficits in oculomotor function.

Studies reveal a prevalence of an oculomotor dysfunction in 51.3% of TBI (Ciuffreda et al., 2007). In a military population, the prevalence range is from 29.9% (outpatient) to 39.7% (in-patient) (Brahm et al., 2009). The prevalence in patients with concussions or mild TBI (mTBI) ranges from 21.6% to 29.0% (Gallaway, Scheiman, & Mitchell, 2016; Master et al., 2016). A rate of 56.7% was found in post-CVA patients being diagnosed with a saccadic deficit (Ciuffreda et al., 2007). Oculomotor deficits (smooth pursuit and saccadic deficits) were found in 62% of whiplash patients at their 2-year follow-up. These deficits were associated with restriction in cervical range of motion, but also with faulty proprioceptive information from the cervical spinal region (Heikkila & Wenngren, 1998).

The main treatment option for deficits in oculomotor function is NORT, with the goal of developing more systematic or organized eye movement patterns. Warren (1993) described that patients with visual field defects will often scan the intact field first and will also spend less time searching in the affected field. He also stated that patients with expressive or receptive aphasia demonstrated a more simplistic visual scanning pattern. These patients will report more fatigue and stop searching for further details within a complex visual pattern, thereby further limiting their ability to verbally express what was seen.

Table 6.3 Visual symptoms associated with oculomotor deficits

Loss of place, skipping lines when reading
Poor reading efficiency
Reading comprehension problems
Dizziness, loss of balance, or vertigo

Binocular (Vergence) Function

Deficits of binocular dysfunction include strabismic and nonstrabismic dysfunctions (Scheiman & Wick, 2014). Nonstrabismic dysfunctions can be categorized into distance and near vision disorders. The two common distance disorders are divergence excess (DE) and divergence insufficiency (DI). In patients with ABI, the DI condition is clinically more observed of the two distance disorders. When a patient exhibits binocular dysfunctions at both distance and near, the two possible diagnoses are basic esophoria and basic exophoria. At near, the two nonstrabismic disorders are convergence insufficiency (CI) and convergence excess (CE). Table 6.4 lists the common symptoms reported by patients with deficits in binocular function.

Convergence Insufficiency (CI) is the most prevalent diagnosis after a TBI. It is 42.5% in a TBI cohort, a range from 42.6% (out-patient) to 48.4% (in-patient) in military TBI patients, and a range from 47.5% to 56.0% in only post-concussive patients (Brahm et al., 2009; Ciuffreda et al., 2007; Gallaway et al., 2016; Master et al., 2016; Tannen et al., 2015). In post-CVA patients, 36.7% had a CI (Ciuffreda et al., 2007).

The prevalence rate of any binocular dysfunction in TBI patients ranged from 56.3% to 62.3% (Ciuffreda et al., 2007; Gallaway et al., 2016). A study by Gianutsos, Ramsey, and Perlin (1988) showed that 73% ($n = 26$) of patients with severe head trauma exhibited some degree of binocular dysfunction. Suchoff et al. (1999) stated that 41.9% of ABI patients show an exodeviation or under-convergent deviation as compared to an occurrence of 2.11% in the normal population. Vertical deviations were seen in 9.7% as compared to an occurrence of 1.6% in normal individuals. The prevalence of esodeviations or over-convergent deviations (1.6%) is not very different from that seen in normal individuals (1.28% occurrence). Ciuffreda et al. (2001) stated that 40% of CVA patients manifest reduced binocular abilities.

The strabismic conditions that can be seen after a brain injury are esotropia, exotropia, and hypertropia. The strabismus can be characterized as being constant or intermittent, and can occur at distance, near, or both. A patient may prefer to consistently fixate with one eye, while the other is turned, or the patient may alternate fixation from one eye to the other. Another characteristic of the strabismus that needs to be assessed is the comitancy or whether the size of the eye turn changes in different positions of gaze. A patient with a non-comitant deviation may report

Table 6.4 Symptoms associated with binocular deficits

Diplopia (double vision) at distance, near, or both; horizontal, vertical, or diagonal
Intermittent or constant eye turn at distance, near, or both; Can be horizontal, vertical, or both
Neck or shoulder discomfort (Padula, 1988a)
Poor object localization (Padula, 1988a)
Poor depth perception
Head turn or head tilt (Suter, 1995)
Poor body posture (Suter, 1995)

diplopia only when looking in one direction or reports more diplopia when looking towards one direction. Noncomitant deviations will occur concurrently with a palsy of a cranial nerve that affects the extraocular muscles, such as cranial nerves III, IV, and VI.

A study by Sabates et al. (1991) found 30% of patients with closed-head trauma (n = 181) reported diplopia and 33% manifested cranial nerve palsies. Prevalence rates of cranial nerve palsies in TBI patients are as follows: 16.2–25% for third nerve palsies, 16.7% for sixth nerve palsies, 32.0–36% for fourth nerve palsies, and 25% for multiple nerve involvement (Falk & Aksionoff, 1992; Suter, 1995). Sabates et al. (1991) also found that 75% of closed-head trauma patients with cranial nerve palsies resolved without surgical intervention within 6 months to a year. At times, surgical correction of an acquired strabismus is indicated to decrease the size of the turn. This is often recommended when the angle of deviation has been stable. Fawcett, Stager, and Felius (2004) determined that patients with acquired cases would have better stereopsis when the constant strabismus was present 12 months or less before surgery. One study stated that third and fourth nerve palsies generally require surgical correction, and 25% of the patients in the study required some form of surgical correction (Sabates et al., 1991). Some TBI patients require more than one surgery; one study showed 50% required more than one surgery, and 30% required more than two (Suter, 1995). It is strongly recommended that surgical intervention in conjunction with NORT will improve visual outcomes post-surgically (size of the turn, frequency of double vision, visual comfort, and possibly a decreased need for multiple surgeries) (Suter, 1995).

The treatment options for the above binocular vision dysfunctions include one or a combination of the following: fusional prism, occlusion (binasal or partial occlusion), surgical correction and/or NORT (Suter, 1995; Padula, 1988b). The main goal is to decrease the severity and frequency of the patient's symptoms. In patients experiencing constant double vision and who are unable to undergo a NOR evaluation, it is recommended to patch the eyes on a daily *alternating* schedule to allow for visual information to enter each eye, to provide peripheral visual cues for each eye, to prevent suppression, and to increase the chances for spontaneous recovery of fusion (Suter, 1995).

Accommodative Function

The neurological pathways for increasing and decreasing accommodation in a non-presbyope is under the autonomic nervous system. The parasympathetic system is responsible for increasing accommodation. The sympathetic system is responsible for decreasing accommodation starting at the hypothalamus and synapsing with the superior cervical ganglion located in the neck (Helvie, 2011; Kaufman, 1992). It has been shown that TBI patients with neck-associated damage, such as whiplash, do have accommodative deficits (Brown, 2003). Therefore, it is important to ensure the

patient is addressing neck related dysfunctions with physical therapy rehabilitation, particularly when accommodative deficits are diagnosed post trauma.

The main types of accommodative dysfunctions include (1) Accommodative Insufficiency, (2) Accommodative Infacility, and (3) Accommodative Excess (Scheiman & Wick, 2014). Accommodative dysfunction is more commonly seen in TBI patients as opposed to CVA patients because a TBI results in more diffuse damage as opposed to the more discrete lesions that occur in a CVA (Leslie, 2001). However, damage to the brain stem area will result in more damage to the areas controlling accommodative, binocular, and oculomotor functions (Chan & Trobe, 2002; Leslie, 2001). Leslie (2001) stated that accommodative dysfunctions should be viewed as disturbances or loss of learned ability to appropriately change accommodation for an object of regard.

The prevalence of accommodative deficits in the general TBI population is 41.1% and ranges from 39.6% (out-patient) to 47.5% (in-patient) in military TBI patients (Brahm et al., 2009; Ciuffreda et al., 2007). There is a higher prevalence in concussed patients, which ranges from 51% to 76% (Gallaway et al., 2016; Master et al., 2016; Tannen et al., 2015). A 20% prevalence rate was seen in mid-facial trauma patients (Leslie, 2001). A rate of 12.5% was seen in post-CVA patients (Ciuffreda et al., 2007). These rates suggest that careful diagnosis and management (including lenses and NORT) of accommodative problems in the ABI population is of utmost importance, particularly in the post-concussive patient.

Table 6.5 lists the common symptoms reported by patients with accommodative dysfunction. Sabates et al. (1991) stated that 13% of patients with closed head trauma experienced headaches, and 6% reported difficulties with reading. However, it must be noted that 29% of the patients also reported a history of post-concussive migraines. It was once thought that visual and ocular side effects of medications may exacerbate deficits on accommodation. A retrospective study by Han et al. (2008) found that commonly prescribed medications (antihypertensives, antidepressants, antianxiolytics, and anticonvulsants) taken by either TBI or CVA patients do not appear to severely exacerbate accommodative dysfunctions.

The common treatment options of accommodative dysfunctions include a near-vision spectacle prescription, and/or NORT. The goal of therapy is to develop flexibility between the accommodative and binocular systems to maximally stabilize vision function. Additionally, some pre-presbyopic patients may require a near-vision prescription at an earlier age than would be expected, due to the difficulties in initiating or sustaining accommodation (Cohen & Rein, 1992).

Table 6.5 Visual symptoms associated with accommodative deficits

Frontal headaches or brow aches
Intermittent or constant blurred vision (distance, near, or both), worsening later in the day
Pain around the eyes during visual activities
Limited ability to read or use computer for long periods (Leslie, 2001)
Limited progress in other rehabilitation therapies involving near-vision work (Leslie, 2001)

NORT Efficacy: Sensorimotor Deficits

Efficacy studies show a high success rate with NORT to treat oculomotor, binocular, and accommodative dysfunctions. Ciuffreda et al. (2008) showed that 90% of TBI patients reported both a marked reduction in one or more primary symptoms and/or a marked improvement or normalization in one or more of their primary clinical signs. Gallaway et al. (2016) performed a retrospective study on post-concussive patients; they differentiated whether a patient's vision dysfunction was successfully remediated or if their clinical findings improved but had not normalized. Of the three diagnoses reviewed, 85% of cases were successful for patients with convergence insufficiency, and 83% of cases were successful for patients with saccadic dysfunction. Accommodative insufficiency cases appear to be harder to treat in that 67% of cases improved while 33% of cases were successfully remediated.

Visual Field Status

The pattern of visual field loss allows for localization of the lesion in the brain. The homonymous hemianopic lesions indicate damage after the optic chiasm in the brain and the more congruent the defect's pattern; it is believed the damage is generally localized more posteriorly along the primary visual pathway (Margolis, 2011). Lesions specific to the parietal lobe areas lead to increased latency with respect to initiating a saccadic eye movement into the affected field of a patient with unilateral neglect (Ciuffreda & Tannen, 1995; Warren, 1993). Damage to the frontal eye fields affects direct voluntary visual searching abilities. A patient may also exhibit reduced awareness of the environment and increased latency of saccadic initiation towards the affected field (Warren, 1993). Deficits in the pathways involving the superior colliculus affect peripheral awareness and eye movements towards unanticipated or new stimuli in the environment (Warren, 1993). Attention deficits associated with eye movements are unique to CVA patients particularly those with hemispheric involvement and a corresponding hemianopic visual field defect (Ciuffreda et al., 2001).

Hemianopic visual field defects can also be associated with a visual unawareness of their visual surrounds. This unawareness of stimuli in the visual field opposite the cerebral lesion is known by many clinical terms: unilateral spatial inattention (USI), visual-spatial inattention, visual hemi-inattention, visual imperception, or visual-spatial neglect. USI results from damage to multiple areas of the brain, which explains why there is so much variability in the clinical presentation of these patients. Based upon their functional symptoms, USI can also be categorized by proximity: personal, peripersonal, and extrapersonal neglect (Margolis, 2011). A patient with personal neglect may have difficulty dressing due to neglect of their own arm or leg. Peripersonal neglect involves the neglect of the affected side in a space within arm's reach. A patient can demonstrate normal behavior with personal

and peripersonal awareness, but may still display symptoms of neglect beyond arm's reach or in extrapersonal space.

Whenever a visual field defect is suspected, the standard of care is to perform the appropriate visual field test. Testing should include confrontational visual fields either with fingers (single stimulus presentation) or with a transilluminator light/penlight in dim illumination. If the patient's awareness is poor, flickering the light by quickly covering and uncovering the light with a finger can maintain their attention. Patients with right brain damage, especially right parietal lobe defects, will demonstrate poor visual awareness (Massucci, 2009; Suchoff & Ciuffreda, 2004). Clinically, confrontational visual fields are subject to significant inter-examiner differences. If possible, an automated visual field should be performed using a screening method, which takes less time to administer and will indicate whether a point was seen or not seen; or a threshold method can be administered, which will quantify the intensity of the light that was visible to the patient. The threshold method provides insight into relative versus absolute defects that are often seen with post-CVA visual field defects. A relative defect indicates a decrease in sensitivity, but the patient will see the presented stimulus if the light intensity is increased; however, a patient with an absolute visual field defect will not be able to see the light no matter how much the intensity is increased.

To screen for USI, a test for extinction during confrontational visual field testing, using simultaneous double stimuli presentation should be performed (Suchoff & Ciuffreda, 2004). A patient with USI will respond to the single stimulus presentation, but not be aware of both stimuli during the simultaneous double stimuli presentation. In severe USI deficits, one hand is placed in the right field and the other is placed in the left field, and the patient will not notice the hand in affected field (Margolis, 2011).

The occurrence of visual field defects in the TBI population ranges from 35.0% to 38.75% (Brahm et al., 2009; Sabates et al., 1991; Suchoff et al., 2008). In combat, TBI for the military, the visual field defects were seen more in the blast-related injuries, which suggests that visual field testing should also be performed in TBI cases and not only for post-CVA cases (Brahm et al., 2009). The occurrence of visual field defects in post-CVA patients during a neuro-optometric rehabilitation evaluation was found to be 66.7% (Suchoff et al., 2008). One study, which evaluated the presence of homonymous hemianopic defects in patients 49 years and older, 52% of those with measurable visual field defects also reported a history of a stroke (Gilhotra, Mitchell, Healey, Cumming, & Currie, 2002). Interesting to note, at least 50% of patients with homonymous hemianopia from a stroke will spontaneously recover during the first 3 months of the incident (Zhang, Kedar, Lynn, Newman, & Biousse, 2006).

Zihl (2000) authored a comprehensive review of visual field deficits and found that 88.8% of the defects were unilateral, while 11.2% were bilateral defects. With respect to etiology, 61.5% of the visual field defects were found to be associated with stroke, 14.6% with cerebral hemorrhage, and 11.3% with closed head trauma. In patients with visual field defects, 50–90% report difficulties with reading, and 17–70% report difficulties with activities that require visual exploratory abilities

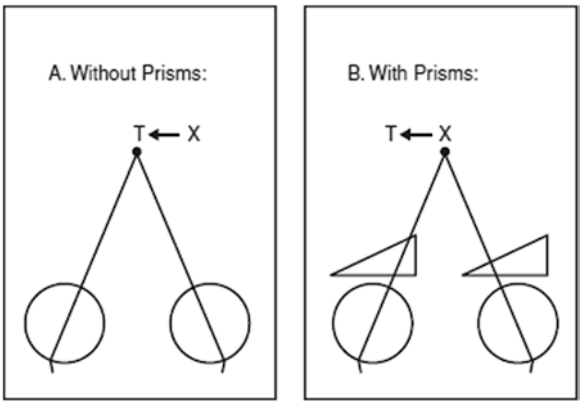


Fig. 6.6 Schematic representation of yoked prism effect when the patient is instructed to gaze straight ahead: (**a**) Without the yoked prisms, where T = a midline object, X = an object to the right of midline. (**b**) With the yoked prisms (base right), where objects T and X are optically displaced laterally to the left by the prisms without any change in eye position (Kapoor, Ciuffreda, & Suchoff, 2001

(Mueller, Poggel, Kenkel, Kasten, & Sabel, 2003). Patients also report inability to safely drive a car (Julkunen, Tenovuo, Jääskeläinen, & Hämäläinen, 2003).

Special considerations regarding visual field deficits are visual neglect and abnormal egocentric localization or visual midline shift syndrome. Suchoff and Ciuffreda (2004) stated that 12–49% of right-brain stroke patients demonstrate visual neglect, and it occurs more frequently in stroke than in TBI patients. Some patients may experience the Riddoch Phenomenon, in which they detect moving objects in the affected field, but not stationery objects (Gerner, 1993). Patients with visual neglect lean away from the area of loss, while patients with a visual field defect without inattention lean or turn toward the area of loss (Padula, 1988b). Yoked prisms (see Fig. 6.6) will bring about observable changes (Suchoff & Ciuffreda, 2004). Since significant improvement in visual neglect can be observed within 6 months of the incident, these patients may benefit from being monitored rather than prescribing the yoked prisms early in the rehabilitative process (Suchoff & Ciuffreda, 2004).

If the visual field defects remain after 6 months, there are several treatment options available, including neuro-optometric rehabilitation therapy (NORT). At the time of the evaluation, different types of field expansion prisms should be tried, prescribed, and success is typically dependent on training the patient to use the system. There are two types of sector prism/spotting prism systems: (1) Gottlieb Visual Field Awareness System™ and (2) Peli Lens™ Prism System. The Gottlieb Visual Field Awareness System utilizes a spectacle-mounted peripherally placed prism button (see Fig. 6.7). The patient with binocular vision will report double vision when viewing through the prism button, but will now notice objects in the hemianopic field (Harris, 2011; Margolis, 2011). The Peli Lens Prism System uses Fresnel prism strips placed above and below the eye in cases of hemianopia (see Fig. 6.8), while a single strip is placed above the eye for a superior quadranopia and an inferior strip is placed below the eye for an inferior quadranopia

Fig. 6.7 Gottlieb Visual Field Awareness System for a left visual field defect (Harris, 2011). Republished with permission of Taylor and Francis Group LLC Books, from Chapter 7: The Use of Lenses to Improve Quality of Life Following Brain Injury, Paul A. Harris, 2011; permission conveyed through Copyright Clearance Center, Inc.

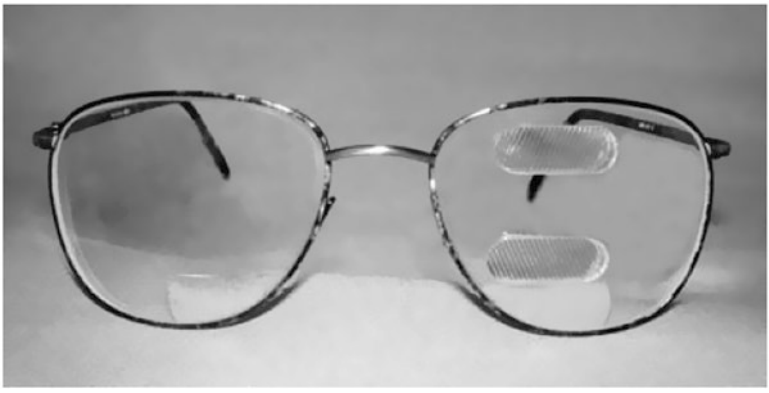


Fig. 6.8 Peli Lens Prism System for a left hemianopic visual field defect (Harris, 2011). Republished with permission of Taylor and Francis Group LLC Books, from Chapter 7: The Use of Lenses to Improve Quality of Life Following Brain Injury, Paul A. Harris, 2011; permission conveyed through Copyright Clearance Center, Inc.

(Margolis, 2011). It is a simultaneous viewing system and unlike the Gottlieb system, the patient notices constant diplopia superiorly and inferiorly of objects located within 20° into the affected field. If the patient has difficulty adapting, especially during ambulation, the inferior prism strip can be removed to minimize visual confusion from the double images. Gottlieb's Visual Field Awareness system showed a 79% acceptance rate in either full-time or part-time wear with a minimum of 18 months of experience with the system (Gottlieb, Freeman, & Williams, 1992). About 74% of patients accepted the Peli Lens Prism System at 6 weeks and then 47% of patients continued to wear the prisms even after 12 months because they reported the prisms were helpful in obstacle avoidance during ambulation (Bowers, Keeney, & Peli, 2008).

These partial or half-field Fresnel prisms are used to increase peripheral awareness (Suchoff and Ciuffreda, 2004; Suter, 1995). However, some patients do not respond

well as they tend to report diplopia peripherally. Clinically, this option is also most effective in cognitively intact patients. Alternatively, full field yoked prisms can also be prescribed for patients with hemianopic field defects and/or USI (Bansal, Han, & Ciuffreda, 2014; Margolis, 2011; Suchoff & Ciuffreda, 2004).

A retrospective study by Bansal et al. (2014) showed that yoked prism was primarily prescribed for patients with homonymous hemianopic/quadranopic visual field defects (58.3%). The most favorable response was "Increased awareness of the blind visual field; missing objects/words less frequently" in 33.3% of the cases; followed by "faster gait and improved balance" in 11.7% of patients. Any yoked prism amounts greater than 8.5^{Δ} were not tolerable due to lens distortion or optical aberrations, as reported in 8.3% of the patients.

Full field yoked prisms are also prescribed for patients with USI/visual neglect only. Bansal et al. (2014) showed that yoked prisms were prescribed in 11.7% of cases to address USI/visual neglect. When the ideal amount of prism is prescribed for USI, the patient reports that they "feel more grounded" or "the world is now moving with me" (Suchoff & Ciuffreda, 2004). USI patients typically will not utilize the sector prism systems due to their lack of awareness into their affected field (Margolis & Suter, 2006). Yoked prisms using magnitudes of 20^{Δ} will bring about observable changes, but may not be optically tolerated (Suchoff & Ciuffreda, 2004). Since significant improvement in visual neglect can be observed within 6 months of the incident, these patients may also benefit from being monitored rather than prescribing yoked prisms early in the rehabilitative process (Suchoff & Ciuffreda, 2004).

NORT may be recommended with or without a yoked prism prescription or with one of the field expansion prism systems. At times, patients will not report immediate subjective improvement, with the prism presented during the evaluation. So, the therapy program will concentrate on compensatory strategies, involving scanning techniques and maximizing visual information processing skills. Specifically, visual-spatial, visual speed of processing, and visual sequential memory are additional areas that should be incorporated into the patient's NORT program (Suter, 2007).

Compensatory strategies should be routinely recommended. Recommendations may include using a finger or brightly colored ribbon on the affected side of a book while the patient is reading. Typoscopes or rulers are helpful in keeping their place when reading. Despite adequate visual acuity, large print reading material will often improve reading speed, while the patient is receiving NORT. Alternatively, the patient can be prescribed a magnifier to enlarge normal-sized print and thereby improve reading fluency.

Visual Restoration/Restitution Therapy

The literature also describes a treatment modality called "visual restoration therapy" or "visual restitution therapy" (VRT), which restores and expands the visual field in patients with optic nerve disease and post-chiasmal brain lesions through

binocular stimulation of areas adjacent to the visual field defect that have residual vision, for example in patients with homonymous hemianopic defects (Poggel, Kasten, & Sabel, 2004; Reinhard et al., 2005; Sabel & Kasten, 2000). It involves a computer-based program, incorporating two half hour home training sessions daily for 6 months (Sabel & Kasten, 2000). Patients should be evaluated for severe binocular, accommodative (for patients under 45 years of age), and/or oculomotor dysfunctions as they may not be able to perform the training due to the onset of visual symptoms that may occur after 15–20 min of sustained near-vision activity. These conditions should be treated prior to starting a VRT treatment program to ensure maximum benefit from the therapy.

Most studies using VRT indicate subjective improvements in activities of daily living, such as reading, visual response time, visual attention, stimulus detection, visual confidence with mobility, and temporal visual processing (Mueller et al., 2003; Mueller, Mast, & Sabel, 2007; Reinhard et al., 2005). These improvements were not affected by the age of the patient, the type of visual field defect, and time since the brain injury (Mueller et al., 2007; Romano, Schulz, Kenkel, & Todd, 2008). Objective improvements were variable, with an average visual field increase of 5° with a range of 0–20°, an average expansion between 12.8% and 17.2%, using a suprathreshold stimulus, and an average of 12.5% expansion was seen using microperimetry, which monitor the degree of eye movements (Marshall et al., 2010; Mueller et al., 2007; Romano et al., 2008; Sabel & Kasten, 2000). Multiple studies have shown that eye movements are not responsible for the field expansion (Kasten, Bunzenthal, & Sabel, 2006; Marshall et al., 2010).

Functional magnetic resonance imaging (fMRI) studies show alteration in brain activity in response to a stimulus in an area of trained nonseeing visual field (border zone) versus an area of nontrained seeing visual field. The greatest responses were seen in the right inferior and lateral temporal, right dorsolateral frontal, bilateral anterior cingulate, and bilateral basal ganglia (Marshall et al., 2008). It has been shown that the degree of visual field expansion and the degree of subjective improvements reported by the patients do not match (Mueller et al., 2007; Sabel, Kenkel, & Kasten, 2004). Several authors suggest that there are complex reasons for this phenomenon, which are substantiated by documented changes in the neural substrate after training and changes, which occur when training is visual-spatially cued, with attention through top-down mechanisms (Poggel et al., 2004; Romano et al., 2008).

NORT Efficacy: Visual Field Defects

Rehabilitation of visual field defects without unilateral spatial neglect involves teaching compensatory scanning strategies, which incorporate the active training of exploratory saccadic eye movements into the area of field loss (Nelles et al., 2001; Reinhard et al., 2005). Efficacy of therapy is not influenced by hemianopic laterality, by lesion location, or by onset of the injury (Kerkhoff et al., 1994). NORT and occupational therapy follow this model of treatment. The literature, including a

systematic review, shows efficacious improvement in areas such as visual scanning, visual search, and functional performance in visually guided activities of daily living (Bouwmeester et al., 2007; Nelles et al., 2001). Bouwmeester et al. (2007) stated that the scanning compensatory therapy was more successful as compared to VRT because the strategies are simple, user friendly, and were applied to real-life situations. A 2012 randomized controlled trial concluded that a computerized visual search compensatory strategy was more effective than traditional occupational therapy and VRT to improve functional deficits after visual field loss (Mödden et al., 2012). Improving visual search abilities made more of an impact than small increases in the visual field. Significant improvements in reading and in activities of daily living were reported.

Abnormal Egocentric Localization/Visual Midline Shift Syndrome

Abnormal egocentric localization (AEL) or visual midline shift syndrome refers to the rotational, not lateral, shift in one's perception of "straight ahead" (Ciuffreda et al., 2016). The two types of shift are the left-right shift and the anterior-posterior shift (Padula, 1988b). This perceptual mismatch creates unusual visual-spatial symptoms as described below. The presence of these symptoms necessitates further evaluation. Some of the more common symptoms reported by a patient with abnormal egocentric localization are listed in Table 6.6.

According to Ciuffreda and Ludlam (2011), AEL is mostly associated with three types of brain injury cases: (1) patients with hemianopia, (2) patients with visual inattention/neglect, and (3) patients with TBI. It can also be clinically associated with vestibular dysfunctions, such as a vestibular neuritis. Approximately 30% of mTBI/TBI patients have AEL (Ciuffreda & Ludlam, 2011).

Yoked prisms are tried to determine what the best treatment option would be; yoked prisms alone, yoked prism and NORT, or NORT alone. Typically, low amounts of yoked prism are trialed at distance or near (Bansal et al., 2014; Ciuffreda & Ludlam, 2011). At times larger prisms can be trialed for near tasks as optical distortions would not be as noticeable as they would be with distance viewing. The patient who reports

Table 6.6 Visual symptoms associated with abnormal egocentric localization/visual midline shift syndrome

A sensation of not feeling grounded
Walking as though they were drunk
A sensation of the floor, ceiling, or walls tilting
Loss of balance
Poor posture
Face, head turn, or head tilt
Postural shifts/veering off when walking

that their vision is much clearer or dramatically notices a change in how they see will be prescribed the yoked prism. If the response is variable or no subjective improvement is noted, then NORT alone is the recommended treatment modality.

Padula (2012) has a protocol for yoked prism, prescribing specifically for the patient with a hemiparesis, who will posture their body as though there was an expansion of space on the affected side and a corresponding contraction of space on the unaffected side (see Fig. 6.9). As an example, a patient with a left hemiparesis will demonstrate a right visual midline shift. The optical characteristics of the prisms will perceptually alter the patient's posture because the prism apex perceptually expands visual space and the base compresses visual space and the patient will then respond to these perceived changes in their visual environment.

Efficacy of yoked prisms in patients with abnormal egocentric localization or visual midline shifts has been founded on alterations in body posture noted upon clinical observations as well as upon quantitative assessments (Kapoor et al., 2001; Padula, 2012). Center of gravity changes have been documented in young, healthy subjects without history of an ABI, using dynamic posturography (Gizzi, Khattar, & Eckert, 1997). Adaptive neurological changes have also been observed after 2 h of wear in patients with right hemispheric lesions (Rosetti et al., 1998). Interestingly, yoked prisms were found to improve higher cognitive levels, particularly with respect to "mental space representation." Rode, Rossetti, and Boisson (2001) found that patients with visual neglect showed improved performance on tasks related to mental imagery. About 24 h after discontinuing the yoked prism, the patients' improvements decreased but not to previous levels, indicating that the prisms also facilitated the learning process during the mental imagery task. The authors suggested that yoked prisms influence plasticity of multisensory integration processes, as well as cognitive processes related to mental representation of visual space.

Visual-Vestibular Status

Poor balance occurs when there is a mismatch in the inputs received from the vestibular, visual, and proprioceptive systems. In the presence of a vestibular dysfunction, inputs from the other two systems become more critical in maintaining balance. In visually busy environments (i.e., supermarkets, crowded streets, and moving trains), patients may experience extreme forms of dizziness because they are unable to suppress the excessive visual movements occurring in the background (Hellerstein & Freed, 1994).

Many of the visual-vestibular dysfunctions relate to the phenomenon of the vestibulo-ocular reflex (VOR). The VOR depends on a stable bifoveal retinal image during high-frequency head movements (greater than 2 Hz), while the cervico-ocular reflex (COR) stabilizes vision during low frequency head movements based upon sensory inputs from the neck and facet joints (Rosen et al., 2001). Patients with VOR deficits experience oscillopsia, blurred vision, decreased dynamic visual acuity, poor depth perception, and/or diplopia (Weiss, 2002). Therefore, deficits in

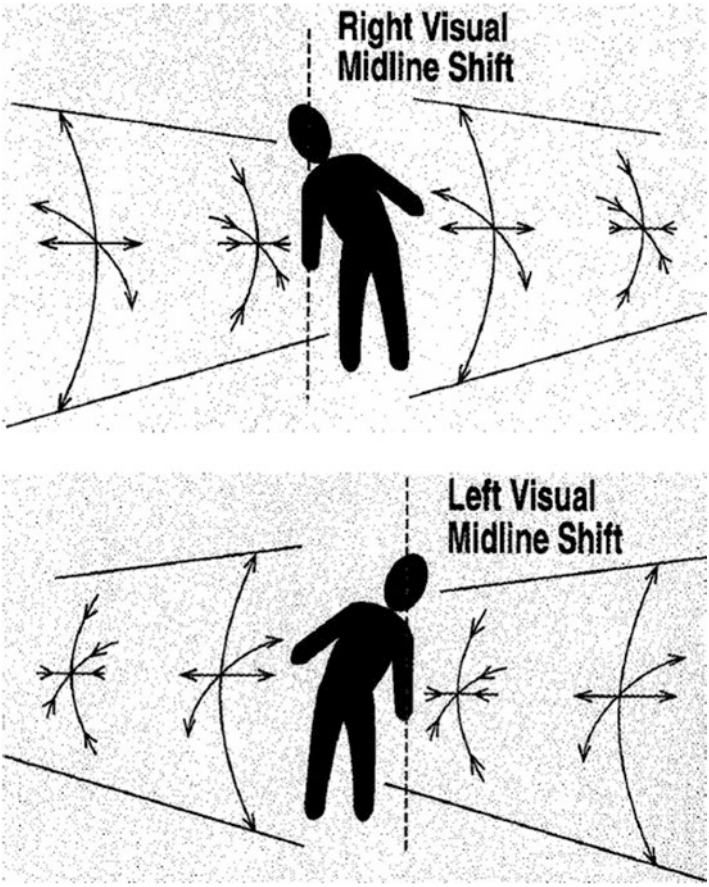


Fig. 6.9 Visual midline shift to the right and left (Padula, 1988b)

oculomotor, binocular, and accommodative function are associated with symptoms of vestibular dysfunction due to a mismatch of visual and vestibular inputs, creating a sense of imbalance. Visual problems will often exacerbate a vestibular problem as evidenced clinically, when patients complete their NORT program and report a decrease in the severity in their vestibular symptoms.

Patients with vestibular dysfunction, as stated by Rosen et al. (2001), often use other sensory modalities such as the cervico-ocular reflex (COR), pursuit and saccadic eye movements to obtain vestibular information. This suggests the need for optimal levels of vision function in the presence of a vestibular dysfunction. Similarly, patients with vestibulospinal reflex (VSR) dysfunction experience postural instabilities that worsen with movement or in areas of poor lighting or uneven surfaces (Rosen et al., 2001). In either VOR or VSR dysfunction, the goal of vision rehabilitation would be to strengthen the patient's ability to rely more on visual input rather than vestibular input. Some commonly associated vision symptoms reported by patients having a vision-vestibular dysfunction are listed in Table 6.7.

Table 6.7 Visual symptoms associated with visual-vestibular deficits

Light-headedness or heavy-headedness
Headache
Vertigo
Motion sickness
Swimmy, fuzzy, and foggy vision during head movement
Feeling disoriented
Sensation that the world is moving
Fullness in the ears
Floating feeling
Nausea
Fatigue
Avoidance of movement or changes in head posture
Jumpy, bouncing, or jerking vision
Blurred vision with head movement

Table 6.8 Visual symptoms associated with visual perceptual deficits

Confuses similarly shaped objects
Difficulty recognizing objects in different orientations
Difficulty recognizing objects close to or overlapping other objects
Visual memory problems
Difficulty following verbal directions
Often asks to repeat instructions

Visual Perceptual Status

Visual perceptual skills or visual information processing skills are required to interact with the environment in a meaningful way. Tests should evaluate for visual memory (both spatial and sequential), visual speed of processing, visual discrimination, visual form perception, visual figure/ground, visual spatial orientation, visual spatial relations, visual perceptual organization, visual motor integration, and visual attention (Groffman, 2011; Han, 2007). Many of these skills will be evaluated in a formal neuro-psychological evaluation when a cognitive assessment is recommended. A formal visual perceptual evaluation can be administered when specific activities of daily living are affected, especially when NORT is being recommended. In these cases, the neuro-optometrist wants to know the severity of the deficits and needs to clinically monitor progress in therapy.

Before attempting to treat visual perceptual deficits, the refractive and sensorimotor status (oculomotor, binocular, and accommodative, if indicated) need to be extensively assessed to determine whether the patient can efficiently and clearly input visual information before he/she can process the information. For example, a patient seeing constant double vision may show some of the symptoms listed in Table 6.8 only because he/she sees two images. This individual may have trouble

walking or picking up objects and would have significant trouble with higher level cognitive processing.

Visual attention is also an important aspect in successfully performing visual tasks. Warren (1993) summarized the process of visual attention as a three-step process, in which the first step involves a disengaging operation, in which the eye ceases to attend to an object, then there is a moving operation in which attention is shifted to a new object, and finally there is a comparing operation, in which the previous object is compared with the new one for similarities and differences. These steps all require adequate sensorimotor function and intact visual perceptual abilities.

Depending on the severity of the ABI, there is a poor prognosis for the complete remediation of visual perceptual deficits. The goal of therapy is therefore to create compensatory strategies that maximize performance in activities of daily living. Auditory strategies, repetition strategies, and different viewing perspectives are practiced, and the patient's initial visual symptoms are carefully monitored for decreases in frequency.

Photosensitivity/Light Sensitivity

Patients with photosensitivity are not photophobic, which is typically an indication of an ocular inflammatory condition. Specifically, for TBI patients, discomfort associated with normal illumination includes the following: atypical light sensitivity, photic-driven headache, and reduced or prolonged dark adaptation (Jackowski, 2001). Light sensitive patients clinically report sensitivity to indoor lighting, in addition to outdoor sunlight. They may be more sensitive to only fluorescent or only to incandescent lighting. Fluorescent lighting emits light at different wavelengths (Smith, 1999). Some of the emitted light is in the UV spectrum and is normally undetectable to the eye, but patients with ABI, especially TBI patients, report more sensitivity to fluorescent as opposed to incandescent lighting.

Jackowski (2001) summarized that 44% of TBI patients report discomfort to bright illumination and a greater frequency showed a decreased threshold for illumination tolerance. Zihl and Kerkhoff (1990) also showed that 39% of TBI patients reported symptoms related to impaired light adaptation. A retrospective study showed that 30–60% of TBI and 18–30% of CVA patients report symptoms of light sensitivity (Craig et al., 2008). A retrospective study on patients with mild TBI by Truong et al. (2014) reported that 10% of patients reported a reduction in their photosensitivity symptoms within 1-year post-injury and 40% after 1-year post-injury. It was also postulated that wearing tinted lenses may slow down the neural adaptation process as the patients continued to wear the tints. In addition, contact lens wearers adapted sooner and reported a decrease in photosensitivity at a higher rate.

Other factors associated with light sensitivity include altered or tonic pupillary diameters or highly fluctuating pupillary responses. Patients with abnormal pupillary responses report sensitivity to all types of intense lighting (Jackowski, 2001). Anomalous patterns of dark adaptation and deficits in peripheral processing systems

possibly exist in TBI patients with photosensitivity (Rosetti et al., 1998). Interestingly, mTBI patients, who report photosensitivity, will demonstrate normal contrast sensitivity levels (Jackowski et al., 1996). Sources confirm a post-retinal explanation rather than a retinal etiology for symptoms of photosensitivity in TBI patients with normal retinal functioning (Du et al., 2005; Zihl & Kerkhoff, 1990).

Much of the research related to the neurological pathways is related more to photophobia and not necessarily photosensitivity as reported by brain injury patients. Sensory pathways around the eye and the orbit generally involve trigeminal and autonomic nervous system innervation. But there are also photosensors called the intrinsically photosensitive retinal ganglion cells (IPRGC) that are nonimage-forming retinal ganglion cells that modulate the circadian cycle (Digre & Brennan, 2011). These cells contain melanopsin, not rhodopsin, and make up 1–3% of the retinal ganglion cells. Instead of following the normal visual pathway from the optic nerve to the lateral geniculate nucleus and then to the visual cortex, the IPRGC, instead, makes connections to the somatosensory and pain areas of the thalamus (Noseda et al., 2010).

The most basic form of management for these patients is a lightly tinted lens for indoor use. Patients with a general sensitivity to light will clinically respond best to a 25–35% brown or gray tint, while the patients with sensitivity to fluorescent lighting may respond better to 15–25% light blue or gray tint. Post-concussive patients reporting photosensitivity will clinically choose the light blue tint. The outdoor tint should be 85–90% dark, using the colors described above. Patients with abnormal pupillary responses and photosensitivity respond best to neutral gray filters (Jackowski, 2001). Studies using CPF-450-S (yellow) filters demonstrated enhanced contrast sensitivity, improved reading rates (not to normal levels), and elimination of hypersensitive responses to light under certain conditions (Jackowski, 2001).

Special lens coatings or lenses, such as antireflective coatings or polarizing lenses, will reduce the effects of glare and excessive reflections. For some patients, polarizing sunglasses significantly decreases the effects of glare from light reflecting off windshields, water, snow, and wet highway surfaces (Brooks & Borish, 1996). Polarized lenses only permit light waves oriented in a specific direction to pass through the lens and enter the eye, resulting in reduction of glare and improved comfort for the photosensitive patient (Brooks & Borish, 1996). Clip-on sunglasses and brimmed hats are also very helpful in increasing patient comfort in brightly lit environments.

Colored Filters and Light Therapy Considerations

Recently, management options for visual discomfort, visual perceptual distortions (that may arise from viewing repetitive patterns or stripes such as text), and for visual snow have included the use of precision ophthalmic tints and/or syntonic phototherapy. Much of the research regarding colored tints/filters or colored overlays to decrease vision symptoms is based upon patients with migraines, with visual

auras, and photophobia. Cortical activity can be altered using red versus blue light; red light suppresses visually provoked beta brain activity in migraineurs. Overall, red tints are more visually comfortable for migraine patients and a tint called Fl-41 (rose-colored) has been shown to even reduce the frequency of migraines in children (Digre & Brennan, 2011; Good, Taylor, & Mortimer, 1991). Precision ophthalmic tints utilize the Intuitive Colorimeter to determine a specific color wavelength tint that is most comfortable for the patient. The appropriate tint for migraine patients was shown to normalize visual cortical activation based upon function magnetic resonance imaging (fMRI), as compared to gray tinted lenses (Huang et al., 2011). Of interest is that IPRGCs are sensitive to the shorter wavelengths or blue color (Digre & Brennan, 2011).

Syntonic phototherapy's role in NOR is increasing due to the diffuse nature of the damage causing unpredictable and variable responses to different treatment modalities. Some patients are unable to visually tolerate therapy due to the severity of their symptoms, while other patients progress in therapy may plateau even though they are still experiencing vision symptoms. Their visual function has improved and there is a decrease in the frequency and severity of their symptoms, but the patient has not fully recovered. In cases such as these, alternative treatment modalities are considered. Syntonic phototherapy works on the concept of balancing the autonomic nervous system through direct retinal input and subsequently affecting the thalamic and hypothalamic regulatory centers (Gottlieb & Wallace, 2010). The structures responsible for pupillary and accommodative function are innervated by the autonomic nervous system (Friedman, 1997; Kaufman, 1992; Thompson, 1992). The sympathetic pathway is responsible for pupillary dilation as well as relaxing accommodation, while the parasympathetic pathway is responsible for pupillary constriction and increasing accommodation. The evaluation involves pupillary assessment and functional color field testing or campimetry, in addition to the NOR evaluation. The treatment involves looking at prescribed color filtered light for less than 20 min for about 20 treatments (Gottlieb & Wallace, 2010; Stern, 2011). Clinically, the treatment can be done prior to therapy, in parallel with therapy, or once a plateau is reached with NORT.

Case Reports

The following cases are examples of the visual functional outcomes of patients who received conventional neuro-optometric rehabilitation therapy (NORT) as part of their overall treatment recommendations. NORT is a modality that treats deficits of binocular, accommodative, oculomotor, and visual perceptual function, by gradually conditioning a patient's vision capabilities in each of the areas listed above and then training the patient to integrate these areas to perform activities of daily living more effectively. This is accomplished through the use of lenses, prisms, mirrors, occlusion, filters (polarized, anaglyphic, and colored), computer programs, and other visual-motor and perceptual-motor activities, which incorporate basic

physiological and optical principles that are inherent to the training of normal vision processes (Ciuffreda, 2002; Scheiman & Wick, 2014).

NORT typically involves a combination of in-office and home-based therapy activities. Generally, patients receive an individualized program of training activities specific to their abilities and to their progress. Therapy can be performed on an individual basis or in a group setting as recommended by the evaluating neuro-optometrist. The activities may be done directly with the optometrist or with a trained vision therapist in a session supervised and programmed by the neuro-optometrist.

Patient 1 is a 58-year-old male who was referred for a vision rehabilitation evaluation by his vestibular therapist for the following vision-related complaints: intermittent blurred vision, reading difficulties, poor attention when reading, difficulty distinguishing lines and spaces between lines, experiencing eyestrain after 3–4 pages of reading, words appearing to swim and overlap when reading, double vision when watching television, frontal-temporal headaches when reading, eyes tearing with sustained reading, difficulty keeping left eyelid open, light sensitivity to indoor lighting and sunlight, and difficulty using laptop computer for sustained periods. Patient 1 also reported difficulty progressing with his vestibular therapy, anxiety and severe dizziness in crowded situations, dizziness and nausea when viewing moving objects, frequent loss of balance, and inability to look at a moving train.

Etiology

Patient 1 was diagnosed with a pituitary microadenoma that was carefully being monitored and treated with medications. He reported a long-standing central vestibular dysfunction.

Pertinent Visual Findings

1. Refractive status: Patient 1 manifested a moderate hyperopic and astigmatic prescription in each eye and was also found to be presbyopic. Best-corrected distance visual acuities were 20/20 in the right and left eyes.
2. Sensorimotor status:

   Oculomotor function: Fixation abilities in the left eye revealed an intermittent jerk nystagmus. His left eye also demonstrated poorer quality of movement with more losses of fixation noted during pursuit and saccadic eye movement testing.

   Binocular function: Patient 1 manifested esophoria and a right hyperphoria at distance. At near, testing revealed a convergence insufficiency and right hyperphoria. His compensatory fusion reserves at both distance (base-in or

divergence ranges) and near (base-out or convergence ranges) were found to be restricted and inadequate for his needs.

3. Visual field status: Patient 1 did not show the typical bitemporal visual field defects associated with pituitary tumors. He did exhibit scattered visual field defects.

Assessment

1. Convergence insufficiency
2. Deficits of pursuit and saccades
3. Photosensitivity
4. Hyperopia, astigmatism, and presbyopia

Recommendations

1. Vision rehabilitation was strongly advised to remediate deficits in his oculomotor and binocular function. Additional goals included increasing the flexibility of his oculomotor and binocular abilities to more comfortably respond to complex visual and moving stimuli. Patient 1's progress in vestibular therapy will be carefully monitored to determine the presence of visual obstacles to his progress.
2. Four pairs of spectacles were advised: (1) polarized prescription sunglasses with a progressive multifocal lens design for outdoor use, (2) progressives with a 35% gray tint and antireflective coating for indoor use, (3) single-vision spectacles with 35% gray tint and antireflective coating for computer use, and (4) single-vision spectacles with 35% gray tint and antireflective coating for prolonged near-vision use, such as reading.
3. Until his visual skills improve, a referral for a low vision magnifier (4×) was advised, so that he will be more comfortable when reading his smaller print books during the early stages of his vision rehabilitation.

Outcome

After completing his vision rehabilitation, Patient 1 reported that he was able to read for much longer periods without the use of his magnifier, use his computer for sustained periods of time, drive confidently at night and in poor weather conditions (snow, rain, and overcast days), and continues to progress in his vestibular therapy program. Patient 1 is currently being followed every 3 months to monitor his visual symptoms. His complicated systemic health history and changes in medications or medical health significantly affects his vestibular and vision function. His refractive

status frequently changes, requiring a new prescription for all four pairs of glasses. In addition, there are times when he requires horizontal and vertical fusional prism for his intermediate and distance diplopia, and there are periods when he does not require it for optimal visual function. Patient 1 is a unique case that was successfully treated with neuro-optometric rehabilitation therapy. However, his complicated medical health requires frequent follow-up evaluations to monitor his visual function.

Patient 2 was a 76-year-old woman referred by an occupational therapist at the inpatient rehabilitation center she attended immediately after her stroke. She experienced the following visual complaints: difficulty reading, poor awareness to the left side (not noticed by patient), decreased peripheral vision to the left, poor visual scanning abilities, bumping into things on the left side, past pointing, and sitting with body shifted to the right. Patient 2 also reported that she was unable to write after the stroke because she wrote with her left hand.

Etiology

Patient 2 experienced a stroke and demonstrated weakness of the upper and lower extremities on the left side. Initially, she was wheelchair bound.

Pertinent Visual Findings

1. Refractive status: Patient 2 manifested a very high myopic prescription with poor visual acuities due to the presence of dense cataracts in each eye. Her best-corrected distance acuities ranged from 20/80 to 20/100 in each eye. Her ophthalmologist followed her every 6 months for the cataracts. She was apprehensive about the surgery and chose to have the cataracts monitored.
2. Sensorimotor status:

   Oculomotor function: Patient 2 demonstrated increased latency in the initiation of her pursuit and saccadic eye movements, particularly to the left side. Standardized testing revealed slow speed, poor accuracy, and difficulty in finding the beginning of the line.

   Binocular function: Patient 2 showed restricted fusion ranges at both distance and near. She also exhibited poor awareness of double vision, which can also be attributed to her poor visual acuities.

3. Visual field status: Left homonymous hemianopia with neglect. Yoked prism (6^{Δ} base left) were trialed and she was able to read faster and subjectively reported she was able to see more to the left. Objectively, Patient 2's body posture immediately improved, and she did not lean to the right while wearing the yoked prisms. Her improved posture was maintained at her 1-month follow-up examination.

Assessment

1. Visual field defect
2. Oculomotor dysfunction
3. Cataracts
4. Myopia and presbyopia

Recommendations

1. The yoked prisms were prescribed and incorporated into her distance prescription. She was advised to keep her old lenses, as patients sometimes report less comfort with the yoked prisms with time as they progress in their other therapies (physical and occupational). A 1-month follow-up was advised to monitor her symptoms and vision findings.
2. Vision rehabilitation was advised at the follow-up visit to improve oculomotor abilities, specifically visual scanning, and accuracy.
3. Large-print reading material was advised as a compensatory strategy because of her decreased acuities, oculomotor deficits, and visual field deficits.

Outcome

Patient 2 attended ten vision rehabilitation sessions twice a month with good compliance with home vision therapy. She reported the following improvements: improved eye-hand coordination, improved ease with reading, less symptoms of visual neglect, increased visual awareness, and less past-pointing. Patient 2 was discharged with maintenance home vision therapy activities, as she indicated that she was scheduled for cataract surgery. A progress vision evaluation was scheduled for 6 months to allow for her eyes to adequately recover from the surgery. Vision rehabilitation will be continued, if indicated at that point.

Patient 3 was an 11-year-old girl evaluated for the following vision complaints: constant blurred distance vision, intermittent near-vision blur, severe headaches, loss of place when reading, using her finger to read, intermittent double vision when reading, dizziness when experiencing headaches, and fatigue after 30 min of schoolwork. Academically, she was strong prior to the TBI and grades worsened afterward. She also reported that her vision worsened since the accident.

Etiology

TBI due to a ceiling collapse.

Pertinent Visual Findings

1. Refractive status: Patient 3 initially did not bring her distance glasses, which she first received 3 months prior to this evaluation. She manifested a low myopic and astigmatic prescription. Best-corrected distance visual acuities were 20/20 in each eye.
2. Sensorimotor status

   Oculomotor function: Patient 3 exhibited saccadic intrusions in the left eye with fixation, and increased latency with pursuit movements in the left eye. NP also showed poor performance on standardized tests of oculomotor function.

   Binocular function: Patient 3 manifested an intermittent exotropia at near, with severely low fusion ranges at both distance and near. She also exhibited a right hyper deviation at both distance and near.

   Accommodative function: Patient 3 showed severe deficits in her accommodative function, particularly with respect to her sustaining ability.

Assessment

1. Intermittent exotropia
2. Oculomotor dysfunction
3. Accommodative insufficiency
4. Myopia

Recommendations

1. Single-vision distance prescription should be worn for distance use only. Prolonged near-vision work should be done without spectacles.
2. Vision rehabilitation was strongly advised to decrease frequency of visual symptoms.

Outcome

Patient 3 attended 20 sessions of vision rehabilitation with the following improvements noted: less double vision when reading, fewer headaches, less loss of place when reading, and no mention of dizziness. Overall, she was more confident academically. Upon reevaluation, her vision dysfunctions were fully resolved.

Conclusion

A neuro-optometric rehabilitation evaluation and subsequent management begins with an understanding of the diffuse and unpredictable nature of brain injury. Based upon the prevalence of binocular, oculomotor, and accommodative deficits, patients with ABI demonstrate significant degrees of dorsal and ventral stream deficits, which contribute to their symptomatology. The ventral stream pathway is clinically assessed, when evaluating the patient's refractive status and accommodative function. The dorsal stream pathway is clinically assessed with oculomotor and vergence/binocular function testing (Sanet & Press, 2011).

Dorsal stream deficits are generally more prevalent, and the rehabilitation protocol will reflect these pathway-specific deficits, as well as incorporate more multisensory integrative, top-down emphasized therapy procedures as the patient progresses through therapy (AOA, 2019). The patient will be able to multitask better and ambulate through a multisensory environment with greater ease after completing their NORT program. Depending on the nature of the injury, therapy is typically initiated and specifically tailored with the goal of maximizing visual function. Each case is unique, and it is often difficult to estimate prognosis in brain-injured patients, as compared to noninjured patients with the same diagnoses.

Sanet and Press (2011) describe neuro-optometric rehabilitation therapy (NORT) as incorporating more cognitively demanding procedures "to correct spatial misperception, enhance spatial localization, and develop more accurate visual spatial maps." This is a more comprehensive approach and will help the patient transfer these new skills quickly and effectively to their activities of daily living. Sanet and Press (2011) add that patients "must have the visual-spatial understanding of where to point and focus the eyes in space to most effectively direct the neuromuscular substrates necessary to perform the mechanics of the task."

The literature strongly emphasizes the need for ABI patients to undergo a comprehensive vision evaluation (Sabates et al., 1991; Suchoff et al., 1999). These authors indicate that most vision symptoms can be resolved with the appropriate refractive correction at distance and near. Additionally, lightly tinted lenses, prisms, and occlusion recommendations are management options for ABI patients who may be experiencing poor progress in their rehabilitation programs as a direct consequence of their undiagnosed vision dysfunctions. Sabates et al. (1991) stated that many patients with vision problems may go undiagnosed for months to years after their injury.

Members of the rehabilitation team should readily recognize those patients suffering from vision dysfunctions secondary to their brain injury. Recognizing vision dysfunction is very difficult for those not specializing in vision because there is no visible sign, such as a broken limb or a red eye, for the provider to see and then make the appropriate referral. In most cases, vision deficits can only be detected based upon direct complaints from the patient, neuropsychological or occupational therapy assessments, or observations made by those working regularly with the patients. Consequently, a brief history including vision symptoms, or a symptom

Table 6.9 Post-traumatic vision syndrome (Padula, 1988b)

Eyestrain headaches
Double vision
Focusing inability
Poor fixation and tracking
Decrease in color function
Staring behavior
Poor visual memory
Glare sensitivity
Balance, coordination, and postural deficits

survey tool should routinely be a part of every evaluation of an individual post injury, since most patients may not consider the importance of their vision in the rehabilitative process. Table 6.9 summarizes the common vision symptoms reported by ABI patients. The common diagnoses and findings that will be reported to the referring provider may include an accommodative dysfunction, convergence insufficiency or exotropia, low blink rate, poor fixation, poor pursuit and saccadic eye movements, and unstable peripheral processing abilities.

Glossary of Clinical Terms (Cline, Hofstetter, & Griffin, 1989; Han, 2007; Kapoor & Ciuffreda, 2005)

Accommodation The ability to change focus and maintain a clear image of an object (when looking from far to near and vice versa), using the eye's crystalline lens-based mechanism.

Accommodative amplitude The closest point of clear vision that is typically measured monocularly.

Accommodative infacility A condition in which slow or difficult accommodative responses are observed in response step changes in lens power.

Accommodative insufficiency A condition in which the measured amplitude of accommodation is less than expected given the patient's age.

Astigmatism Unequal refractive error in orthogonal meridians of the eye. Rays of light from infinity come to a focus at two different distances relative to the retina, with accommodation minimally stimulated.

Binocular Viewing with two eyes at the same time.

Contrast sensitivity The ability to detect threshold contrast targets. Measuring contrast sensitivity measures the ability to resolve spatial properties across a range of spatial frequencies and levels of contrast (Warren, 1993).

Convergence The turning inward of the eyes toward each other.

Convergence excess A condition in which esophoria is greater at near than far.

Convergence insufficiency The condition in which exophoria is greater at near than far, with a receded near point of convergence and reduced relative fusional convergence at near.

Diplopia The condition in which a single object is perceived as two objects rather than one; double vision.

Divergence A deviation or relative movement of the two eyes outward from parallelism.

Divergence excess A condition in which exophoria is greater at distance than near and often associated with an exotropia.

Divergence insufficiency A condition in which there is greater esophoria at distance than near, and is often associated with esotropia.

Emmetropia Essentially no refractive error present.

Esophoria A condition in which the two eyes intersect in front of the plane of regard when fusion is disrupted.

Exophoria A condition in which the two eyes intersect beyond the plane of regard when fusion is disrupted.

Exotropia A type of strabismus in which the nonfixating eye is turned outwards.

Fixation Ocular alignment with the image of the fixated target falling on the fovea; may be performed one eye at a time (i.e., monocularly) or with both eyes at the same time (i.e., binocularly).

Fusion Single, cortically integrated vision under binocular viewing conditions.

Fusional prism The amount of prism an individual can fuse.

Fusional range The range over which the vergence system can be stimulated by the addition of prisms binocularly and still maintain single, binocular vision at both distance (6 m) and near (40 cm). Three parameters are recorded: the first is the amount of prism at which the patient reports blurred vision; the second is the amount of prism at which the patient reports diplopia; and, the third is the amount of prism at which the patient regains fusion.

Hemianopia Hemi-field visual field defect, which may be unilateral or bilateral (i.e., homonymous or bitemporal).

Hyperopia Far-sightedness; when rays of light from infinity come to a focus behind the eye, with accommodation minimally stimulated.

Monocular Viewing with one eye at a time.

Myopia Near-sightedness; when rays of light from infinity come to a focus in front of the eye, with accommodation minimally stimulated.

Nystagmus Rapid involuntary oscillation or movement of the eyes, the presence or absence of which may be diagnostic of neurological and vision disorders.

Oculomotor When the eyes move (includes fixation, pursuit, and saccade) to follow targets moving laterally, vertically, or obliquely in one plane, with no change in depth.

Oscillopsia Illusory movement of the world generally related to vestibular dysfunction.

Presbyopia Normal age-related, physiological loss of focusing ability.

Prism A lens that deviates the path of light as it passes through it. A prism is characterized by the base and the apex and is described by the direction of the base. Prisms move images away from the base and towards the apex. Prisms are measured in units called prism diopters. One prism diopter indicates that the image is moved by 1 cm at a distance of 1 m (Brooks & Borish, 1996).

Pursuit Slow, continuous, and conjugate eye movement used when the eyes follow an object as it is moved slowly and smoothly.

Refractive status The degree to which images on the retina are not focused.

Saccade Rapid, step-like conjugate eye movement that redirects the line of sight from one position to another.

Strabismus An anomaly of binocular vision in which one eye fails to intersect an object of regard.

Stereopsis Relative depth perception based on horizontal retinal image disparity.

Vergence When the two eyes move to track targets moving in depth.

Versional eye movements When the two eyes move (includes fixation, pursuit, and saccade) to follow targets moving laterally, vertically, or obliquely in one plane, with no change in depth.

Vestibulo-ocular reflex (VOR) Rapid, reflex movement of the eyes, which functions to counteract head movements and maintain stable gaze on an object.

Yoked prism Prisms with bases oriented in the same direction.

Appendix 1: American Optometric Association (AOA)—Convergence Insufficiency Symptom Survey (CISS) (American Optometric Association, 2019)

Convergence Insufficiency Symptom Survey (CISS)

Name: _____ Date: ___/___/_____

Clinician/Assistant instructions: Pose the following questions exactly as written. If the patient responds with "yes" - please qualify with frequency choices. Do not give examples.

Patient instructions: Please answer the following questions about how your eyes feel when reading or doing close work.

Possible Subjective Symptoms	Frequency				
	Never (0)	Infrequently/ not very often (1)	Sometimes (2)	Fairly often (3)	Always (4)
1. Do your eyes feel tired when reading or doing close work?					
2. Do your eyes feel uncomfortable when reading or doing close work?					
3. Do you have headaches when reading or doing close work?					
4. Do you feel sleepy when reading or doing close work?					
5. Do you lose concentration when reading or doing close work?					
6. Do you have trouble remembering what you have read?					
7. Do you have double vision when reading or doing close work?					
8. Do you see the words move, jump, swim or appear to float on the page when reading or doing close work?					
9.Do you feel like you read slowly?					
10. Do your eyes ever hurt when reading or doing close work?					
11. Do your eyes ever feel sore when reading or doing close work?					
12. Do you feel a "pulling" feeling around your eyes when reading or doing close work?					
13. Do you notice the words blurring or coming in and out of focus when reading or doing close work?					
14. Do you lose your place while reading or doing close work?					
15. Do you have to re-read the same line of words when reading?					
Total score _____	____ x 0	____ x 1	____ x 2	____ x 3	____ x 4

For Children (< age 21) total score = **16 or higher** is suggestive of convergence insufficiency.
For Adults total score = **21 or higher** is suggestive of convergence insufficiency.

Reference: Borsting EJ, Rouse MW, Mitchell GL, et al and the CITT group. Validity and reliability of the revised convergence insufficiency symptom survey in children. Optometry and Vision Science 2003; 80(12):832-838.

Appendix 2: Post Trauma Vision Symptoms (PTVS)

TBI/VISUAL SYMPTOMS REQUIRING OPTOMETRIC REFERRAL

PATIENT NAME: _____

MRN: _____

Please consider each symptom and rate the presence of symptoms by marking:

0: If the symptom is not present
1: If the symptom is rarely present
2: If the symptom if occasionally present
3: If the symptom if frequently present

Post Trauma Vision Symptoms (PTVS)

	Degree symptom is present			
SYMPTOM	0	1	2	3
Emergent Visual Conditions				
Flashes of light				
Floaters in field of vision				
Restricted field of vision				
"Curtains" billowing into field of view				
Urgent Visual Conditions				
Inability to completely close eyes				
Difficulty moving or turning eyes				
Pain with movement of the eyes				
Pain in or around eyes				
Wandering eye				
Double vision				
TBI/ABI Optometric Vision Rehabilitation Conditions				
Blurred vision for distance viewing				
Blurred vision for near viewing				
Slow shift of focus from near to far to near				
Difficulty copying or taking notes				
Pulling or tugging sensation around eyes				
Discomfort/eyestrain while reading				
General fatigue while work/reading				
Unable to sustain near work or reading for periods of time				
Eyes get tired while reading				
Covering, closing one eye				
Headaches while reading				
Loss of place while reading				
Missing a portion of their vision				
Bumping into objects or not seeing objects on one side more so than other				
Easily distracted when reading				
Slower speed of reading				
Decreased attention span				
Reduced concentration ability				
Difficulty remembering what has been read				
Disorientation				
Loss of balance				
Poor posture				
Face, head turn or head tilt				
Bothered by movement in environment				
Bothered by crowded environments				
Light sensitivity				
A sensation of the floor, ceiling or walls tilting				
Dizziness				
A sensation of the room spinning				
A sensation of not feeling grounded				
Postural shifts/veering off when walking				
TOTAL				

Appendix 3: Brain Injury Vision Symptom Survey (BIVSS) (Laukkanen et al., 2017)

My brain injury was: ___ years ago My age is: ___ years today's date: _____

☐ I have had a medical diagnosis of brain injury (check box if true) Cause of injury: _____

☐ I sustained a brain injury without medical diagnosis (check box if true) _____

☐ I have NOT ever sustained a brain injury (check box if true)

Please check the most appropriate box, or circle the item number that best matches your observations. All information will be held in confidence. Thank you for your help!

SYMPTOM CHECKLIST *Circle a number below:*

Please rate each behavior. **How often does each behavior occur?** (circle a number)	Never	Seldom	Occasionally	Frequently	Always
EYESIGHT CLARITY					
Distance vision blurred and not clear -- even with lenses	0	1	2	3	4
Near vision blurred and not clear -- even with lenses	0	1	2	3	4
Clarity of vision changes or fluctuates during the day	0	1	2	3	4
Poor night vision / can't see well to drive at night	0	1	2	3	4
VISUAL COMFORT					
Eye discomfort / sore eyes / eyestrain	0	1	2	3	4
Headaches or dizziness after using eyes	0	1	2	3	4
Eye fatigue / very tired after using eyes all day	0	1	2	3	4
Feel "pulling" around the eyes	0	1	2	3	4
DOUBLING					
Double vision -- especially when tired	0	1	2	3	4
Have to close or cover one eye to see clearly	0	1	2	3	4
Print moves in and out of focus when reading	0	1	2	3	4
LIGHT SENSITIVITY					
Normal indoor lighting is uncomfortable – too much glare	0	1	2	3	4
Outdoor light too bright – have to use sunglasses	0	1	2	3	4
Indoors fluorescent lighting is bothersome or annoying	0	1	2	3	4
DRY EYES					
Eyes feel "dry" and sting	0	1	2	3	4
"Stare" into space without blinking	0	1	2	3	4
Have to rub the eyes a lot	0	1	2	3	4
DEPTH PERCEPTION					
Clumsiness / misjudge where objects really are	0	1	2	3	4
Lack of confidence walking / missing steps / stumbling	0	1	2	3	4
Poor handwriting (spacing, size, legibility)	0	1	2	3	4
PERIPHERAL VISION					
Side vision distorted / objects move or change position	0	1	2	3	4
What looks straight ahead--isn't always straight ahead	0	1	2	3	4
Avoid crowds / can't tolerate "visually-busy" places	0	1	2	3	4
READING					
Short attention span / easily distracted when reading	0	1	2	3	4
Difficulty / slowness with reading and writing	0	1	2	3	4
Poor reading comprehension / can't remember what was read	0	1	2	3	4
Confusion of words / skip words during reading	0	1	2	3	4
Lose place / have to use finger not to lose place when reading	0	1	2	3	4

References

American Optometric Association. (2019). *Penlight red/green (PLRG) procedure for screening of convergence insufficiency*. Retrieved May 3, 2019, from https://www.aoa.org/documents/optometrists/PLRG-CI-Card.pdf

Bansal, S., Han, E., & Ciuffreda, K. J. (2014). Use of yoked prisms in patients with acquired brain injury: A retrospective analysis. *Brain Injury, 28*(11), 1441–1446.

Bouwmeester, L., Heutink, J., & Lucas, C. (2007). The effect of visual training for patients with visual field defects due to brain damage: A systematic review. *Journal of Neurology, Neurosurgery, and Psychiatry, 78*, 555–564.

Bowers, A. R., Keeney, K., & Peli, E. (2008). Community-based trial of a peripheral prism visual field expansion device for hemianopia. *Archives of Ophthalmology, 126*(5), 657–664.

Brahm, K. D., Wilgenburg, H. M., Kirby, J., Ingalla, S., Chang, C. Y., & Goodrich, G. L. (2009). Visual impairment and dysfunction in combat-injured service members with traumatic brain injury. *Optometry and Vision Science, 86*, 817–825.

Brooks, C. W., & Borish, I. M. (1996). *System for ophthalmic dispensing* (2nd ed.). Boston, MA: Butterworth-Heinemann.

Brown, S. (2003). Effect of whiplash injury on accommodation. *Clinical and Experimental Ophthalmology, 31*(5), 424–429.

Chan, R. P., & Trobe, J. D. (2002). Spasm of accommodation associated with closed head trauma. *Journal of Neuro-Ophthalmology, 22*(1), 15–17.

Cho, M. H., & Benjamin, W. J. (1998). Correction with multifocal spectacle lenses. In W. J. Benjamin (Ed.), *Borish's clinical refraction* (pp. 888–927). Philadelphia, PA: W.B. Saunders Company.

Ciuffreda, K. J. (2002). The scientific basis for and efficacy of optometric vision therapy in non-strabismic accommodative and vergence disorders. *Optometry, 73*, 735–762.

Ciuffreda, K. J., Han, Y., Kapoor, N., & Suchoff, I. B. (2001). Oculomotor consequences of acquired brain injury. In I. B. Suchoff, K. J. Ciuffreda, & N. Kapoor (Eds.), *Visual & vestibular consequences of acquired brain injury* (pp. 77–88). Optometric Extension Program: Santa Ana, CA.

Ciuffreda, K. J., Kapoor, N., Rutner, D., Suchoff, I. B., Han, M. E., & Craig, S. (2007). Occurrence of oculomotor dysfunctions in acquired brain injury: A retrospective analysis. *Optometry, 78*(4), 155–161.

Ciuffreda, K. J., & Ludlam, D. P. (2011). Egocentric localization: Normal and abnormal aspects. In P. S. Suter & L. H. Harvey (Eds.), *Vision rehabilitation: Multidisciplinary care of the patient following brain injury* (pp. 213–281). Boca Raton, FL: CRC Press.

Ciuffreda, K. J., Rutner, D., Kapoor, N., Suchoff, I. B., Craig, S., & Han, M. E. (2008). Vision therapy for oculomotor dysfunction in acquired brain injury: A retrospective analysis. *Optometry, 79*(1), 18–22.

Ciuffreda, K. J., & Tannen, B. (1995). *Eye movements for the clinician*. St. Louis, MO: Mosby.

Ciuffreda, K. J., Yadav, N. K., & Ludlam, D. P. (2017). Binasal occlusion (BNO), visual motion sensitivity (VMS), and the visually-evoked potential (VEP) in mild traumatic brain injury and traumatic brain injury (mTBI/TBI). *Brain Sciences, 7*(8), 98.

Ciuffreda, K. J., Yadav, N. K., & Thiagarajan, P. (2016). Traumatic brain injury: Visual consequences, diagnosis, and treatment. *Advances in Ophthalmology and Optometry, 1*, 307–333.

Cline, D., Hofstetter, H. W., & Griffin, J. R. (1989). *Dictionary of visual science*. Radnor, PA: Chilton Trade Book Publishing.

Cohen, A. H., & Rein, L. D. (1992). The effect of head trauma on the visual system: The doctor of optometry as a member of the rehabilitation team. *Journal of American Optometry Association, 32*(8), 530–536.

Craig, S. B., Kapoor, N., Ciuffreda, K. J., Suchoff, I. B., Han, M. E., & Rutner, D. (2008). Profile of selected aspects of visually-symptomatic individuals with acquired brain injury: A retrospective study. *Journal of Behavioral Optometry, 19*(1), 7–10.

Digre, K. B., & Brennan, K. C. (2011). Shedding light on photophobia. *Journal of Neuro-Ophthalmology, 32*, 68–81.

Du, T., Ciuffreda, K. J., & Kapoor, N. (2005). Elevated dark adaptation threshold in traumatic brain injury. *Brain Injury, 19*(13), 1125–1138.

Falk, N. S., & Aksionoff, E. B. (1992). The primary care optometric evaluation of the traumatic brain injury patient. *Journal of American Optometry Association, 63*(8), 547–553.

Fawcett, S. L., Stager, D. R., & Felius, J. (2004). Factors influencing stereoacuity outcomes in adults with acquired strabismus. *American Journal of Ophthalmology, 138*, 931–935.

Friedman, N. E. (1997). The pupil. In K. Zadnik (Ed.), *The ocular examination: Measurement and findings* (pp. 33–50). Philadelphia, PA: W.B. Saunders Company.

Gallaway, M., Scheiman, M., & Mitchell, G. L. (2016). Vision therapy for post-concussion vision disorders. *Optometry and Vision Science, 93*, 68–73.

Gerner, E. W. (1993). Visual sequelae of closed head trauma. In S. Mandel, R. T. Sataloff, & S. R. Schapiro (Eds.), *Minor head trauma: Assessment, management, and rehabilitation* (pp. 235–244). New York, NY: Springer-Verlag.

Gianutsos, R., Ramsey, G., & Perlin, R. R. (1988). Rehabilitative optometric services for survivors of acquired brain injury. *Archives of Physical Medicine & Rehabilitation, 69*, 573–578.

Gilhotra, J. S., Mitchell, P., Healey, P. R., Cumming, R. G., & Currie, J. (2002). Homonymous visual field defects and stroke in an older population. *Stroke, 33*, 2417–2420.

Gizzi, M., Khattar, V., & Eckert, A. (1997). A quantitative study of postural shifts induced by yoked prism. *Journal of Optometry and Vision Development, 28*, 200–203.

Godts, D., Tassignon, M., & Gobin, L. (2004). Binocular vision impairment after refractive surgery. *Journal of Cataract & Refractive Surgery, 30*(1), 101–109.

Good, P. A., Taylor, R. H., & Mortimer, M. J. (1991). The use of tinted glasses in childhood migraine. *Headache, 31*, 533–536.

Goodale, M. A. (2010). The functional organization of the central visual pathways. In G. N. Dutton & M. Bax (Eds.), *Visual impairment in children due to damage to the brain* (pp. 1–19). London: Mac Keith Press.

Gottlieb, D. D., Freeman, P., & Williams, M. (1992). Clinical research and statistical analysis of a visual field awareness system. *Journal of the American Optometric Association, 63*, 581–588.

Gottlieb, R. L., & Wallace, L. B. (2010). Syntonic phototherapy. *Photomedicine and Laser Surgery, 28*(4), 449–452.

Groffman, S. (2011). Acquired brain injury and visual information processing deficits. In P. S. Suter & L. H. Harvey (Eds.), *Vision rehabilitation: Multidisciplinary care of the patient following brain injury* (pp. 397–426). Boca Raton, FL: CRC Press.

Han, M. E. (2007). Chapter 8: The role of the neuro-rehabilitation optometrist. In J. Elbaum & D. M. Benson (Eds.), *Acquired brain injury: An integrative neuro-rehabilitation approach* (pp. 146–175). New York, NY: Springer.

Han, M. E., Craig, S. B., Rutner, D., Kapoor, N., Ciuffreda, K. J., & Suchoff, I. B. (2008). Medications prescribed to brain injury patients: A retrospective analysis. *Optometry, 79*, 252–258.

Harris, P. A. (2011). The use of lenses to improve quality of life following brain injury. In P. S. Suter & L. H. Harvey (Eds.), *Vision rehabilitation: Multidisciplinary care of the patient following brain injury* (pp. 213–281). Boca Raton, FL: CRC Press.

Heikkila, H. V., & Wenngren, B.-I. (1998). Cervicocephalic kinesthetic sensibility, active range of cervical motion, and oculomotor function in patients with whiplash injury. *Archives of Physical Medicine and Rehabilitation, 79*, 1089–1094.

Hellerstein, L., & Freed, S. (1994). Rehabilitative optometric management of a traumatic brain injury patient. *Journal of Behavioral Optometry, 5*(6), 143–148.

Helvie, R. (2011). Neural substrates of vision. In P. S. Suter & L. H. Harvey (Eds.), *Vision rehabilitation: Multidisciplinary care of the patient following brain injury* (pp. 45–76). Boca Raton, FL: CRC Press.

Huang, J., Zong, X., Wilkins, A., Jenkins, B., Bozoki, A., & Cao, Y. (2011). fMRI evidence that precision ophthalmic tints reduce cortical hyperactivation in migraine. *Cephalalgia, 31*, 925–936.

Iskander, D., Cohen, A. H., & Kapoor, N. (2010). Optometric management of a patient with parietal lobe injury. *Journal of Behavioral Optometry, 6*, 143–149.

Jackowski, M. M. (2001). Altered visual adaptation in patients with traumatic brain injury. In I. B. Suchoff, K. J. Ciuffreda, & N. Kapoor (Eds.), *Visual & vestibular consequences of acquired brain injury* (pp. 145–173). Santa Ana, CA: Optometric Extension Program.

Jackowski, M. M., Sturra, J. F., Taub, H. A., & Turke, M. A. (1996). Photophobia in patients with traumatic brain injury: Uses of light-filtering lenses to enhance contrast sensitivity and reading rate. *NeuroRehabilitation, 6*, 193–201.

Julkunen, L., Tenovuo, O., Jääskeläinen, S., & Hämäläinen, H. (2003). Rehabilitation of chronic post-stroke visual field defect with computer-assisted training. *Restorative Neurology and Neuroscience, 21*, 19–28.

Kapoor, N., & Ciuffreda, K. J. (2005). Vision problems. In J. M. Silver, T. W. McAllister, & S. C. Yudofsky (Eds.), *Textbook of traumatic brain injury* (1st ed., pp. 405–417). Washington, DC: American Psychiatric Publishing.

Kapoor, N., Ciuffreda, K. J., & Suchoff, I. B. (2001). Egocentric localization in patients with visual neglect. In I. B. Suchoff, K. J. Ciuffreda, & N. Kapoor (Eds.), *Visual & vestibular consequences of acquired brain injury* (pp. 131–144). Santa Ana, CA: Optometric Extension Program.

Kasten, E., Bunzenthal, U., & Sabel, B. A. (2006). Visual field recovery after vision restoration therapy (VRT) is independent of eye movements: An eye tracker study. *Behavioural Brain Research, 175*, 18–26.

Kaufman, P. L. (1992). Accommodation and presbyopia: Neuromuscular and biophysical aspects. In W. M. Hart (Ed.), *Adler's physiology of the eye: Clinical application* (9th ed., pp. 391–411). St. Louis, MO: Mosby Year Book.

Kerkhoff, G., Münßinger, U., & Meier, E. K. (1994). Neurovisual rehabilitation in cerebral blindness. *Archives of Neurology, 51*, 474–481.

Laukkanen, H., Scheiman, M., & Hayes, J. R. (2017). Brain injury vision symptom survey (BIVSS) questionnaire. *Optometry and Vision Science, 94*, 43–50.

Leslie, S. (2001). Accommodation in acquired brain injury. In I. B. Suchoff, K. J. Ciuffreda, & N. Kapoor (Eds.), *Visual & vestibular consequences of acquired brain injury* (pp. 56–76). Santa Ana, CA: Optometric Extension Program.

Margolis, N. W. (2011). Evaluation and treatment of visual field loss and visual-spatial neglect. In P. S. Suter & L. H. Harvey (Eds.), *Vision rehabilitation: Multidisciplinary care of the patient following brain injury* (pp. 77–151). Boca Raton, FL: CRC Press.

Margolis, N. W., & Suter, P. S. (2006). Visual field defects and unilateral spatial inattention: Diagnosis and treatment. *Journal of Behavioral Optometry, 17*(2), 31–37.

Marshall, R. S., Ferrera, J. J., Barnes, A., Zhang, X., O'Brien, K. A., Chmayssani, M., … Lazar, R. M. (2008). Brain activity associated with stimulation therapy of the visual borderzone in hemianopic stroke patients. *Neurorehabilitation and Neural Repair, 22*, 136–144.

Marshall, R. S., Chmayssani, M., O'Brien, K. A., Handy, C., & Greenstein, V. C. (2010). Visual field expansion after visual restoration therapy. *Clinical Rehabilitation, 24*(11), 1027–1035.

Massucci, M. E. (2009). Prism adaptation in the rehabilitation of patients with unilateral spatial inattention. *Journal of Behavioral Optometry, 20*, 101–105.

Master, C. L., Scheiman, M., Michael Gallaway, M., Goodman, A., Robinson, R. L., Master, S. R., & Grady, M. F. (2016). Vision diagnoses are common after concussion in adolescents. *Clinical Pediatrics, 55*(3), 260–267.

Mödden, C., Behrens, M., Damke, I., Eilers, N., Kastrup, A., & Hildebrandt, H. (2012). A randomized controlled trial comparing 2 interventions for visual field loss with standard occupational therapy during inpatient stroke rehabilitation. *Neurorehabilitation and Neural Repair, 26*(5), 463–469.

Mueller, I., Mast, H., & Sabel, B. A. (2007). Recovery of visual field defects: A large clinical observational study using vision restoration therapy. *Restorative Neurology and Neuroscience, 25*, 563–572.

Mueller, I., Poggel, D. A., Kenkel, S., Kasten, E., & Sabel, B. A. (2003). Vision restoration therapy after brain damage: Subjective improvements of activities of daily life and their relationship of visual field enlargements. *Visual Impairment Research, 5*(3), 157–178.

Nelles, G., Esser, J., Eckstein, A., Tiede, A., Gerhard, H., & Diener, H. C. (2001). Compensatory visual field training for patients with hemianopia after stroke. *Neuroscience Letters, 306*, 189–192.

Noseda, R., Constandil, L., Bourgeais, L., Chalus, M., & Villanueva, L. (2010). Changes of meningeal excitability mediated by corticotrigeminal networks: A link for the endogenous modulation of migraine pain. *The Journal of Neuroscience, 30*(43), 14420–14429.

Padula, W. V. (1988a). Chapter VI: The neuro-optometric rehabilitation examination. In W. V. Padula (Ed.), *Neuro-optometric rehabilitation* (3rd ed., pp. 78–87). Santa Ana, CA: Optometric Extension Program.

Padula, W. V. (1988b). Chapter XIV: Post-trauma vision syndrome caused by head injury. In W. V. Padula (Ed.), *Neuro-optometric rehabilitation* (3rd ed., pp. 179–193). Santa Ana, CA: Optometric Extension Program.

Padula, W. V. (2012). Visual midline shift syndrome. In W. V. Padula, R. Munitz, & W. M. Magrun (Eds.), *Neuro-visual processing rehabilitation: An interdisciplinary approach* (pp. 78–89). Santa Ana, CA: Optometric Extension Program.

Poggel, D. A., Kasten, E., & Sabel, B. A. (2004). Attentional cueing improves vision restoration therapy in patients with visual field defects. *Neurology, 63*, 2069–2076.

Reinhard, J., Schreiber, A., Schiefer, U., Kasten, E., Sabel, B. A., Kenkel, S., … Trauzettel-Klosinski, S. (2005). Does visual restitution training change absolute homonymous visual field defects? A fundus controlled study. *British Journal of Ophthalmology, 89*, 30–35.

Rode, G., Rossetti, Y., & Boisson, D. (2001). Prism adaptation improves representational neglect. *Neuropsychologia, 39*, 1250–1254.

Romano, J. G., Schulz, P., Kenkel, S., & Todd, D. P. (2008). Visual field changes after a rehabilitation intervention: Vision restoration therapy. *Journal of the Neurological Sciences, 273*, 70–74.

Rosen, S. A., Cohen, A. H., & Trebling, S. (2001). The integration of visual and vestibular systems in balance disorder—A clinical perspective. In I. B. Suchoff, K. J. Ciuffreda, & N. Kapoor (Eds.), *Visual and vestibular consequences of acquired brain injury* (pp. 174–200). Santa Ana, CA: Optometric Extension Program.

Rosenfield, M. (1997). Accommodation. In K. Zadnik (Ed.), *The ocular examination: Measurements and findings* (pp. 87–122). Philadelphia, PA: W.B. Saunders.

Rosetti, Y., Rode, G., Pisella, L., Farne, A., Li, L., Boisson, D., & Perenin, M. T. (1998). Prism adaptation to a rightward optical deviation rehabilitates left hemispatial neglect. *Nature, 395*, 166–169.

Sabates, N. R., Gonce, M. A., & Farris, B. K. (1991). Neuro-ophthalmological findings in closed head trauma. *Journal of Clinical Neuro-ophthalmology, 11*(4), 273–277.

Sabel, B. A., & Kasten, E. (2000). Restoration of vision by training of residual functions. *Current Opinion in Ophthalmology, 11*, 430–436.

Sabel, B. A., Kenkel, S., & Kasten, E. (2004). Vision restoration therapy (VRT) efficacy as assessed by comparative perimetric analysis and subjective questionnaires. *Restorative Neurology and Neuroscience, 22*, 399–420.

Sanet, R. B., & Press, L. J. (2011). Spatial vision. In P. S. Suter & L. H. Harvey (Eds.), *Vision rehabilitation: Multidisciplinary care of the patient following brain injury* (pp. 77–151). Boca Raton, CA: CRC Press.

Scheiman, M., & Wick, B. (2014). *Clinical management of binocular vision* (4th ed.). Philadelphia, PA: Lipponcott Williams and Wilkins.

Smith, N. A. (1999). Lighting for occupational optometry. In *HHSC handbook No. 23*. Leeds: H and H Scientific Consultants Ltd..

Starr, C., Evers, C. A., & Starr, L. (2011). Chapter 30: Sensory perception. In C. Starr, C. A. Evers, & L. Starr (Eds.), *Biology concepts and applications 8e* (pp. 489–502). Belmont, MA: Brooks/Cole Cengage Learning.

Stern, C. D. (2011). Photophobia, light, and color in acquired brain injury. In P. S. Suter & L. H. Harvey (Eds.), *Vision rehabilitation: Multidisciplinary care of the patient following brain injury* (pp. 213–281). Boca Raton, FL: CRC Press.

Suchoff, I. B., & Ciuffreda, K. J. (2004). A primer for the optometric management of unilateral spatial inattention. *Optometry, 75*, 305–317.

Suchoff, I. B., Kapoor, N., Ciuffreda, K. J., Rutner, D., Han, M. E., & Craig, S. (2008). The frequency of occurrence, types, and characteristics of visual field defects in acquired brain injury: A retrospective analysis. *Optometry, 79*, 59–65.

Suchoff, I. B., Kapoor, N., Waxman, R., & Ference, W. (1999). The occurrence of ocular and visual dysfunctions in an acquired brain-injured patient sample. *Journal of the American Optometric Association, 70*(5), 301–309.

Suter, P. S. (1995). Rehabilitation and management of visual dysfunction following traumatic brain injury. In M. J. Ashley & D. K. Krych (Eds.), *Traumatic brain injury rehabilitation* (pp. 187–219). Boca Roton, FL: CRC Press.

Suter, P. S. (2007). Peripheral visual field loss & visual neglect: Diagnosis & treatment. *Journal of Behavioral Optometry, 18*(3), 78–83.

Tannen, B., Darner, R., Ciuffreda, K., Shelley-Tremblay, J., & Rogers, J. (2015). Vision and reading deficits in post-concussion patients: A retrospective analysis. *Vision Development & Rehabilitation, 1*(3), 206–213.

Thompson, H. S. (1992). The pupil. In W. M. Hart (Ed.), *Adler's physiology of the eye: Clinical application* (9th ed., pp. 391–411). Mosby Year Book: St. Louis, MO.

Truong, J. Q., Ciuffreda, K. J., Han, M. E., & Suchoff, I. B. (2014). Photosensitivity in mild traumatic brain injury (mTBI): A retrospective analysis. *Brain Injury, 28*(10), 1283–1287.

Vogel, M. S. (1992). An overview of head trauma for the primary care practitioner: Part II—Ocular damage associated with head trauma. *Journal of American Optometry Association, 63*(8), 542–546.

Warren, M. (1993). Hierarchical model for evaluation and treatment of visual perceptual dysfunction in adult acquired brain injury, Part I. *The American Journal of Occupational Therapy, 47*(1), 42–54.

Weiss, L. M. (2002). Visual-vestibular interaction in the acquired brain injured patient. *Journal of Optometric Vision Development, 33*, 33–41.

Zhang, X., Kedar, S., Lynn, M. J., Newman, N. J., & Biousse, V. (2006). Natural history of homonymous hemianopia. *Neurology, 66*, 901–905.

Zihl, J. (2000). *Rehabilitation of visual disorders after brain injury* (pp. 11–90). East Sussex: Psychology Press Ltd.

Zihl, J., & Kerkhoff, G. (1990). Foveal photopic and scotopic adaptation in patients with brain damage. *Clinical Vision Sciences, 5*(2), 185–195.

Chapter 7
The Role of Occupational Therapy in Neurorehabilitation

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What Is Occupational Therapy?

Occupational therapy (OT) can be defined as "the therapeutic use of everyday life activities (occupations) with individuals or groups for the purpose of enhancing or enabling participation in roles, habits, and routines in home, school, workplace, community, and other settings" (AOTA, 2014). The goal of OT is to maximize independence in all areas of occupation, including activities of daily living (ADLs), instrumental activities of daily living (IADLs), rest and sleep, education, work, play, leisure, and social participation. As per the World Federation of Occupational Therapists (2012), "In occupational therapy, occupations refer to the everyday activities that people do as individuals, in families and with communities to occupy time and bring meaning and purpose to life. Occupations include things people need to, want to and are expected to do." Occupational therapists look at the dynamic and interactive relationship between clients, environmental factors, and occupations in order to assess and provide interventions and strategies for improved independence. Occupational therapists assist with engagement and participation by addressing the underlying factors, performance skills, performance patterns, and occupations directly to meet the goals of clients within their natural context.

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OT Following Acquired Brain Injury (ABI)

Occupational therapists working with clients who have sustained acquired brain injuries provide treatment utilizing remedial and compensatory approaches. Remedial or restorative care involves addressing the underlying skills impacting independence to facilitate active progress and maximize functional potential. When restorative change isn't feasible or functional, occupational therapists utilize their clinical knowledge to make recommendations and provide compensatory strategies. Habilitative care involves adaptive strategies, techniques, or modifications to allow the individual to complete tasks utilizing their current skill set. To determine the most optimal approach, an activity analysis is completed. This involves breaking down each task into its component parts to determine impediments to performance. Activity analysis is essential in the rehabilitation process to assess and establish meaningful and achievable objectives for the client. Occupational therapists working with this cohort utilize a holistic approach to address physical, cognitive, visual, psychosocial, and behavioral impairments which can be impacted following a neurological event.

Occupational therapists implement comprehensive and extensive assessment and intervention. This becomes increasingly important in this practice arena, due to the abstract nature of providing clinical services for individuals with brain injuries. Individuals who experience similar acquired brain injuries, impacting the same localized region of the brain, can exhibit completely different presentations. One individual may present with a dense left hemiparetic limb and exhibit severe left neglect, whereas another individual can exhibit no perceptual challenges, however can exhibit mild fine motor coordination impairments. In contrast, two individuals who receive the same hip replacement or experience the same Colles fracture will typically exhibit comparable symptomatology and, in turn, receive a more standardized protocol for treatment. In essence, this is what makes working with a client with an acquired brain injury unique and complex. Typical presentations guide treatment; however, each client must be evaluated and treated individually based on demonstrated needs.

Evaluation

In order to provide client-centered intervention strategies, an occupational therapist's role begins with an evaluation. This involves development of an occupational profile and analysis of occupational performance. A thorough clinical interview is needed which includes open-ended questions about past medical history, history of present illness, and a holistic inquiry into current deficits, prior and current level of function, and environmental factors (AOTA, 2014). An occupational profile consists of the client's autobiographical information, subjective description of problems interfering with occupational engagement, and concerns which impact roles and participation based on values and interests (AOTA, 2014). This is collected through

the client directly and family as appropriate. It is helpful to have a family member or caregiver present during the evaluation, with the client's consent, to collaborate or clarify the information provided by the client.

An analysis of occupational performance occurs to supplement personal goals with information from objective measures and assessment tools. Relevant tools for evaluation are selected and administered to create goals and delineate applicable intervention approaches. It is the job of the occupational therapist to determine alignment between self-report of functioning and clinically determined needs in order to develop insight, safely perform daily tasks, and implement appropriate interventions to maximize functional potential. The comparison between self-report and objective findings will allow occupational therapists to generate a relevant treatment plan, including short-term goals, long-term goals, and clinically selected modalities and intervention strategies. A treatment plan is referenced to guide treatment and should be continually reanalyzed and adapted as needed to meet the dynamically changing needs and skill set of clients.

The assessment process is guided by activity demands, personal influences, and contextual factors and based directly on the areas of occupation, including ADLs, IADLs, rest and sleep, education, work, play, and social participation (AOTA, 2014). These areas will be further explored in the coming sections.

Areas of Occupation

Activities of Daily Living (ADLs) and Instrumental Activities of Daily Living (IADLs)

According to AOTA (2014), activities of daily living (ADLs) include basic activities of daily living (BADLs) and instrumental activities of daily living (IADLs). These activities are "fundamental to living in a social world; they enable basic survival and well-being" (Christiansen & Hammecker, 2001). Activities of daily living are the most fundamental area of occupation that individuals partake in to care for themselves. ADLs encompass bathing, toileting, dressing, swallowing/eating, feeding, functional mobility, personal device care, personal hygiene and grooming, and sexual activity (AOTA, 2014). Instrumental activities of daily living (IADLs) are intertwined with ADLs, notably more multifaceted in nature. By definition, IADLs are "activities to support daily life within the home and community that often require more complex interactions than those of ADLs" (AOTA, 2014). IADLs include of care of others, care of pets, child rearing, communication management, driving, community mobility, financial management, health management and maintenance, home establishment and management, meal preparation and cleanup, religious and spiritual activities and expression, safety and emergency maintenance, and shopping. When there is a change in functional performance or efficiency/efficacy of performance, or independence is not reduced, client's skills are addressed to facilitate improved independence.

Occupational therapists utilize client's reports and clinical observation to determine level of assistance, safety, and functional performance with daily tasks. To assess performance in ADLs and IADLs following a neurological injury or event, occupational therapists target physical, cognitive, and visual skills associated with task participation, allowing determination of level of independence. To determine assistance level in an outpatient setting, ADL and IADL skills can be directly addressed through task practice. Performance can be simulated to promote carryover to the most natural context in the home, or facilitate generalization of skills.

When addressing barriers to self-care, physical impairments are most notably identified and targeted in traditional outpatient practice. In the case of a neurological event, a comprehensive assessment of physical, visual, psychosocial, emotional, and cognitive skills occurs to determine the impact of these skills on daily functioning (Mercier, Audet, Herbert, Rochette, & Dubois, 2001). Occupational therapists evaluate skill requirements, including but not limited to physical skills of range of motion, muscle strength and endurance, sensation, balance, and coordination; visual skills of perception, scanning, acuity, and extraocular eye movements; and cognition including attention, sequencing, processing speed, and executive functions. In relation to clients with neurological injuries, all facets of performance must be examined to successfully pinpoint which area is impacting occupational performance. Often, skills are intertwined and must be addressed in isolated performance and then gradually integrated and upgraded to encompass a variety of skills in real-life engagement.

Occupational therapists are skilled in assessing and analyzing task performance to determine areas of needs. When initiating an assessment of ADL and IADL skills, performance should occur without assistance or support in order to properly and objectively determine a client's baseline. Clinicians are trained in various forms of cueing to provide physical, verbal, visual, and tactile support to perform task components and whole task completion. This could manifest as providing a visual anchor to turn head to neglected side to locate items needed for toileting, providing one-step commands to recall sequence during oral hygiene activities, or providing support to a body part to reach overhead for grooming hair.

Occupational therapists work collaboratively with clients and caregivers to determine performance factors and environmental constraints. Performance can then be replicated to simulate home activities in order to maximize success. Interventional strategies include improving skills through therapeutic exercise and therapeutic activities, providing provisions and adaptive equipment to aid in performance, and determining strategies to maximize independence with the client's current skill set. An example of remediating an underlying performance skill includes challenging dynamic sitting balance to facilitate improved performance during lower body dressing or bathing. On the other hand, providing compensatory strategies or adaptations might be warranted in place of remediating the skill. For example, occupational therapists may recommend use of a dressing stick to account for decreased sitting balance to complete dressing or utilize a caregiver's support to set up all required items if visuospatial deficits are present (such as rotating clothes with proper directionality). Additionally, an occupational therapist could provide education on hemi-techniques to complete dressing effectively with the use of one upper extremity.

Dysfunction in ability to maintain a home and complete meal preparation tasks can result from physical, cognitive, and perceptual deficits. The occupational therapist will assess skills and identify underlying barriers to develop a client-centered treatment plan in order to increase occupational performance and independence. Restorative skills are addressed to maximize independence in the least restrictive capacity, followed by education of any modifications or strategies to be implemented. Adaptations can include environmental modification to increase accessibility within the person's home. Adaptive equipment can include use of a rocker knife, one-handed cutting board for meal preparation, or dycem as a nonskid surface while cleaning dishes. In order to increase success, various strategies for energy conservation and work simplification can be implemented. Rest breaks, seated activity performance, or reorganization with placement of items in more accessible locations (including lowering of items on shelves) can be used to compensate for reduced endurance and activity tolerance. Through task evaluation and observation of performance skills, therapists can further analyze if memory deficits, initiation/termination, organization, sequencing, or cognitive flexibility difficulties might interfere with safety when cooking or following a recipe. Once skills are properly identified as hindrances to performance, deficit areas can be targeted. Additionally, strengths are important and relied upon to assist and enhance independence. For example, if awareness of deficit is noted as a strength during evaluation, occupational therapists can use this to improve the client's use of strategies to rotate head to a side where a visual field cut is present.

When targeting IADLs, evaluation of skills extends to community and work reintegration. Functioning in the community is a component of being an active, engaged member in society. Community skills must be assessed to determine the impact of a neurological injury on an individual's functioning, as well as determining if barriers are rooted in physical, cognitive, visual, psychological, socioemotional, or behavioral difficulties. Essential components of community access include functional mobility, safety, and appropriate and effective interactions with others. Evaluation for environmental awareness and visual scanning deficits will occur to determine safety moving around in public environments which are well-traveled, open, and can be unpredictable. Evaluation of social interactions in public locations such as grocery stores and restaurants will also be assessed, in addition to topographical orientation skills and ability to be flexible in ever-changing environments. Physical negotiation and appropriateness of durable medical equipment (DME) and assistive devices used can be explored. *See Chap. 8 for further information on functional mobility*. Appropriate referrals can also be made to maximize community reintegration, including to a wheelchair clinic or vendor to evaluate for power wheelchairs or one-arm-drive wheelchairs to enhance access in mobility-related ADLs.

With completion of a full evaluation and occupational profile, relevant IADLs can be determined. Money management skills are another IADL that can be addressed. Assessment occurs in a progression from simple to complex tasks to allow the client to meet the gradual task demands. An individual's ability to visually discriminate and identify monetary amounts, as well as summate or calculate monetary differences when purchasing an item, are fundamental and foundational skills for completing a functional money exchange in the community. Managing finances,

paying bills, and budgeting are all areas to be assessed for participation in complex money management. Mental operations, planning, and attention to detail are some of the skills as potential underlying factors to be assessed if difficulty in task performance is determined. It is important to assess and understand both the task-based difficulties and the performance difficulties to address the occupation and promote functional change.

To maximize safe functioning in ADLs and IADLs, home and driving evaluations are commonplace in practice. Occupational therapists can make recommendations for adaptive equipment and DME to be able to enter and function in the home with maximal success. Utilizing observation and simulation of tasks, occupational therapists can assess performance of ADLs in the home. Recommendations can then be made based on assessment and findings including removing clutter, placing items in accessible locations for physical and visual deficits, or placing items in a consistent location for cognitive deficits. Physical adaptations including adding ramps, grab bars, and tub bench/shower chair may be recommended to increase safety and independence in the home. In regard to driving evaluations, occupational therapists will assess and identify predriving skills in the clinic. If deemed medically appropriate, skills will be evaluated. Evaluation will occur to determine appropriateness of return to driving, including but not limited to assessing motor processing speed, divided attention skills, visual field testing, and visual perception. Following assessment, skills will be addressed in treatment if driving is determined to be an appropriate goal. By evaluating essential performance skills for driving, occupational therapists can make clinical recommendations as part of the interdisciplinary team to determine readiness. A referral can be made for an on-road assessment to determine functional performance and safety with a driving instructor or specialist.

Rest and Sleep

Rest and sleep are integral to maintaining a healthy and active lifestyle and support participation in other meaningful daily activities. Rest can include identifying a need to relax, reducing involvement in taxing activities to facilitate relaxation and resting, and engaging in activities to restore energy (AOTA, 2014). The occupation of sleep includes both sleep preparation and sleep participation (AOTA, 2014). Sleep preparation encompasses engaging in bedtime routines, determining desired sleep times and patterns, and preparing the environment to support sleep, while sleep participation encompasses sustaining sleep without disruptions (AOTA, 2014). The role of occupational therapy in this area can include assisting clients to develop healthy habits and bedtime routines, particularly with clients who have difficulty successfully planning, prioritizing, and problem-solving. To facilitate comfort in preparation for rest and sleep, occupational therapists also work with clients to manage pain and educate clients on comfortable positioning for painful hemiparetic upper extremities.

Education

Participation in education can include activities such as formal education participation, informal personal education and interest exploration, and informal personal education participation such as classes or training (AOTA, 2014). Participation in education, both formally and informally, requires a variety of skills including physical, cognitive, and visual. Following an acquired brain injury, clients can be at various stages of returning to education including at the elementary, intermediate, secondary, or collegiate level. Occupational therapists can facilitate remediation of skills including attention, executive functions, problem-solving, abstract reasoning, and processing speed to facilitate readiness to participate in education. This can be done by simulating educational activities, such as listening to lectures and taking notes in preparation for answering questions. This can also include challenging clients to complete novel problem-solving through an assignment given by the clinician.

In addition to remediation of skills, occupational therapists can educate clients and caregivers on utilization of strategies to compensate for cognitive deficits. For example, utilizing planners to improve the ability to plan and prioritize assignments effectively. OTs can also educate clients regarding facilitating supportive environments for completing assignments, such as quiet and distraction-free work spaces for clients with difficulty sustaining attention. In conjunction with the interdisciplinary team, occupational therapists utilize clinical observation and assessment to determine any necessary accommodations that may benefit clients in the educational environment.

Work

The occupation of work can include exploring employment interests and pursuits, employment seeking and acquisition, job performance, retirement preparation/adjustment, volunteer exploration, and volunteer participation (AOTA, 2014). In order to comprehensively evaluate return-to-work skills for clients who have an acquired brain injury, it is recommended that occupational therapists evaluate the client's interests, physical and cognitive capacities, and work behaviors (Davis, Wheeler, & Acord-Vira, 2017). Occupational therapists utilize their expertise in task analysis from the client or family report of job responsibilities to determine the necessary client factors to successfully perform the client's job of choice. It is important to assess a client's physical capacity to perform their specific job (Davis et al., 2017). For example, if a client is required to complete repetitive lifting as part of their job, it is important to assess that they have the necessary upper extremity muscle strength, coordination, endurance, and balance to complete this task. In addition to assessing physical capacity, occupational therapists evaluate cognitive performance in relation to returning to work. This includes formal and informal evaluation of attention, memory, judgment, insight, problem-solving, initiation, planning, cognitive flexibility, self-monitoring, or other pertinent cognitive skills. Evaluation of work behaviors,

such as the ability to arrive on time, complete work in a timely manner, follow directions, and be flexible, is an important component of evaluating a client's readiness to return to work (Davis et al., 2017). It is imperative for the client and interdisciplinary team to determine if it is appropriate for a client to return to his/her previous job with or without assistance or accommodations, or to explore an alternative and more appropriate job or role.

In preparation for the client returning to previous competitive employment following an acquired brain injury, occupational therapists may work on remediating the aforementioned skills and behaviors. Occupational therapists will attempt to simulate activities that would be required in the client's job while working to remediate the necessary skills. For example, if a client's job requires secretarial or clerical work, the clinician can plan a session requiring a client to file and organize paperwork, take simulated phone messages, or create employee schedules. The process of remediating cognitive and physical skills, and work behaviors, is completely individualized to the client's current personal goals as well as strengths, limitations, and job requirements. Occupational therapists will also work with clients to enact compensatory strategies or environmental modifications to facilitate success in their job.

If the occupational therapist, interdisciplinary team, client, and/or client's workplace determine that the client cannot return to previous employment, exploration of other appropriate roles becomes the focus of therapy. Appropriate alternative recommendations may include participation in a structured day program, volunteering, or vocational retraining. The occupational therapist's awareness and understanding of community resources become vital. The occupational therapist will need to educate the client and caregivers about these recommendations and the processes for participation in these community resources.

Case Study #1

AB is a 49-year-old female who experienced symptomatology of headaches, dizziness, and vomiting. She was transferred to a nearby hospital where she was diagnosed with a left cerebellar hemorrhage and underwent a craniotomy to remove a cerebellar hematoma. She was hospitalized for approximately 2 weeks, followed by being transferred to a rehabilitation hospital for 1½ weeks where she received occupational, physical, and speech therapies. AB was discharged home and received home care services prior to being referred for outpatient therapy services, specializing in neurorehabilitation. AB was evaluated and determined a candidate for outpatient occupational, physical, and speech therapies.

At initial evaluation, AB demonstrated impairments with muscle and grip strength, fine and gross motor coordination, left-sided attention, safety awareness, attention, and executive functions. Through the use of formal/informal assessment of deficits and a clinical interview to determine an occupational profile, the goals for therapy included improving safety awareness, grip strength, fine motor coordination, selective attention, educating client on strategies for improved left-sided attention, and improving independence in desired daily occupations. Over the course of

therapy, AB made significant improvements in all set short-term goals, resulting in an upgrade of objectives and incorporation of additional goals including executive functions and divided attention. As the client became more functionally independent and safe during basic and complex ADLs, the discussion began about client's goals for community reintegration. AB desired to return to her prior job in hospitality, and this goal became realistic as her therapy continued. Occupational therapy began to shift focus to incorporate work reintegration skills.

For AB's job in hospitality, the necessary job duties and responsibilities included setting up rooms for presentations and luncheons, setting up refreshments, greeting guests, and utilizing public transportation for traveling to work. Work simulation was integrated into sessions, including requiring the client to gather desired items on a coffee cart and set up refreshments. The occupational therapists also challenged the necessary underlying skills to complete community and work tasks, such as planning, organization, problem-solving, attention to detail, and divided attention. In her sessions, AB and the occupational therapist worked on accessing the public transportation website and determining the necessary subway route to get to and from work, further incorporating mental flexibility, time management, and topographical orientation skills. The client determined that her friend would initially commute with her to determine any concerns. While AB still demonstrated reduced complex problem-solving and attention to detail in novel tasks, she was able to successfully complete work simulation tasks and utilize strategies to compensate for her remaining deficits. It is typically recommended that if clients return to work, they do so on a part-time basis with any necessary accommodations, to enable OT and the client to continue addressing functional concerns during therapy and allow for gradual reintegration to promote success. Interdisciplinary communication regarding appropriateness of return to work, as well as the client's obtaining clearance from their medical team such as their doctor, is also typically recommended. AB was able to return to work, gradually reintegrating to a full-time schedule without any reported difficulty.

Leisure

Leisure can be defined as participation in activities that are not an obligation and occur during discretionary time (Parham & Fazio, 1997). An occupational therapist's role in this area can include both leisure exploration and leisure participation (AOTA, 2014). Leisure exploration includes identifying interests, skills, and leisure opportunities, while leisure participation includes planning and participating in identified desired leisure activities and balancing these activities with other obligatory areas of occupation (AOTA, 2014). Occupational therapists utilize multiple methods to assist clients in identifying desired leisure pursuits, including informal questionnaires and discussions, or utilizing role checklists and interest checklists. Occupational therapists can assist clients to identify barriers to participating in leisure activities at home or in the community and problem-solve compensatory strategies to support leisure participation. This can include one-handed strategies or adaptive equipment to participate in

meaningful hobbies. Leisure interests can be included as part of therapy sessions to address other skills, for example, utilizing a client's interest in gardening as a means of addressing deficits with sequencing or fine motor skills.

Social Participation

Social participation with others can occur in the community within a neighborhood, organization, or other group and also occurs with families, peers, and friends both in person and through the use of technology (AOTA, 2014). For many survivors of acquired brain injury, developing a network to participate with socially can be a vital part of recovery. This can include existing relationships with friends, family, and peers, with the addition of organizations and groups that provide social opportunities. Occupational therapists can refer clients to support groups and community programs external to the rehabilitation facility. Additionally, facilities can create their own support or wellness-based groups that convene on a regular basis to provide the opportunity for social participation for clients and caregivers. Occupational therapists can also assist clients in maintaining relationships with family and friends by improving effective communication, which may include improving skills in accessing and utilizing computers, apps, and technology.

Play

Play includes any activity that provides entertainment, amusement, or enjoyment including spontaneous or organized activities (Parham & Fazio, 1997). Engagement in play can be an incredibly important and meaningful area of occupation for pediatric clients who have sustained a brain injury. Play can include both exploration of play and participation in play (AOTA, 2014). Occupational therapists can work with clients utilizing play as a part of the therapy session. For example, if a client enjoys playing a specific board game, this game can be incorporated into therapy sessions to challenge various skills such as gross or fine motor coordination, visual perception, attention, or problem-solving, depending on the game being played and the client's goals. Additionally, occupational therapists can work with clients, parents, and families to develop adapted or compensatory strategies for clients to engage in meaningful play activities.

Upper Extremity Function

Following an acquired brain injury, one may experience upper extremity (UE) impairments, such as paresis, loss of fractionated movement, changes in somatosensation, and abnormal muscle tone (Lang, Bland, Bailey, Schaefer, & Birkenmeier, 2013).

A comprehensive physical evaluation will include quantitative measures utilizing standardized tests to assess active range of motion (AROM), passive range of motion (PROM), muscle strength, spasticity, gross motor control, fine motor control, and sensation to measure changes in UE functional capacity.

In addition to determining the presence or severity of each impairment utilizing these measures, it's important to assess the impact these changes have on functional performance. Upper extremity capacity and performance should be assessed separately because what a person is capable of is not necessarily what they are doing functionally during their day. This is completed through observation of the person performing a task or self-reported measures. When using self-reported measures such as the Stroke Impact Scale, Motor Activity Log, or pain scales in the assessment of UE function or pain, it is important to remember that this information may be biased due to cognitive deficits (Lang et al., 2013).

Brunnstrom stages of motor recovery also provides a guideline of anticipated recovery stages of the UE based on muscle spasticity and limb synergies of hemiplegic clients following stroke (Naghdi, Ansari, Mansouri, & Hasson, 2010) (*see Chaps. 4 and 8 for additional information)*. The combination of these assessments will guide clinical decision-making regarding the therapeutic approach to enable clients to return to functional, meaningful lives (Doucet, 2012).

Range of Motion (ROM): AROM/PROM

Prior to evaluating range of motion (ROM), the therapist will assess the client for self-reported pain and observe shoulder and scapular positioning. It is important to determine if abnormal postures, glenohumeral subluxation, or scapular asymmetries such as scapular winging or tipping are present. Scapular AROM/PROM are assessed next, as scapulohumeral rhythm is necessary for optimal function of the shoulder. "Scapulothoracic rhythm is important because it sets the most functional position of the Glenohumeral joint, prevents active insufficiency of the scapulohumeral muscles, and allows for sufficient ROM between the humeral head and the subacromial space" (Hardwick & Lang, 2011). When this relationship is altered, it may contribute to shoulder instability, shoulder impingement, and hemiplegic shoulder pain (HSP) which will affect the entire upper extremity (Levangie & Norkin, 2005).

The evaluation of UE ROM is completed through goniometry and observation of an individual completing various tasks. During goniometric measurement, both passive and active range of motion are assessed. Active range of motion will measure the range of movement that a client can actively move a joint without assistance. It will also inform the clinician of any synergies that are present. Passive range of motion will inform the clinician of any limitations either structurally, caused by pain, or due to spasticity. The clinician will also assess for abnormal end feel of each joint. If there are limitations in functional ROM, the clinician will further evaluate to locate the source of the deficit and make appropriate recommendations.

Pain

Pain is commonly reported by clients following acquired brain injury and may impact function, quality of life, and recovery. Pain may result from various etiologies including hemiplegic shoulder pain, shoulder hand syndrome, spasticity-related pain, musculoskeletal pain, and central poststroke pain (Harrison & Field, 2015). Pain is typically assessed using the Numerical Rating Scales (NRS) or Wong-Baker Faces Pain Rating Scale (WBFPRS). When using the NRS, a client is asked to verbalize their pain on a 0–10 scale, with 0 being no pain and 10 being the worst possible pain (Jensen & Karoly, 2011). The WBFPRS is typically used if a client is unable to express the pain they are experiencing due to age, aphasia, or other cognitive deficits. It includes six drawings of faces representing six various levels of pain. No pain is scored as 0 with a smiling face and the worst possible pain as 10 with a crying face (Tomlinson, von Baeyer, Stinson, & Sung, 2010).

Hemiplegic shoulder pain is a prevalent symptom after acquired brain injury. The etiology of hemiplegic shoulder pain is multifactorial and may include poor handling or positioning of the UE due to sensory and perceptual deficits, impaired motor control, and altered peripheral or central nervous system activity. This may lead to various clinical diagnoses such as rotator cuff tendonitis or tear, abnormal tone (spasticity or flaccidity), impingement, bursitis, shoulder hand syndrome, adhesive capsulitis, brachial neuralgia, sympathetically mediated pain, and referred pain (Maxwell & Nguyen, 2013; Caglar et al., 2016). Shoulder hand syndrome also known as complex regional pain syndrome (CRPS) is another cause of pain in the hemiparetic UE with symptoms including pain, swelling, altered temperature, altered tactile sensation, and changes in skin texture and color (Harrison & Field, 2015). Treatments will vary based on the extent of the pain reported and any comorbidities. The OT may refer the client to a pain specialist, or a physiatrist if the pain persists.

Spasticity and Hypertonicity (See Chap. 8 for Additional Information)

Spasticity is an upper motor neuron disorder causing a velocity-dependent uncontrolled increase in tonic stretch reflexes resulting in increased muscle tone. Positive signs of the upper motor neuron phenomena include dystonia, spastic co-contraction of agonist and antagonist muscles, spasms, clonus, and abnormal reflex responses, while negative signs include muscle weakness, fatigue, and impaired dexterity. Onset of spasticity is highly variable, although usually occurs within the first few weeks of injury (Thibaut et al., 2013). Although mild spasticity in the beginning stages of motor recovery can be beneficial to help clients hold an object, stand, or transfer, moderate to severe spasticity may cause significant pain, contractures, joint subluxations, or dislocations limiting mobility which may affect a client's

independence in activities of daily living and quality of life (Chang et al., 2013). In the upper extremity, spasticity commonly presents as a flexor synergy pattern of shoulder internal rotation and adduction, with flexion of the elbow, wrist, and fingers and pronation of the forearm. The Modified Ashworth and Tardieu Scales are common clinical measures the OT may use in the evaluation of spasticity (Thibaut et al., 2013). The occupational therapist will identify and continually evaluate how spasticity is limiting an individual's function and adjust the course of treatment accordingly.

Occupational therapists utilize a variety of facilitatory or inhibitory techniques that focus on management of tone, including proprioceptive neuromuscular facilitation techniques, stretching, weight-bearing, electrical stimulation, taping, vibration, or splinting. Functional activities are also incorporated into treatments to improve ROM, strength, and endurance. Client and caregiver education on proper positioning and handling of the upper extremity is a vital component of treatment to prevent pain, injury, or contracture and to improve quality of life and ensure carryover into the home.

If spasticity is resistant to these treatments, a referral to a physiatrist may be necessary, depending on the degree of spasticity present, as mentioned before. Communication between physiatry and occupational therapy is crucial to effectively manage tone and spasticity. The occupational therapist's clinical observations and evaluations will assist the physician in determining whether pharmacological or other methods are an appropriate treatment to maximize motor function and functional independence during ADLs.

Low Tone

Glenohumeral subluxation (GHS) of a hemiplegic shoulder is a frequent complication of motor impairment following acquired brain injury, due to weakness of the shoulder muscles, joint capsules, and ligamentous structures. This may cause incorrect alignment between the humerus and scapula resulting in pain for many clients (Dohle, Rykman, Chang, & Volpe, 2013). GHS is often assessed through palpation of the space between the acromion and the head of the humerus in fingerbreadths or using calipers. If low tone is present, the occupational therapist will educate the client and caregivers on safe positioning and handling of the upper extremity to reduce the risk of subluxation. Positioning recommendations may include using an arm trough, pillows, or lap tray when a client is seated, while pillows are commonly positioned to support the affected upper extremity while in bed. Occupational therapists may also recommend a sling to support the upper extremity during ambulation and transfers.

While a sling can be beneficial to support the shoulder joint and prevent soft tissue damage, caution must be heeded with regard to the amount of time the client remains in the sling, as well as the type of sling used. Traditional slings place the upper extremity in a position of shoulder internal rotation and adduction with

the elbow in flexion. This positioning encourages soft tissue shortening and should be avoided. The GivMohr sling is commonly recommended by the OT as its dynamic qualities encourage movement and allows for more normal postural reactions of the UE while standing.

Kinesiology taping is commonly used by OTs in clients with low tone in addition to traditional rehabilitation techniques and may help reduce pain, improve joint stability, and provide proprioceptive feedback to promote proper body alignment (Jaraczewska & Long, 2006).

Splinting

When clients have experienced changes in the joints and soft tissue structures (like muscles or ligaments) of arms and hands due to spasticity or low tone, the OT will assess if splinting is required to decrease the risk of contractures, assist in the normalization of tone, increase or maintain ROM, prevent skin breakdown, and keep the UE in a functional position. The splint chosen will depend on the degree of tone present at the finger, wrist, and elbow joints. The OT will fabricate or order a splint based on the client's needs and educate the client and caregivers on donning/doffing, the splint wearing schedule, and skin care precautions to ensure proper use of the splint. Common splints for the hand and wrist include the SaeboStretch, resting hand splint, a custom wrist cock-up, and thumb opposition splint. The Dynasplint is a dynamic splint for the elbow that helps to increase joint range of motion by applying a low-load prolonged stretch that can be modified or adjusted as appropriate over time. As an individual's functional movement and tone changes, the splints will require modification as appropriate. The effectiveness of splinting the hemiplegic hand remains questionable, as many double-blind studies have failed to prove significant changes in spasticity of the wrist (Thibaut et al., 2013).

Muscle Strength: MMT/Dynamometry/Pinch Strength

Once AROM/PROM assessments are completed, the therapist will assess the individual's muscle strength utilizing manual muscle testing (MMT), dynamometry, and pinch strength assessments. A 2005 study found that hand strength and functional activities of daily living (ADLs) are directly correlated (Rajan, Premkumar, Rajkumar, & Richard, 2005). Manual muscle testing is a measure used to assess the strength of both upper extremities in either gravity-minimized or against gravity planes. Scores are ranked from no contraction to a contraction against gravity with maximal resistance. MMT will be assessed in all UE planes.

Dynamometry is a quantitative and objective method that assesses an individual's grip strength using a portable handheld dynamometer. Three maximum voluntary contractions are completed with each hand and the scores are averaged and compared

to normative standards (Beebe & Lang, 2009). Decreased grip strength may impact an individual's ability to cut, open containers, or hold an object.

Following an acquired brain injury, an individual may lack enough pinch force to complete daily activities including fastener management, ability to write, or open and close containers. Three types of pinch strength are assessed utilizing a pinch gauge: tip pinch, thumb and index fingers; lateral/key pinch, thumb pad and lateral aspect of index finger; and palmar pinch/3-jaw chuck, thumb, index, and middle fingers (Flinn, Trombly Latham, & Robinson Podolski, 2008). The individual performs each pinch with three successive trials for each hand and the scores are averaged and compared with normative standards.

Coordination: Fine Motor and Gross Motor Control

When assessing coordination occupational therapists utilize client's reports, clinical observation, and standardized testing to determine level of impairment and impact of performance during ADLs. Deficits in coordination following acquired brain injury can greatly impact a client's quality of life and independence during daily tasks. Clients with deficits in coordination may experience difficulty completing basic ADLs such as self-care or difficulty during work- and school-related tasks such as typing or writing.

Fine motor control (FMC) is the ability to effectively utilize the muscle groups in the wrist and hands to manipulate small objects in a timely manner. Standardized assessment of FMC is completed utilizing specialized tests such as the Purdue Pegboard Test, 9-Hole Peg Test, and Grooved Pegboard Test (Lang et al., 2013), or through observing a client's performance during various daily tasks including shoe tying, buttoning, or coin manipulation.

Gross motor control (GMC) involves the larger muscles of the body that control movement. Functional assessment of GMC is completed utilizing specialized tests such as the Box and Block Test or observation of a client completing various tasks such as dressing, brushing hair, or reaching for objects on shelves. Other assessment tools that occupational therapists may utilize to assess coordination are the Jebsen Taylor Test of Hand Function, O'Connor Tweezer Dexterity Test, or Minnesota Dexterity Test (Lang et al., 2013).

Ataxia is a specific coordination impairment, often caused by cerebellar lesions, which inhibits smooth or coordinated gross movements. Basic activities of daily living such as buttoning a shirt or drinking from a cup may become arduous for clients with ataxia.

Sensation, spasticity, clonus, or myoclonus may also impact coordination. Myoclonus is involuntary uncontrollable twitches or jerks caused by sudden muscle contractions that occur in an irregular pattern. Clonus is an involuntary, repetitive, rhythmic contraction of a muscle that is observed after forcible extension of the wrist. Please refer to the spasticity and sensation sections for more information regarding the impact on coordination.

Sensation

Somatosensory impairment can include the inability to sense pain, UE position, touch, and temperature and can have a major impact on coordination and safety. The evaluation and treatment of sensory deficits is important to ensure an individual's safety while completing ADL tasks. Occupational therapists commonly evaluate somatosensory impairments including touch (light touch/discrimination), limb position sense (proprioception/kinesthesia), tactile object recognition (stereognosis), and temperature discrimination. The Semmes Weinstein Monofilament Exam (SWME) is a commonly used measure for assessing sensation. Individuals with sensory deficits may demonstrate difficulty picking up objects or placing their affected arm in a shirt sleeve, or may not be able to sense the water temperature in the shower. An example of impaired sensation includes a client being unaware of their UE positioned near their wheel while seated in a wheelchair, which may result in injury to the UE.

Assessing Functional Use of the Affected Upper Extremity

Commonly used evaluation tools to assess UE impairments and activity limitations include the Fugl-Meyer Assessment with subsets that assess motor function, balance, sensory function, joint function, and pain in hemiplegic clients (Sullivan et al., 2011). The Action Research Arm Test has four subsets including grasping, gripping, pinching, and gross movement to assess UE function (Beebe & Lang, 2009). The Wolf Motor Function Test (WMFT) evaluates UE impairments and activity limitations with 15 tasks, incorporating joint segmented movements and timed integrative functional movements (Lang et al., 2013). The original version of the Chedoke Arm and Hand Activity Inventory (CAHAI) assessment is comprised of 13 items that require the use of both arms (Lang et al., 2013). The Functional Upper Extremity Levels (FUEL) assesses a client's upper extremity functional and physical performance following a stroke (Van Lew et al., 2015), and the Motor Assessment Scale utilizes a task-oriented approach to assess everyday motor function in clients including eight subsets and an assessment of tone. Deciding which measure to use will be a clinical decision by the OT based on the client's cognitive and physical ability to complete the measure, how much time is available, the tools available, and the OT's ability to administer and interpret the measure.

Occupational therapists utilize the results of the evaluation process to determine appropriate treatment options for retraining the upper extremity and improve performance and safety during ADLs. The decision to utilize a specific treatment technique or modality is derived from various frames of reference, including the biomechanical frame of reference and rehabilitative model. The biomechanical model focuses on designing a treatment program to address endurance, ROM, and strengthening exercises including isometric, isokinetic, and isotonic muscle strengthening and training in the affected UE (Flinn, Jackson, McLaughlin Gray, & Zemke, 2008).

Remedial treatment of physical deficits should focus on the functional goals of the individual and can include therapeutic exercise/activities and functional training to improve arm and hand function. Additional specific restorative techniques and tools currently utilized by OTs include functional electrical stimulation (FES), transcutaneous electrical nerve stimulation (TENS), vibration, biofeedback, proprioceptive neuromuscular facilitation (PNF), constraint-induced movement therapy (CIMT), functional training orthoses including the SaeboFlex/Reach, weight-bearing, robotic-assisted therapy, mirror therapy, graded repetitive arm supplementary program (GRASP program), LSVT BIG, virtual reality, mental imagery, bilateral arm training, repetitive task practice (RTP), task-oriented training (TOT), and rhythmic auditory stimulation (RAS). Compensatory interventions will focus on preventing UE deformities and preserving functional performance and safety during activities of daily living.

Case Study #2

YZ is a 32-year-old male with an unremarkable past medical history who experienced left-sided weakness while at work as a corrections officer. Imaging revealed a right intraparenchymal hemorrhage with a partial evacuation 4 days later. YZ received occupational, physical, and speech therapies during his hospitalization and was discharged home after 1 month with a referral to receive outpatient neurorehabilitation services. Upon the initial occupational therapy evaluation, he presented with limited to no active range of motion in his left shoulder, elbow, forearm, and wrist and was unable to extend his digits, nor oppose his thumb. He was unable to complete gross motor coordination testing utilizing the Box and Block Test, nor could he complete fine motor coordination testing utilizing the 9-Hole Peg Test. He presented with significantly reduced gross grasp muscle strength of 11.7 lb (avg. 110.4 ± 21.7). The aforementioned deficits negatively impacted his level of independence in all basic and complex ADLs.

Based on the client's age, motivation, and strong rehabilitation potential, OT services were recommended three times per week with the focus on increasing the functional use of his left nondominant UE, increasing his independence during ADLs, returning to work, and driving. Initial OT treatments included active and active assisted range of motion exercises in planes of motion that were limited, and resistive exercises were incorporated for planes of motion that were within functional limits. Vibration and electrical stimulation were used to elicit wrist and digit extension, while hand strengthening exercises focused on improving grip strength. As YZ regained motor functioning in his left upper extremity, various therapeutic activities were incorporated to improve GMC and FMC, including use of the Flint Music Glove. YZ's occupational therapist frequently discussed any difficulties experienced in the home or community in an effort to develop treatment plans that would improve independence in ADLs and prepare him for return to work and driving.

YZ demonstrated significant active gains in functional use of his left upper extremity throughout his 8-month course of OT treatment. At the time of discharge, all left UE active planes of motion were within normal limits with full (5/5) strength and gross grasp increased to 110.8 lb (avg.110.4 ± 21.7). He could complete the Box and Block Test for GMC with 51 blocks in 1 min (avg. 81.3 ± 8.1) and FMC tests including the 9-Hole Peg Test in 24.89 s (avg. 18.47 ± 2.94) and Grooved Pegboard Test in 83.2 s (avg. 65.3 ± 8.5). He became independent in all basic and complex ADLs and was referred for an on-road driving evaluation by his occupational therapist, which he successfully passed. YZ was scheduled to return to work with accommodations (such as a gradual increase from light duty) after receiving medical clearance. YZ was advised to continue engagement in his home exercise programs to maintain the gains achieved in therapy.

Cognition

Cognition refers to a range of information processing functions that enable people to concentrate, think, plan, remember, problem-solve, self-monitor, and execute goal-directed behavior (AOTA, 2013; Radomski, Anheluk, Bartzen, & Zola, 2016). Acquired brain injury often leads to impairments in a variety of cognitive domains, which together often leads to problems in everyday functioning. OT's distinctive role when addressing cognition is the interaction between cognition and its effect on functional participation and independence. The occupational therapist will evaluate an individual's cognitive status and determine its impact on function and, subsequently, develop a treatment plan to address the area(s) of deficit. Intervention includes strategies to enhance the cognitive skill, or incorporate the use of compensatory strategies, with the goal of increasing an individual's safety and level of independence. One way to remediate cognitive skills is computerized exercises, which have been shown to measurably improve cognitive skills such as memory (O'Neil-Pirozzi & Hsu, 2016). In this study by O'Neil-Pirozzi and Hsu (2016), a number of participants reported a positive impact of these exercises on cognitive abilities, life satisfaction, and everyday functioning.

Attention

According to Tsaousides and Gordon (2009), attention is a "complex mental activity that refers to how an individual receives and begins to process internal and external stimuli." Sohlberg and Mateer (1987) proposed that attention can be organized into five hierarchical levels: focused attention, sustained attention, selective attention, alternating attention, and divided attention. Deficits in attention can significantly reduce a person's ability to function in all activities of daily living, as attention is a foundational skill that underlies and supports all other cognitive abilities

(Haskins et al., 2013). Deficits in attention may pose safety risks (e.g., a child's safety may be at risk if the parent is unable to sustain his/her attention when bathing the child, or gets distracted while the children are playing outside near a busy street).

Focused attention is the most basic level of attention and refers to a person's ability to recognize and perceive specific sensory information. Sustained attention refers to the ability to maintain focus on one task over an extended period of time (e.g., reading a book or having a conversation). Selective attention refers to the ability to maintain focus while filtering out distractors. A distractor may either be internal or external. An example of internal distractors includes being hungry or having a headache which may directly influence a person's ability to focus, whereas an example of an external distraction may be the sound of the air-conditioning vent above your head. Most people have no difficulty filtering out the noise, or may not even notice the background noise; however, a person with an acquired brain injury may find the noise unbearable and may not be able to focus on the task at hand. Initially, distractions and complexity of tasks may be kept to a minimum when focusing on attention. As clients recover, distractions should be introduced into many of the activities during treatment sessions to help improve complex attention skills. The occupational therapist may begin introducing distractions into the environment such as background noise (e.g., music or someone else talking on the phone). The challenge is for the client to be able to achieve accuracy and maintain mental endurance in the midst of such distractions. When distractions are first being introduced, it may be best to have the client do activities that are familiar and have been successful to the client. Once mastery is achieved, it would be best to move to activities that the person is unfamiliar with or ones that require new learning.

Alternating attention refers to the ability to switch focus between tasks. A client who sustained a brain injury may have difficulty resuming engagement or focus on a task when interrupted. When a client has difficulty alternating their attention, the occupational therapist can create interventions requiring the client to switch focus between functional tasks to improve these skills. For example, an individual may be folding laundry when his/her child asks for help with his/her homework. The person must be able to shift his/her attention to the new task of assisting the child complete the homework, but then resume engagement in the initial task of folding laundry.

The final, and most complex, level is divided attention. This refers to the ability to multitask, or attend to more than one stimulus simultaneously. A person with impaired divided attention may notice difficulty answering questions that a doctor is asking while trying to fill out the medical office forms. Driving and meal preparation are two examples of tasks that require the ability to successfully divide attention. When driving, an individual must not only be able to focus on what he/she is doing but also focus on the actions of other drivers on the road and other stimuli in the environment (e.g., maintaining appropriate speed, anticipating traffic lights, actively scanning the road). During meal preparation, an individual may have to focus on preparing multiple dishes simultaneously. Deficits with divided attention may result in burning the main dish while preparing a side dish for dinner.

Attention Process Training (APT) is a program developed by Sohlberg and Mateer (1987) that is used to rehabilitate attention following a brain injury. It is

designed to be used with children, adolescents, and adults with mild, moderate, or severe attention deficits following a brain injury or other neurological conditions. Tasks are presented in a hierarchical manner, from simple to more complex. Stimuli can be presented in an auditory or visual manner (Sohlberg & Mateer, 1987).

Time Pressure Management Training (Fasotti, Kovacs, Eling, & Brouwer, 2000) is another technique used to rehabilitate attention processing skills and speed. It serves to provide individuals with compensatory strategies to deal with time pressure in daily life (Winkens, Van Heugten, Wade, & Fasotti, 2009). This type of training can be utilized to increase insight into deficit areas, as well as incorporate improvement of these skills into daily life.

Executive Functions

Executive functions refer to integrative cognitive processes that determine goal-directed and purposeful behavior (Haskins et al., 2013). It refers to an individual's coordinated ability to plan, initiate, organize, and execute complex, purposeful behavior (Cramm, Krupa, Missiuna, Lysaght, & Parker, 2013). Executive dysfunction is common in individuals with a frontal lobe injury. Injury in this area of the brain can impact an individual's ability to perform daily tasks in the home and community that he/she didn't have trouble performing prior to the injury. The OT may provide structured cueing and compensatory strategy training including structured problem-solving training techniques such as goal management training (Levine et al., 2000) and/or facilitate simulated or supervised engagement in functional tasks in the home and community.

Impairment of executive functions may impact a person's ability to initiate engagement in a task. Initiation is the "getting started" phase of an activity. A client with reduced initiation may leave the recycling items sitting next to the front door for days before he/she takes them out to the bin. If a client has difficulty initiating, the occupational therapist may provide verbal or visual cueing to facilitate participation in a task, such as the use of a daily checklist (e.g., a checklist consisting of all activities completed during a person's morning routine). Having the client set deadlines for tasks that need to be completed, or setting up a reward system for tasks completed, is also an additional strategy that may be used to increase the client's initiation. On the other hand, a client with a brain injury may have difficulty terminating, or stopping, engagement in a task. They may demonstrate difficulty finishing the task of washing dishes, although the dishes are already clean. The occupational therapist may recommend the use of a timer with an audible alarm to facilitate termination of the task.

The ability to plan, organize, and sequence the steps of a task is necessary for successful task completion. An example would be planning a vacation. One must determine the vacation destination, make all necessary traveling arrangements, develop an itinerary, and estimate the total cost for meals, activities, and souvenirs while staying within the desired budget. The occupational therapist would identify which specific skill(s) is impeding performance and then provide appropriate

intervention to address the underlying skills with task-based performance. After addressing the specific skill(s) during treatment sessions, the occupational therapist would assess the client's ability to generalize these skills during occupational performance. As in the situation mentioned earlier, generalization of the performance skills would be the ability to complete a simulated vacation planning activity during a treatment session with task demands paralleling the demands of real life.

Memory (Refer to Chap. 9 for Further Detail)

Memory refers to a person's ability to hold on to pieces of information for either a short or an extended period of time. Remembering a phone number while dialing is an example of short-term memory, while recalling your significant other's birthday is an example of long-term memory.

As mentioned previously, it is an occupational therapist's unique role to determine the interaction between cognition and functional performance. Examples of functional memory impairments may include forgetting to transfer the laundry from the washer to the dryer, forgetting to turn off the oven when cooking a meal, or not recalling strategies learned during previous therapy sessions. The client might be educated on use of external aids, such as use of a daily or weekly checklist, use of a written visual planner, or use of an alarm system. The occupational therapist will develop functional goals incorporating education on and the use of compensatory strategies for memory or other cognitive deficits.

Praxis

Praxis is the ability to perform skilled or learned movements and is essential for participation in activities of daily living. The inability to perform such movements is called apraxia and can negatively impact quality of life after an acquired brain injury. Apraxia is a motor planning disorder caused by damage to the cerebrum and can be classified into three subtypes: ideomotor, ideational, and limb-kinetic apraxia.

Ideomotor apraxia impacts the ability to perform a common gesture upon verbal command, despite intact sensory, motor, and language function (e.g., waving, using a hammer, or shaking someone's hand). Clients with ideomotor apraxia present with uncoordinated movements while performing a task. "Limb-kinetic apraxia differs from classical ideomotor apraxia in a number of ways. For instance, limb-kinetic apraxia tends to be independent of modality (e.g., verbal command versus imitation), and there is typically no voluntary-automatic dissociation" (Goldmann Gross & Grossman, 2008). Limb-kinetic apraxia is the inability to produce precise movements resulting in clumsy distal limb movements.

Ideomotor and limb-kinetic apraxia may affect coordination of movements during ADLs, while ideational apraxia is the inability to conceptualize, sequence,

and carry out a motor act. Individuals with ideational apraxia are unable to interact effectively with objects or his/her environment. He/she does not have the "idea" of the object's use—what to do with the object or how to use the object. This may present as an individual attempting to brush his/her limb with a toothbrush during his/her morning routine. The occupational therapist will help facilitate the relearning of these once familiar, routine tasks. Relearning of the task is achieved through repeated engagement in the desired functional task, utilizing hand-over-hand assistance, or the use of cueing to facilitate successful participation.

Vision and Perception (Refer to Chap. 6 for Further Detail)

Following an acquired brain injury, clients can experience a wide range of visual disturbances and perceptual changes. More specifically, clients may experience diplopia (double vision), difficulty with visually focusing, oculomotor dysfunction, and difficulty with saccades and smooth ocular pursuits, among other things (Cohen, n.d.). Visual scanning can be evaluated utilizing a cancellation task to determine ability to locate all desired visual targets, organize the visual scanning pattern, and detect any inattention to one or both sides of a near point visual field. Occupational therapists also utilize informal measures such as assessing the client's ability to locate the clinician's finger in various locations in the visual field, having a client track a visual stimulus, and/or having a client move between multiple visual stimuli to assess the aforementioned skills. Functional environmental awareness can be evaluated and treated utilizing an environmental scavenger hunt, or having clients locate specific items in a given room. An important role of the occupational therapist is providing a referral to and/or being in communication with a neuro-optometrist or neuro-ophthalmologist to obtain more comprehensive information regarding the client's visual and perceptual functioning.

Occupational therapists will utilize various methods to remediate any of the previously mentioned skills. In order to remediate these skills, occupational therapists can utilize functional visual scanning activities such as having a client locate specific items on a shelf to simulate grocery shopping, or locating target items in a written text. Occupational therapists may also educate clients on oculomotor exercises challenging skills such as visual fixation, smooth pursuits, saccades, or vergence. As a means to compensate for reduced visual field or unilateral inattention, occupational therapists educate clients on utilizing the "lighthouse strategy" which includes scanning to both sides of a visual field like the lights at the top of a lighthouse, in order to improve awareness of the entire visual field, ensure safety, and prevent injury (Niemeier, 1998). Using the lighthouse strategy is useful while the client functionally ambulates throughout the home and community, or when searching for items in a given space to ensure thoroughness and safety.

Unlike a visual field loss, the areas of the brain dedicated to eyesight are intact in clients that present with visual spatial neglect, or hemispatial neglect; however, treatment strategies used to treat neglect are similar to treatment utilized when

addressing a visual field loss. For example, visual scanning training is utilized to facilitate awareness and attention to the affected side. The OT may educate clients and caregivers on use of compensatory strategies, such as the lighthouse strategy, to promote attention to the neglected side, or use visual anchors to cue the client to look to the affected side. Use of external auditory cues may be utilized, such as attaching a bell on the limb of the affected side, with the purpose of promoting awareness to the neglected side and encouraging use of the affected side of the body, when the client hears the auditory input provided. Clients with neglect may be unaware of this impairment; therefore, treatment of neglect may sometimes incorporate cognitive retraining to increase a client's insight into his/her deficit(s).

Visual perception is the multifaceted process of receiving external stimuli, processing the sensory information, and converting it into meaningful sensory knowledge which provides individuals with a visual interpretation of the environment (Cooke, McKenna, & Fleming, 2005). In other words, visual perception is the brain's interpretation of what the eyes are seeing through a combination of visual and cognitive processes. The ramification of visual perceptual deficits can be seen in various ways. "The disorders may result in difficulties organizing, processing, and interpreting information perceived visually, and acting appropriately based of this information" (Cooke et al., 2005). Occupational therapy treatment techniques for perceptual deficits fall into two categories: remedial and adaptive. Use of either approach is based on client factors and specific areas of difficulty (Neistadt, 1990). The occupational therapist, client, and caregivers will determine the appropriate treatment techniques based on a variety of factors.

Visual perception encompasses body scheme, left/right discrimination, understanding of directionality, and positioning in space, all of which can be addressed in occupational therapy treatment sessions. Individuals with visual perceptual deficits can have difficulty interpreting information about themselves and their environment. These skills are important for successful and safe engagement in basic and complex ADLs. An example is not bumping into objects in the environment, like a hot stove in a crowded kitchen, by knowing where the body is positioned in space.

Visuospatial relations include the ability to comprehend and interpret the position of objects and their interaction with each other, as well as a person with his/her environment. This may be presented as difficulty negotiating the stairs due to difficulty determining the distance and height required to move his/her lower extremity to ascend and descend the stairs safely. Impairment with visuospatial orientation, or the rotation of an item in space, might present as an individual having difficulty orienting his/her shirt correctly when getting dressed. The individual may attempt to thread his/her head through the smaller arm hole of the shirt. Figure-ground entails the ability to identify and locate items from the environment or background. Visual closure involves the ability to finish the complete shape of an object, if the item is not fully in view. Deficits in either of these areas may impact a client's ability to locate a specific utensil from a drawer of cooking utensils or car keys from a cluttered cabinet.

Occupational therapists use a range of assessment tools and screenings to measure visual perceptual abilities and to determine their impact on function and safety.

These may range from the completion of tabletop tasks to clinical observation of the client completing a functional task. Subsequently, treatment may consist of similar activities to address the deficits observed during the assessment. Occupational therapists, neuro-optometrists (see Chap. 6), and/or neuro-ophthalmologists work collaboratively and interactively to intervene and provide required interventions and strategies for deficits in vision and perception (Cooke et al., 2005).

Discharge

Discharge from skilled outpatient OT services can occur for a variety of reasons. This may include achieving goals and returning to desired occupations, achieving maximum benefit from OT services in this setting, self-discharge by the client, or due to exhaustion of insurance benefits. Discharge recommendations will vary depending on client goals, but often include referral to support groups and community programs. Prior to discharge, the OT and client will review previously dispensed home exercise programs if still appropriate and adjust or upgrade the home exercise program to be continued after discharge. Discharge recommendations can also include return to work if appropriate, or participation in a structured day program, volunteering, or vocational training. In preparation for discharge, the occupational therapist, client, interdisciplinary team, and caregivers work together to determine desired and appropriate discharge recommendations.

The Integrative Role of OT with the Interdisciplinary Team

It is imperative that occupational therapists function as members of the interdisciplinary team. Frequent communication between team members facilitates best practice for each discipline. Interdisciplinary communication allows each discipline to reinforce carryover of compensatory strategies, cues, and assistance to promote consistency. The occupational therapist's role is to help improve client's performance and independence on functional and meaningful daily activities while incorporating underlying physical, cognitive, visual, or behavioral deficits. It is the responsibility of the occupational therapist, and all interdisciplinary team members, to communicate with all disciplines, clients, and caregivers to promote an effective team approach to therapy.

Summary of Chapter

Occupational therapy's role in neurological rehabilitation is to assist clients to maximize independence in all areas of occupation that are individually meaningful and realistic to each client. In this specialized setting, occupational therapists address

remediating underlying physical, cognitive, and/or visual and perceptual skills in order to facilitate improved performance and safety during engagement in daily activities. Occupational therapists also educate clients and caregivers on compensatory strategies for basic and complex ADL performance. The client, caregivers, occupational therapist, and interdisciplinary team all collaborate to promote a functional and meaningful recovery following an acquired brain injury.

References

American Occupational Therapy Association. (2013). Cognition, cognitive rehabiliation, and occupational performance. American Journal of Occupational Therapy, 67, S9–S31. https://doi.org/10.5014/ajot.2013.6759

American Occupational Therapy Association. (2014). Occupational therapy practice framework: Domain and process (3rd ed.). *American Journal of Occupational Therapy, 68*(Suppl. 1), S1–S48. https://doi.org/10.5014/ajot.2014.682006

Beebe, J. A., & Lang, C. E. (2009). Relationships and responsiveness of six upper extremity function tests during the first 6 months of recovery after stroke. *Journal of Neurologic Physical Therapy, 33*(2), 96–103. https://doi.org/10.1097/NPT.0b013e3181a33638

Caglar, S. N., Akin, T., Aytekin, E., Komut, E. A., Ustabasioglu, F., Okur, S. C., … Yalcinkaya, E. Y. (2016). Pain syndromes in hemiplegic patients and their effects on rehabilitation results. *Journal of Physical Therapy Science, 28*(3), 731–737.

Chang, E., Ghosh, N., Yanni, D., Lee, S., Alexandru, D., & Mozaffar, T. (2013). A review of spasticity treatments: Pharmacological and interventional approaches. *Critical Reviews in Physical and Rehabilitation Medicine, 25*(1–2), 11–22.

Christiansen, C. H., & Hammecker, C. L. (2001). Self care. In B. R. Bonder & M. B. Wagner (Eds.), *Functional performance in older adults* (pp. 155–175). Philadelphia, PA: F. A. Davis.

Cohen, A. H. (n.d.). *Visual problems associated with acquired neurological events.* Retrieved from http://www.braininjuries.org/brain_injury_double_vision.html

Cooke, D., McKenna, K., & Fleming, J. (2005). Development of a standardized occupational therapy screening tool for visual perception in adults. *Scandinavian Journal of Occupational Therapy, 12*, 59–71.

Cramm, H., Krupa, T., Missiuna, C., Lysaght, R. M., & Parker, K. C. H. (2013). Broadening the occupational therapy toolkit: An executive functioning lens for occupational therapy with children and youth. *American Journal of Occupational Therapy, 67*, e139–e147.

Davis, D., Wheeler, S., & Acord-Vira, A. (2017). Evaluation of return to work skills following traumatic brain injury. *SIS Quarterly Practice Connections, 2*(1), 26–28.

Dohle, C., Rykman, A., Chang, J., & Volpe, B. (2013). Pilot study of a robotic protocol to treat patients with chronic stroke. *Journal of Neuroengineering and Rehabilitation, 10*, 88.

Doucet, B. M. (2012). Neurorehabilitation: Are we doing all that we can? *American Journal of Occupational Therapy, 66*(4), 488–493.

Fasotti, L., Kovacs, F., Eling, P., & Brouwer, W. H. (2000). Time pressure management as a compensatory strategy training after closed head injury. *Neuropsychological Rehabilitation, 10*(1), 47–65.

Flinn, N. A., Jackson, J., McLaughlin Gray, J., & Zemke, R. (2008). Optimizing abilities and capacities: Range of motion, strength, and endurance. In M. V. Radomski & C. A. Trombly Latham (Eds.), *Occupational therapy for physical dysfunction* (pp. 573–597). Philadelphia, PA: Lippincott Williams & Wilkins, a Wolters Kluwer business.

Flinn, N. A., Trombly Latham, C. A., & Robinson Podolski, C. (2008). Assessing abilities and capacities: Range of motion, strength, endurance. In M. V. Radomski & C. A. Trombly Latham

(Eds.), *Occupational therapy for physical dysfunction* (6th ed., pp. 91–185). Philadelphia, PA: Lippincott, Williams & Wilkins.

Goldmann Gross, R., & Grossman, M. (2008). Update on apraxia. *Curr Neurol Neurosci Rep, 8*(6), 490–496.

Hardwick, D., & Lang, C. (2011). Scapula and humeral movement patterns and their relationship with pain: A preliminary investigation. *International Journal of Therapy and Rehabilitation, 18*(4), 210–220.

Harrison, R. A., & Field, T. S. (2015). Post stroke pain: Identification, assessment, and therapy. *Cerebrovascular Diseases, 39*(3–4), 190–201. https://doi.org/10.1159/000375397

Haskins, E., Cicerone, K., Dams-O'Connor, K., Eberle, R., Langenbahn, D., & Shapiro-Rosenbaum, A. (2013). *Cognitive rehabilitation manual* (1st ed.). Reston, VA: American Congress of Rehabilitation Medicine.

Jaraczewska, E., & Long, C. (2006). Kinesio taping in stroke: Improving functional use of the upper extremity in hemiplegia. *Topics in Stroke Rehabilitation, 13*, 31–42.

Jensen, M., & Karoly, P. (2011). Self-report scales and procedures for assessing pain in adults. In *Handbook of pain assessment* (3rd ed., pp. 19–44). New York, NY: Guilford Press.

Lang, C. E., Bland, M. D., Bailey, R. R., Schaefer, S. Y., & Birkenmeier, R. L. (2013). Assessment of upper extremity impairment, function, and activity following stroke: Foundations for clinical decision making. *Journal of Hand Therapy, 26*(2), 104–115.

Levangie, P. K., & Norkin, C. C. (2005). *Joint structure and function: A comprehensive analysis* (4th ed.). Philadelphia, PA: F.A. Davis.

Levine, B., Roberson, I. H., Clare, L., Carter, G., Hong, J., Wilson, B. A., … Stuss, D. T. (2000). Rehabilitation of executive functioning: An experimental validation of goal management training. *Journal of International Neuropsychology & Sociology, 6*, 299–312.

Maxwell, A. M. W., & Nguyen, V. Q. C. (2013). Management of hemiplegic shoulder pain. *Current Physical Medicine and Rehabilitation Reports, 1*, 1–8.

Mercier, L., Audet, T., Herbert, R., Rochette, A., & Dubois, M. F. (2001). Impact of motor, cognitive and perceptual disorders on ability to perform activities of daily living after stroke. *Stroke, 32*, 2602–2608.

Naghdi, S., Ansari, N. N., Mansouri, K., & Hasson, S. (2010). A neurophysiological and clinical study of Brunnstrom recovery stages in the upper limb following stroke. *Brain Injury, 24*(11), 1372–1378.

Neistadt, M. E. (1990). A critical analysis of occupational therapy approaches for perceptual deficits in adults with brain injury. *American Journal of Occupational Therapy, 44*(4), 299–304.

Niemeier, J. P. (1998). The lighthouse strategy: Use of a visual imagery technique to treat visual inattention in stroke patients. *Brain Injury, 12*(5), 399–406. https://doi.org/10.1080/026990598122511

O'Neil-Pirozzi, T. M., & Hsu, H. (2016). Feasibility and benefits of computerized cognitive exercise to adults with chronic moderate-to-severe cognitive impairments following an acquired brain injury: A pilot study. *Brain Injury, 30*(13–14), 1617–1625. https://doi.org/10.1080/02699052.2016.1199906

Parham, L. D., & Fazio, L. S. (Eds.). (1997). *Play in occupational therapy for children*. St. Louis, MO: Mosby.

Radomski, M. V., Anheluk, M., Bartzen, M. P., & Zola, J. (2016). Effectiveness of interventions to address cognitive impairments and improve occupational performance after traumatic brain injury: A systematic review. *American Journal of Occupational Therapy, 70*, 7003180050.

Rajan, P., Premkumar, R., Rajkumar, P., & Richard, J. (2005). The impact of hand dominance and ulnar and median nerve impairment on strength and basic daily activities. *Journal of Hand Therapy, 18*(1), 40–45.

Sohlberg, M. M., & Mateer, C. A. (1987). Effectiveness of an attentional training program. *Journal of Clinical and Experimental Neuropsychology, 9*, 117–130.

Sullivan, K. J., Tilson, J. K., Cen, S. Y., Rose, D. K., Hershberg, J., Correa, A., … Duncan, P. W. (2011). Fugl-Meyer assessment of sensorimotor function after stroke: standardized training procedure for clinical practice and clinical trials. *Journal of the American Heart Association, 42*, 427–432.

Thibaut, A., Chatelle, C., Ziegler, E., Bruno, M.-A., Laureys, S., & Gosseries, O. (2013). Spasticity after stroke: Physiology, assessment and treatment. *Brain Injury, 27*(10), 1093–1105.

Tomlinson, D., von Baeyer, C. L., Stinson, J. N., & Sung, L. (2010). A systematic review of faces scales for the self-report of pain intensity in children. *Pediatrics, 126*, 1168–1198.

Tsaousides, T., & Gordon, W. A. (2009). Cognitive rehabilitation following traumatic brain injury: Assessment to treatment. *Mount Sinai Journal of Medicine, 76*, 173–181.

Van Lew, S., Geller, D., Feld-Glazman, R., Capasso, N., Dicembri, A., & Pinto Zipp, G. (2015). Development and preliminary reliability of the functional upper extremity levels (FUEL). *American Journal of Occupational Therapy, 69*(6), 6906350010p1–6906350010p5. https://doi.org/10.5014/ajot.2015.016006

Winkens, C., Van Heugten, C. M., Wade, D., & Fasotti, L. (2009). Training patients in time pressure management, a cognitive strategy for mental slowness. *Clinical Rehabilitation, 23*(1), 79–90.

World Federation of Occupational Therapists. (2012). *Definition of occupation.* Retrieved from http://www.wfot.org/aboutus/aboutoccupationaltherapy/definitionofoccupationaltherapy.aspx

Chapter 8
The Role of the Physical Therapist on the Neuro-Rehabilitation Team

Kristen Murray, Nicole Aquino, and Julianne Nugent

Introduction to Physical Therapy and Acquired Brain Injury (ABI)

A physical therapist (PT) in a neuro-rehabilitation population is one who specializes in the evaluation and treatment of individuals with movement problems due to disease or injury of the nervous system in which the goal is to aid in recovery, minimize, and/or compensate for any residual functional alterations or deficits.

Unlike orthopedic injuries, neurological insult or injury can result in total body or global functional deficits. It is imperative for a neurologic PT to be able to perform a comprehensive clinical examination which includes review of past medical history (PMH), subjective reports, balance, transfer ability, gait, strength, range of motion (ROM), muscle tone, coordination, vision, vestibular, proprioceptive, posture, deep tendon reflexes (DTR), sensation and safety awareness. The PT must also consider any comorbidities, client age, and prior level of function. With the information obtained from the examination and evaluation, the PT will develop a customized and individualized plan of care, addressing and prioritizing the most debilitating deficits in a systematic and logical manner.

There is no "one-size fits all" recovery protocol for neurological injuries such as cerebrovascular accidents (CVA), aneurysm ruptures, or anoxic injuries like there would be for a total hip replacement. Although individuals who suffer a neurological

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injury can present with similar deficits such as pyramidal strength imbalances, it is the role of the PT to determine exactly how these deficits will affect the individual and the best approach to address them.

Evaluation and Treatment

ROM/Strength

During the evaluation, the physical therapist will assess the client's range of motion and strength. When bringing the ambulatory client back to the evaluation room, a quick look at the client's gait can help predict if manual muscle testing (MMT) will be against gravity or gravity eliminated. The therapist must also consider if the client will be able to tolerate traditional MMT positioning. For example, if a client is unable to tolerate side-lying on his or her hemiparetic side, or lying prone due to shoulder pain, the therapist must assess the client in a more comfortable position. The physical therapist must first start by assessing active range of motion (AROM) during the evaluation. If the client appears to be lacking AROM, the physical therapist should further assess the joint passively.

There are several different muscle grade scales that are commonly used when assessing muscle strength. The most common muscle grading scale consists of a 0–5 numeric scale. Scoring a 0/5 would be interpreted as an absent muscle contraction, whereas 5/5 would be interpreted as a muscle contraction sustaining maximal resistance.

During the evaluation, if the physical therapist determines that a joint has limited range of motion, both actively and passively, the therapist should assess the end feel of the joint. If the end feel appears to be firm, the joint is limited within, and the therapist can look into passive stretches and joint mobilizations to improve range of motion to further improve the client's function. Typically, the clients in this setting have decreased strength, which limits the motion in the joint. Over time, if the joint is not stretched passively, the joint will begin to lose motion and ultimately lead to a contracture. Therapists must be aware of this and refer clients for bracing as needed as well as encourage frequent positional changes throughout the day to prevent the development of contractures.

If the client can achieve full AROM, the therapist can progress to assessing strength utilizing manual muscle testing. It is important to thoroughly and accurately assess strength and ROM, in order to predict how these deficits and limitations will hinder the client's mobility. The examination should always begin by assessing ROM and strength prior to assessing coordination. Clients who present with impairments in these areas will present with coordination deficits such as decreased accuracy and control, due to lack of range of motion and strength.

A PT's treatment plan will be tailored to each client's functional deficits. Therapeutic exercise is one treatment approach used to target decreased muscle strength of the upper and lower extremities. Decreased muscle strength can lead to

multiple compensatory mechanisms which decrease energy efficiency, ultimately limiting client's tolerance to daily activities. Our goal as therapists is to limit compensatory movements to allow for proper muscle firing and mechanics to retrain the brain how to move in the most energy efficient manner. It is important to select the most appropriate exercise for the client. If the exercise is too challenging for the client, it is likely the therapist will see increased compensatory strategies to complete the task.

When determining the correct exercise to prescribe to each client, a therapist must accurately grade the strength of each individual muscle group. If a client presents with a 1 or 1+/5 muscle strength, the physical therapist will prescribe isometric exercises to the client. An isometric exercise is defined as an exercise where the muscle length and joint don't move or change in length during the contraction. This contraction is typically a 10 s hold. If a client is unable to move his or her lower extremity against gravity, the therapist must prescribe gravity-eliminated exercises. Once the client builds up enough strength to move his or her lower extremity against gravity, the therapist will advance the exercise program.

Open chain exercise is defined as exercises where the most distal segment, typically the hand or foot, is moved freely. These exercises typically target and isolate one muscle group. Closed chain exercise is defined as exercises where the most distal segment is typically fixed to a support surface. These exercises typically target multiple muscle groups and focus on more functional movements. A study posted in the Journal of Athletic Training by Bunton, E. E. discusses the benefits from closed kinetic chain exercises, "Closed kinetic chain rehabilitation is shown to decrease shear forces, increase proprioception, and increase muscle group coordination. Closed kinetic chain rehabilitation is an economical, efficient, and effective means of rehabilitation, with the ultimate goal of enhancing proprioception, thus gaining lower extremity joint stability" (Bunton, Pitney, Cappaert, & Kane, 1993). Targeting closed chain activities to activate multiple muscle groups will improve motor coordination as well as improve proprioceptive input during the task. Allowing clients to identify certain muscle activation patterns for specific functional tasks will allow for further carryover from session to session.

Coordination

"Coordination is the ability to execute smooth accurate, controlled movement" (O'Sullivan, Schmitz, & Fulk, 2014). In order to successfully coordinate a movement, one's somatosensory, visual, and vestibular systems must work together in conjunction with the neuromuscular system. There are several types of coordination that physical therapists assess. Intralimb coordination considers movements within a single limb. For example, assessing the use of one shoulder flexing and extending. Interlimb coordination considers two or more limbs moving together. For example, assessing a client walking with a reciprocal arm swing. Visual motor coordination considers the integration of visual and motor tasks to accomplish a goal. For example, writing,

drawing, or riding a bike. Eye–hand coordination utilizes visual motor coordination to complete activities of daily living, such as brushing teeth, bathing, and eating. It is important to perform a coordination examination to determine how one's initiation, control, timing, sequencing, and accuracy of movements will impact overall functional mobility and activities of daily living.

Non-equilibrium coordination tests will assess how the client moves without challenging the client's balance. Does an increase of speed affect the quality of movement? Does occluding vision alter the quality of movement? There are several tests therapists use to identify non-equilibrium coordination deficits. Finger-to-nose is tested by positioning the client's shoulder in 90° of abduction with their elbow extended. The client is asked to bring the tip of his or her index finger to the tip of his or her nose. The therapist can start unilaterally and advance to bilateral and then to vision occluded. Other coordination tests include finger to therapist's finger, finger to finger, alternate finger to nose, finger opposition, rapid alternating movements, tapping of the hands/feet, and heel to shin.

Sensation

When evaluating sensation, the physical therapist should begin testing superficial and then progress to deep sensation. Superficial sensory testing will evaluate the anterior spinothalamic tract, ultimately assessing light touch. The physical therapist will use a cotton ball, tissue, or soft fabric when assessing light touch. The therapist will then assess the lateral spinothalamic tract, ultimately assessing pain (sharp/dull) and temperature. The equipment needed for these assessments include a clean unused safety pin and a hot/cold discrimination kit. When assessing sensation, the physical therapist should always explain and demonstrate the procedure and then perform the assessment.

When assessing deep sensory testing, the therapist will be evaluating the dorsal column medial lemniscal, consisting of proprioception of static positioning, vibration perception, and kinesthesia awareness. The therapist should test the client's range of motion prior to assessing proprioception, starting at the great toe and progressing proximally throughout each joint. Testing at the most distal joint then moving proximally will be the most efficient in determining if sensation is absent. The therapist will move the joint throughout its mid-range of motion, staying away from end ranges. The therapist will hold the joint in a static position, asking the client if he or she feels the extremity "up" or "down". The client's eyes will be closed when performing this test. This examination assesses joint position sense and awareness of joints at rest. Vibration sensation is tested by application of rapidly oscillating or vibratory stimuli. It is common for elderly clients to have diminished vibration sense in their feet. Kinesthesia testing is similar to the static positional sense assessment. The client is asked to verbalize the direction of movement while the extremity is in motion by the therapist. Another way to assess this is to have the client simultaneously replicate the motion with the opposite extremity.

When assessing combined cortical testing, the client must have intact light touch in order to proceed to this part of the examination. Stereognosis perception examines tactile object recognition. The objects used are small familiar objects such as a paper clip, coin, and key and are shown to the client beforehand. The therapist places one object in the client's hand and he or she manipulates the object and verbally identifies what it is with eyes closed. Tactile localization assesses the ability to localize touch sensation. The client is asked to identify the location by pointing or verbally describing the area that has been touched. Two-point discrimination assesses the ability to perceive two points applied to the skin simultaneously with a two-point discriminator. The client will respond whether they feel one or two stimuli. With each successful application, the two tips are continuously brought closer together until the stimuli are perceived as one. Normal distance for fingertip is 3–4 mm and 20 mm or greater for larger areas such as a back.

Graphesthesia assesses the ability to recognize letters, numbers, or shapes traced on the palm of the client's hand. This test is useful as a substitute for stereognosis when paralysis prevents grasping an object. Barognosis assesses the recognition of weight. Small objects of similar size, but varying weights are place in the client's hand and he or she must identify if the object is "heavier" or "lighter" than the object before it.

When assessing sensation, PTs must take into consideration the client's cognition, as the test is very subjective based on the client's response. A PT must consider comorbidities and other underlying factors that may alter results.

Muscle Tone, Spasticity, and Synergistic Patterns

According to O'Sullivan sixth Edition, "Tone is defined as the resistance of muscle to passive elongation or stretch" (O'Sullivan et al., 2014). There are two main categories that muscle tone is divided into. One category is hypertonia, which involves increased tone above the normal resting level. "Spasticity is a motor disorder characterized by a velocity dependent increase in muscle tone with increased resistance to stretch. Increased tonic contraction of muscles is seen at rest, resulting in abnormal typical resting patterns" (O'Sullivan et al., 2014). "Clonus is characterized by cyclical, spasmodic alteration of muscular contraction and relaxation in response to sustained stretch of a spastic muscle" (O'Sullivan et al., 2014). Clonus is most commonly found in the plantar flexors. When assessing spasticity, the therapist must place the client in a relaxed position in supine. The limb is first tested slowly, establishing an end point of range of motion. The therapist will perform the test by quickly moving the limb in the direction of stretching the muscles being tested. The key to the test is the speed of which the limb is moved. Spasticity may begin to diminish with multiple repetitions therefore it is essential to assess the spasticity within the first few repetitions. All upper and lower extremities should be tested, in order to compare and accurately assess. In order to objectively measure a client's spasticity, physical therapists utilize the Modified Ashworth Scale (see Chap. 4, Table 4.4). This scale is

commonly used across rehabilitation facilities and clinics, allowing therapists to accurately quantify the severity of spasticity in a client.

The second category of tone is hypotonia, which involves decreased tone, below the normal resting level. "Flaccidity is present immediately after a stroke and is due primarily to the effects of cerebral shock. It is generally short lived, lasting a few days or weeks" (O'Sullivan et al., 2014). Depending on the location of the lesion, the client's flaccidity may persist longer. For example, if the location is the primary motor cortex, the client may have residual hypotonicity presenting with little to no resistance to passive movement, diminished stretch reflexes, and hyperextensibility of involved joints.

DTR Scale

0 Absent, no response—always abnormal
1+ A trace response or needs reinforcement to achieve
2+ A brisk response, normal
3+ Hyperactive without clonus, may or may not be normal
4+ Hyperactive with unsustained clonus, abnormal
5+ Hyperactive with sustained clonus, always abnormal

DTRs are normal if they are 1+, 2+, or 3+ unless they are asymmetric or there is a dramatic difference between the arms and the legs.

There are specific synergistic patterns that occur following a CVA in both the upper and lower extremities. The flexor synergistic pattern consists of the upper extremity being positioned in scapular retraction, elevation, or hyperextension, shoulder abduction and external rotation, elbow flexion, forearm supination, and wrist and finger flexion. The lower extremity components of the flexor synergistic pattern consist of hip flexion, abduction, external rotation, knee flexion, ankle dorsiflexion and inversion, and toe dorsiflexion. The extensor synergistic pattern of the upper extremity consists of scapular protraction, shoulder adduction and internal rotation, elbow extension, forearm pronation, and wrist and finger flexion. The lower extremity components of the extensor synergy pattern consists of hip extension, adduction, and internal rotation, knee extension, ankle plantar flexion and inversion, and toe plantar flexion (O'Sullivan et al. 2014).

Brunnstrom Stages of Motor Recovery

Motor recovery is one of the most important treatment goals for clients and physical therapists post stroke. A valid, reliable, and responsive measure assessing post-stroke motor function is essential for appropriate clinical decision-making, treatment

planning, and research (e.g., outcome studies). The Brunnstrom recovery stages (BRS) is a short and easily administered measure for assessing motor function. The BRS contains three items for the arm (BRS-A), the hand (BRS-H), and the leg (BRS-L), all of which are rated on a six-level Likert-type scale. These items are usually used individually to describe the motor function of the hemiparetic arm, hand, and leg (Langhorne, Coupar, & Pollock, 2009). Signe Brunnstrom theorizes that reflexes should be used to elicit movement when none exist (normal developmental sequence), and proprioceptive and extroceptive stimuli also can be used therapeutically to evoke desired motion or tonal changes. Brunnstrom indicated that the restoration of motor function of individuals with hemiplegia occur in an almost standardized fashion. Regardless of the severity, sequential recovery stages are characterized by the presence of spasticity and synergistic patterns. It should also be noted that it is possible for limbs to present in different stages of motor recovery at the same time. The degree and speed to which an individual proceeds through these stages is extremely variable and it is possible to skip stages and/or plateau at a certain stage. It is vital for physical therapists to be able to assess independent limb stages in order to determine the most effective treatment during that specific stage (Brunnstrom, 1966).

Stage 1: Flaccidity (immediately after the onset)

No movement in the affected muscles, active movement of the affected muscles is not possible.

Stage 2: Spasticity and basic synergistic patterns are present

The basic limb synergies or some of their components appear as weak associated reactions or voluntary attempts of movement.

Stage 3: Spasticity increases

Basic limb synergies or some of their components are voluntary performed and spasticity peaks.

Stage 4: Spasticity decreases

Significant motor control emerges and an increasing number of movement combinations outside of basic limb synergies become possible, some abnormal movement is still present, spasticity is significantly decreased.

Stage 5: Spasticity continues to decrease

Complex movement, motor control and coordination improve, individual joint movements are well under control although increased concentration may be required.

Stage 6: Coordination reappears

Spasticity is no longer present and isolated coordinated joint movements are possible and appear normal or near normal.

Bed Mobility and Transfers

When evaluating bed mobility, the client's ability to go from supine to sit, sit to supine, scoot as well as roll left and right are the main focus of assessment. When assessing and helping clients relearn bed mobility, it is important to remember that there are multiple ways to perform each task. Rolling can be performed either non-segmentally, as a log roll, or segmentally initiating from the upper or lower extremities. Most adults with impairments resulting from an ABI will utilize a non-segmental technique, with no trunk rotation, in moving from supine to side-lying (Fell, Lunnen, & Rauk, 2018). In going from supine to sit, two common techniques are often seen. The first technique includes log rolling to side-lying and then pushing up from the lower arm while simultaneously swinging the lower extremities over the edge. The second technique begins in supine, the client then props up both forearms or extended arms while flexing the trunk, followed by a pivot of the lower extremities over the edge of the bed to a sitting position.

A transfer is any movement from one surface or location to another. When documenting a transfer, it is important to note the level of assistance required, any assistive devices used, and the surfaces the transfer was performed to and from. Some more commonly seen transfers include variants of sit to stand, stand to sit; including stand pivot, and squat pivots, floor transfers; including floor to stand and floor to wheelchair, car transfers and transfers in bed mobility. Any variant in a sit-to-stand transfer can be performed from a standard chair, wheelchair, or bed/plinth amongst other seating surfaces. It is important to assess and document the surface as the firmness of the surface can affect the client's ability to perform the task. While performing transfers, the client may require the use of an assistive device for balance upon immediate standing. Such devices include variants of a cane, crutch, or walker. Other adaptive tools such as a slide board or bed rails may also be utilized depending on the client's impairments.

In performing sit-to-stand transfers, things to consider prior to initiating the movement include seat height, seat firmness, and decision to use arm rest or not. Other preparatory actions include optimal trunk alignment, foot positioning, and assistive devices that may be needed once upright. Sit to stands can be broken down into typical movement sequences. The first phase is a flexion momentum, where forward lean of the trunk is attained. The second phase is a momentum transfer, where weight shifts help to produce a lift off from the seating surface. The third phase is extension, where the body begins to straighten and elongate. The final phase is stabilization, where once the body is elongated, stability is required to maintain the vertical position (Fell et al., 2018). Stand to sit is performed through eccentric muscle use, where knees flex, to lower to the chair, and hips flex, to counterbalance a posterior shift of center of mass.

As with all other training of movement, the therapist will provide variant inputs of manual cueing, visual cueing, and verbal cueing to increase the client's ability to successfully complete the task. Success in performing a sit to stand from one seating surface will not necessarily mean success and safety in performance on a different seating surface. When working with a client, if deemed appropriate, seating surface,

chair height, use of arms should be varied to allow increased learning. As the client's ability to complete these transfers improves, the transfer can be made more difficult by adding dual tasks, such as transferring with a cup of water or by making the task a variant of the sit-to-stand transfer such as a stand-squat or stand-pivot transfer. Of importance to note in performing stand-pivot transfers, it is often easier for the client to perform this transfer toward the stronger side.

Knowing a client's ability to perform transfers and bed mobility can help the clinician determine appropriate goals. Oftentimes the client will require family/caregiver assistance. Safety and mechanics in a transfer are important to teach in caregiver training so as to limit injury to the client and anyone assisting the transfer. Given below is a guideline as to what each level of assistance entails.

Levels of Physical Assistance (Giles, 2015; Pierson & Fairchild, 2008)

Independent: *client does not require any physical supervision or assistance from another person to consistently perform the activity safely in an acceptable time frame.*

Modified independent: *client uses adaptive equipment or assistive device to complete the task.*

Supervision: *client requires a therapist to observe throughout the completion of the task, client may require verbal cueing, directions, or instructions from person positioned close by, but not touching.*

Contact guard: *client requires the therapist to maintain contact in order to complete the task.*

Minimal assistance: *client requires 25% assist from the therapist to complete the task.*

Moderate ssistance: *client requires 50% assist from the therapist to complete the task.*

Maximal assistance: *client requires 75% assist from the therapist to complete the task.*

Dependent: *client is unable to participate and the therapist must provide all the effort to perform the task. Multiple people may be required to perform fully dependent transfers.*

Car Transfers

Car transfers are similar to sit-to-stand transfers and stand-pivot transfers. Before performing car transfers with the client's automobile, components of the transfer can be mocked in the clinic and practiced by the client as well as the client's caregiver. Practice in the clinic will insure improved safety when the transfer

progresses to an automobile. When preparing for the car transfer, it is important to leave plenty of room between the car and curb or edge of driveway. If a client is transferring from the wheelchair, it is best to angle the wheelchair in 45–90°, as close to the open passenger seat as possible. The assisting person or therapist should be between the client and car door. At this time, brakes should be locked and all obstacles of arm rests or leg rests removed. The car seat in most cases is best utilized in the fully pushed back and semi reclined position. The car seat position may vary from client to client, and the starting seat position may be different than the ending seat position. Once preparatory actions are complete, the sit-to-stand or stand-pivot transfer can be performed. The door should not be used to assist in stabilizing, as it is unstable itself. On descent into the car, it is important to emphasize the forward trunk lean in order to avoid bumping the client's head on the door frame. Once the client is seated, the assisting person or therapist can help to get the lower extremities within the car. In performing a transfer out of the car, the person's wheelchair or assistive device should be placed in the same position as described above. The transfer to rise from the car seat will apply the same techniques as a traditional sit to stand. Oftentimes a lower sedan will be more difficult to rise from as compared to a van or SUV. On the contrary, a van or SUV may prove to be more challenging to get into in comparison to a sedan. Some tools that can assist in the car transfer include pivot discs, car handle assists, and the use of a plastic bag on fabric seats. Different modifications and adaptations such as ramps, hoists, and pivoting seats are available to make an automobile more accessible.

Balance

Balance can be looked at as a person's ability to maintain and control their center of gravity while moving variant distances from midline. The body's ability to maintain balance is determined upon integration of somatosensory, visual, and vestibular information within the central nervous system. To identify impairments in one or any of the above systems, balance testing is performed in environments of firm surface, compliant soft surfaces such as foam, uneven surfaces, or sloped surfaces, as well as with eyes open and eyes closed. Testing of balance occurs with a variety of elements not only to identify the system with impairment but the altered test positions and variations, mock the environmental factors, and obstacles that a person may encounter in daily life.

Balance assessments are performed from both a seated and standing position. Balance is further analyzed during supported and unsupported scenarios as well as during static and dynamic activities. A quick ordinal balance grade can be assigned for each of the above scenarios. The ordinal scale grades include poor, fair, and good. Although these ratings are subjective and are similar from clinic to clinic, variability does exist. In addition to an ordinal balance grading scale, many more objective standardized tests of balance exist including the Berg Balance Scale, Fregley–Graybiel Ataxia Test Battery, Fugl-Meyer Sensorimotor Assessment of

Balance Performance Battery, Functional Reach Test, Romberg test, Timed Get Up and Go Test, Tinetti Performance Oriented Mobility Assessment, and Clinical Test of Sensory Interaction in Balance. Each standardized test has a correlated cutoff score that is helpful in determining a client's risk of fall as well as helping to identify the areas of impairment.

In providing therapeutic intervention to balance impairment, one must first identify what systems are leading to the balance difficulties. Balance is determined upon multisystem inputs. Numerous studies have looked at each system's involvement into balance and have found that lower extremity weakness, restricted joint mobility, vestibular dysfunction, as well as cognitive and perceptual deficits all have the ability to alter balance and strategic responses to maintain upright within limits of stability (Fell et al., 2018). It is also important to consider the impact of medications on a person's steadiness on feet.

No one specific exercise or intervention has greater outcomes or is of more value when it comes to training for balance. Many therapeutic interventions and approaches to improving balance exist. The commonality is that the level of assistance is gradually decreased as the client improves their ability to safely negotiate and handle each task. Balance training can range from receiving perturbations, weight shifting, altered bases of support, throwing, catching, overhead activities, visual fixation, and head movements. All tasks can be applied in the appropriate positioning for the client whether it be seated, tall kneel, half kneel, bipedal standing, single leg stance, or during ambulation. As the person's balance improves, the therapist will decrease the amount of assistance and support that the client uses during each exercise. As the client is able to handle the environment without assistance, additional resistance or weight can be used to alter the client's response to the same previously mastered exercise.

Studies on alternate forms of balance training include mental imagery practice, tai-chi, treadmill training, and aquatic therapy. When traditional therapies are unable to yield desired results, it is worth considering what other approaches can be employed to help a client increase balance and work toward a functional improvement.

Formalizing a client-specific plan to improve balance should include objectives of education on safety and fall prevention, correcting identified impairments, teaching and helping the client gain compensatory strategies when appropriate, improving client confidence, returning to ADLs, and recreational activities (Fell et al., 2018).

Fall Recovery

Fall percentage increases with age regardless of impairment. This increased probability of a fall, impending health risk factors, and the inability to recover from a fall, necessitates practice for fall recovery. Fall recovery includes a sequence of transfers through multiple positions. Like other transfers, fall recovery can be performed in variant ways. A recovery to standing or sitting is easiest to perform if there is a chair or other stable object that the client can use for support. One recovery method that

can be trialed would be as follows: From the floor, clients are taught to roll to their side, then push up to side sitting with an extended arm. From there the client can place the other hand on the floor and push up into the quadruped position. Next the client would place a hand on the chair and work to a tall kneel position. From the tall kneel position, one leg will be brought forward into half kneel. Finally, the client will push up with both upper extremities into a standing position. Practicing fall recovery can be intimidating as it is sometimes difficult for the client to get down on the floor without actually falling. Reece and Simpson (1996) suggests a backward chaining method that breaks down the fall recovery into smaller components (Thomas, 2015). The components are simplified to begin and progressively build toward being supine on the floor. By the time the client is lying on the floor, the client has already practiced all steps to get up from this position multiple times.

Gait

A gait pattern is a complex series of muscle activations through what is known as stance and swing phase. Analyzing and assessing gait in the neurological population occurs on a regular basis. Being able to identify what is occurring during a client's gait cycle is helpful in determining the focus of treatment. Oftentimes in clients with ABI, it is not just one phase of gait being impacted or one muscle group that is not activating optimally.

Observational gait analysis is the observation of kinematic patterns of movement used for gait (Shumway-Cook & Woollacott, 2012). When a client's gait is impaired, it is important to begin assessment on the stance phase on the impaired side(s). The first thing that needs to occur during stance phase is postural control and supporting the body against the forces of gravity, followed by progression of the body forward through force generation. If a client does not have stability during stance phase, the preceding mobility and motion of impaired and non-impaired limbs will be altered. Every joint and motion will eventually be analyzed but a good starting point will be to look proximally. What is the client's ability to activate the core and postural extensors? Does the client's hip drop or rotate? Are the hips and shoulders aligned during weight shift into stance phase? Breaking down the assessment into the three planes of motion, sagittal, frontal, and transverse and working proximally to distally will help organize the assessment. Once stance phase has been analyzed, the swing phase that produces forward locomotion needs to be assessed. Though not often used in the clinic secondary to time constraints and complexity of forms, several standardized forms exist to help structure an observational gait analysis. These assessment forms include the Rancho Los Amigos gait analysis form, Gait Assessment Rating Scale, and Rivermead Visual Gait Assessment.

Several theories of force generation exist for a normal gait. The "active push-off' theory formulized that the energy generated by ankle plantar flexors is carried up the chain to the trunk to create forward progression. The controlled roll off theory also exists hypothesizing that forward locomotion is a controlled falling of the body

over the ankle by deceleration of ankle plantar flexors (Shumway-Cook & Woollacott, 2012). Regardless of the theories that exist, what is obvious is that when assessing a client with a neurological injury, what normally occurs during gait is no longer occurring.

Analysis of gait may be done with a variety of bracing intact. Such orthoses as ankle foot orthoses (AFO) or hip ankle foot orthoses (HKAFO) may be worn secondary to little or no muscle activation and decreased ability to volitionally produce movement. Taking into consideration some of the theories of gait described above, and any knowledge of a decreased ability to produce muscular activity, the therapist should consider optimizing and utilizing other muscle groups to achieve the desired phase of gait. For example, if the client demonstrates decreased ankle dorsiflexion to effectively clear the foot, the therapist can utilize and assess the hip flexors force production to provide an increased foot clearance during the pre-swing phase.

Gait may be altered not only by muscular weaknesses but by many other factors such as pain, vision, comorbid orthopedic issues or upper/lower motor neuron lesions. When analyzing gait and ambulation, it is important to assess the ability to perform the movement as well as the quality of that movement. Gait assessment can be impacted conversely where the client has the strength to produce normal walking patterns, but tone and spasticity may prevent the client from completing normal mechanics and produce spastic gait. Some pathological gait patterns that are seen in the neurological population are as follows (Stanford Medicine, n.d.; Delisa, 1998):

Hemiplegic gait

Cause: Pyramidal strength deficits
Characteristics: Arm-adduction with flexion at the shoulder, elbow, wrist, and fingers
Leg extended with plantar flexion of the foot and toes. Leg advances in a semi-circle (circumduction) secondary to foot drop and hypertonia of extensors.

Neuropathic gait (steppage gait, Equine gait)

Cause: Weak or paralyzed dorsiflexors (foot drop)
Characteristics: Upon transitioning from swing-to-stance phase, the forefoot cannot be lifted. The advancing leg is lifted higher to clear the foot. Upon contacting the floor, the foot may audibly slap the ground secondary to lack of eccentric dorsiflexors.

Trendelenburg gait

Cause: Hip abductor weakness
Characteristics: Uncompensated: In stance phase of the affected side, there will be ipsilateral lateral pelvic protrusion, there will be a drop of the pelvis on the unaffected side greater than 5°
Compensation: Lateral trunk lurch over the affected hip during stance, keeping the center of gravity over the hip.

Ataxic gait

Cause: Injury to cerebellum and sensory deficits in lower limbs
Characteristics: Unsteady, uncoordinated with exaggerated movements and variable leg placements.

Parkinsonian gait

Cause: Parkinson's disease and disorders of the basal ganglia
Characteristics: Bradykinesia, tremor and rigidity may be present

Client is often flexed forward with small and shuffled steps. Initiation of movement may be challenging, may become stuck and unable to move forward (freezing of gait). Involuntary increase in cadence may occur (festination).

Choreiform gait

Cause: Dystonia, chorea, athetosis
Characteristics: Irregular, jerky, and involuntary movements. Walking may accentuate the movement disorder.

To be successful in analyzing and assessing gait in clients with ABI, it is important to have a sound understanding of typical gait sequencing and mechanics. This will assist clinicians in choosing the most appropriate assistive device and orthotic to allow for a more energy efficient and improved gait pattern.

Orthotics are commonly seen in the neurological population. Orthotics can be custom fabricated or "off the shelf." The most common types of orthoses seen are ankle foot orthoses (AFO). There are variations of the AFO including the solid AFO, posterior leaf spring AFO, articulating AFO with stops or assists, as well as floor reaction AFOs. Depending on the AFO chosen, the ankle will receive support medially and laterally, as well as possible modifications for increased dorsiflexion or limiting amounts of plantar flexion. The knee ankle foot orthoses (KAFO) encompasses the entire lower extremity and has the ability to lock and unlock at the knee joint for standing and seated activities. The hip knee ankle foot orthosis (HKAFO) includes another hinge at the hip with the ability to lock and unlock. There are clear benefits of orthoses, such as improved alignment, increased stability, as well as preventing contracture or deformity (Fell et al., 2018). Despite the benefits of the orthoses, time may be spent out of the orthoses. Training without the orthoses, when appropriate, allows the client increased opportunity to strengthen weak muscles as well as encourage motor learning to limit dependence on the brace. A physical therapist can help the client and family in determining an appropriate wearing schedule of the orthosis, not only to check for irritation and skin breakdown but also to promote strengthening out of the brace.

Assistive devices (AD), much like orthoses, are external tools that can help promote increased alignment, stability, mobility, and improve client confidence and safety in the home and community. Common assistive devices include the single axis cane (SAC), hemi walker, quad cane (narrow base or wide base), crutches (axillary or loftstrand), rollator, and walker (standard or rolling). Choosing the correct assistive device requires assessment of the client's strength, balance, and

cognitive function. Determining a client's cognitive level is vital to appropriate AD selection as some devices require more attention to sequence and placement than others. Collaboration with occupational therapists and speech therapists will allow for a better insight into the client's cognitive status. A narrow base quad cane may provide the appropriate support and stability to a client based on physical impairment, but the required sequencing of the cane can prove to be too challenging cognitively and ultimately decrease safety. In this case, the client might be more suited to use a rolling walker. Once the AD is chosen, the therapist will help measure and set the AD to the appropriate height. It is not uncommon for a client to progress from one AD to another. Multiple assistive devices may be concurrently recommended for clients depending on environmental factors. For example, a client may be ambulating with a rolling walker in the home but may use a quad cane and handrail on the staircase to access the bedroom. In another example, a client may ambulate with a rollator in the community but utilizes a SAC in the home. The client is not necessarily relying on the rollator for the large base of support but utilizing it for the seat during longer commuting distances.

Gait training should begin on firm, flat surfaces and gradually build in difficulty. A client may begin gait training with pre-gait activities such as plantigrade position. Thereafter, weightshifts and external perturbations can be added into the treatment plan. As the person improves movement and ability to negotiate across the current firm, flat surface, parameters such as distance, ambient features, attentional demands, obstacle avoidance, terrain, physical load, and postural transitions can all be added to vary the complexity of ambulation (Fell et al., 2018). With changing the environment and confines of the task, the client will begin to learn how to ambulate with both anticipatory and reactive postural controls. Daily living and negotiating the environment indoors and outdoors requires reacting to unpredictable factors. It is important for clients to be exposed to these environmental variabilities while in physical therapy to insure safety when encountered outside of the clinic. Some examples on how to alter ambulation and gait training tasks include changing gait speed and direction on command, obstacle avoidance, negotiation of ramps, curbs, stairs, and uneven surfaces (grass, carpet, boardwalks, brick). Other ambulation modifiers can include external pushes, pulls, carrying objects of different weights and shapes, and cognitive dual tasks such as talking on the phone or having a conversation with the therapist (Fell et al., 2018).

Stair Negotiation

Stair negotiation is similar to ambulation, in that concentric and eccentric force productions as well as control over center of gravity and mass is required to successfully complete the task. Stair negotiation can be broken down into a stance and swing phase. When initially assessing stair negotiation, assess ascent followed by descent. Ascending stairs primarily requires concentric activations and is less tasking to clients than the eccentrically controlled descent. Normally ascending and descending the stairs would be performed in a reciprocal pattern with the ability to

control and stabilize through a changing center of mass without a handrail. It is not uncommon to find that clients with ABI will complete elevations in a step-to-step pattern while using handrail(s) and assistive devices. When ascending the stairs, the safest pattern is typically leading and stepping up with the stronger leg followed by the impaired leg. On the descent, the safest and easiest sequencing is typically leading with the impaired leg followed by the stronger leg, ultimately utilizing the eccentric control of the stronger lower extremity.

Stair negotiation is a goal for many clients. As discussed in the evaluation of stair negotiation, "up with the good, down with the bad" is the typical step pattern used in negotiating elevations when an impairment is present. As a client's strength and stability improves, the client will advance from a step to pattern to a step through, reciprocal pattern. Each pattern can be practiced with varying upper extremity support and use of assistive devices. As the client's ability to negotiate the stairs progresses, modifiers to a stair task can include changes in stair number, height, depth, terrain, as well as obstacle avoidance on stairs and addition of external factors like heavy grocery bags or other persons on stairs.

Activity Tolerance

After an acquired brain injury, a client's activity tolerance will frequently be impacted secondary to deconditioning. A client with an extended bed rest or new brain injury will rapidly encounter decreases in maximum oxygen consumption, cardiac output, and muscular strength.

When targeting activity tolerance as a main deficit, the therapist will design a treatment plan with the ultimate goal of increasing the client's heart rate to challenge the client's cardiovascular system and activity tolerance. The Borg Rating of Perceived Exertion Scale is a way for physical therapists to measure the intensity level of a physical activity by allowing a client to subjectively rate their exertion during exercise. It gives the therapist an idea of how hard the client feels he or she is working during a physical activity. According to the Center for Disease Control and Prevention, "There is a high correlation between a person's perceived exertion rating times 10 and the actual heart rate during physical activity, so a person's exertion rating may provide fairly good estimate of the actual heart rate during activity."(Borg, 1998) The Borg Rating of Perceived Exertion Scale is rated on a 6–20 point scale. The range of 6–11 represents light activity such as sitting or walking. The range of 12–16 represents somewhat moderate to difficult activity, like when exercising. The range of 17–20 represents very difficult activity with maximal exertion. One can monitor how the treatment plan is directly affecting clients based on their RPE and then alter the plan based on the rates given in order to attain goals. Measuring a client's ability to perform tasks for a determined length of time and gathering information on perceived rate of exertion can be used to track and prescribe exercise intensity independent of type of injury, age, gender, or modality of therapeutic exercise (Scherr et al., 2012). When activity tolerance is a goal for clients,

it is important to remember precautions of cardiopulmonary changes that can occur with exercise. Blood pressure will increase with exercise approximately 7–10 mmHg per MET of physical activity. Exercise is contraindicated in those with uncontrolled severe hypertension, BP ≥ 180/110 (Ghadieh & Saab, 2015).

Community Mobility

A client may be able to ambulate in a hallway or within the home, but this does not translate into a safe and successful ability to negotiate the community. Cognitive systems in locomotion greatly affect the client's ability to safety negotiate in the community. When ambulating in a familiar environment, successful negotiation of a new task or unexpected obstacle is often not demanded. Ambulation and postural balance are considered to be automatic but in new unfamiliar settings a client's cognition will have a determining effect on how safe and successful they can negotiate despite the automatic ambulation condition. In a study by Chen et al. (1996), the ability to negotiate obstacles while dividing attention was observed. It was found that obstacle contact was increased when subjects had divided attention. When negotiating in the community, new external perturbations from a crowded sidewalk or dual tasking of negotiating obstacles while determining when it is safe to cross a street proved to be more challenging. The community offers a variety of obstacles such as uneven pavements and terrains, variant curb heights, ramps, and escalators that are not re-creatable in a clinic setting.

One way to begin preparing for community negotiation is to assess the client's walking speed and walking distance. In a systematic review of distance requirements for community ambulation, it was found that the largest community sites were those of warehouses, super stores, and hardware stores with required ambulation distances of 600 ft up to 2222 ft. The lowest mean requirements of distance were areas at the front and back of the house, which required the ability to ambulate 52–62 ft. Sites considered essential for ambulation are physicians' offices, banks, drug stores, department stores, and grocery stores which have required ambulation distances of 65–1250 ft. The average distance in commercial crosswalks was found to be 32–88 ft and residential crosswalks between 32 and 42 ft. In being able to successfully cross the street, studies also suggested that clients must be able to maintain a certain speed (Salbach et al., 2014).

Walking speeds can be used as a predictor of a client's ability to negotiate in either the home or community. For individuals with a stroke, clients ambulating at <0.4 m/s are more likely to be household ambulators. At speeds of 0.4–0.8 m/s, clients are considered limited community ambulators, at >0.8 m/s community ambulators and at speeds of >1.2 m/s clients are considered to be normal community ambulators and safe to cross the street (Bowden, Balasubramanian, Behrman, & Kautz, 2008; Middleton, Fritz, & Lusardi, 2015). The suggested walking speeds and their correlating classifications can vary from country to country as it was determined that in densely populated areas, both required walking speed and distances to

negotiate the community increased, as compared to more rural areas. In the United States, the mean walking speed to cross a street was found to be 1.32 m/s (Salbach et al., 2014). The next step would be to assess a client's ability to negotiate uneven surfaces and obstacles during dynamic movement. Only by taking a client outside and observing negotiation in the community, will a therapist be able to truly assess the client's environmental awareness, safety, balance, cognition, endurance, and ability to safely negotiate in the community.

Home Assessment

An often overlooked but vital role of a PT is to perform a proper and comprehensive home assessment. The Joint Commission on Accreditation of Healthcare Organizations recognizes the importance of home assessments. Standard EC.1 from the Joint Commission's *Comprehensive Accreditation Manual for Home Care* states: "The organization also plans for physical safety in the client's environment to reduce the risk of injuries or threats to life and health within the individual's environment to the extent possible, the organization designs a method(s) to identify risk" (Joint Commission on Accreditation of Healthcare Organizations, 1996). Mechanical falls are common after stroke, with between 14% and 65% of stroke survivors experiencing falls while in hospital (Teasell, McRae, Foley, & Bharadwaj, 2002) and up to 73% of stroke survivors experiencing a fall in the first 6 months after discharge to home (Mackintosh, Goldie, & Hill, 2005). A major part of this home evaluation is determining what assistive devices and adaptive equipment are necessary and what modifications need to be done to allow for safe mobility in the home and decrease the risk of falls. PTs can use a variety of assessment tools to assist with improving safety in the home such as the Functional Environment Assessment (FAE) and the Safe at home method (Securing a Functional Environment with the Anemaet–Trotter Home Observation and Modification Evaluation).

Performing an in-depth home assessment allows for a customized plan to ensure a safer home environment while decreasing the risk of falls, decreasing dependence on caregivers, and giving clients more independence and efficiency with ADLs. Home assessments will also allow the PT to prescribe the most appropriate and safest home exercise plan based on the specific environmental set up of the client's home. The Centers for Disease Control (CDC) provides a home safety checklist that can easily be conducted by a PT or can be given to the client's family member in order to determine risk factors.

Family and Caregiver Education

Family and caregiver training is essential to carryover of safety skills learned during physical therapy sessions. A comprehensive PT treatment plan should include education, demonstration, and training of activities that will be performed on a daily

basis within the home in order to reduce compensatory movements, unsafe sequencing of transfers, ambulation or stair negotiation, as well as aiming to facilitate neuroplasticity with proper repetitive movements or exercises. Treating clients, family members, and caregivers as part of the multidisciplinary team can assist in the speed of recovery, decrease the risk of falls, and help to reestablish safe movement outside of the clinic. Communication with a client's support system is key in managing expectations of recovery. Emphasizing the importance of compliance with home exercise programs as well as the carryover of transfer techniques is crucial to decreasing overall caregiver assistance and minimizing risks of injury within the home.

Vestibular Rehabilitation Therapy (VRT)

Head trauma can directly damage the vestibular organ or vestibular nerve, the brainstem, and the visual and oculomotor pathways, leading to vestibular disorders (Allison, 1999). Vestibular testing of clients with head trauma shows that head trauma may damage the peripheral and central vestibular structures, simultaneously or separately. A recent study of client's status post-mild head trauma and subsequent vestibulopathy compared radiological findings and clinical assessment, focusing on reduction of cognitive functions, severity of symptoms, and time to recovery. They were shown to have significant axonal impairment irrespective of the prevailing peripheral vestibular symptoms. These findings support the hypothesis that post-traumatic vestibulopathy has a central axonal injury component (Alhilali, Yaeger, Collins, & Fakhran, 2014). Head trauma can cause the loss or reduction of function in a semi-circular canal which can lead to peripheral vestibular symptoms. This may be the result of the injury or also occur as a side effect of medication prescribed after head trauma (e.g., ototoxic aminoglycoside antibiotics).

Vestibular disorders can be due to organic, structural, or sub-structural damage localized at different vestibular levels, from the labyrinth to the cortical regions. They can also involve non-vestibular structures, such as neck proprioceptors, whose signals integrate with the vestibular afferentation, as well as humoral mechanisms. A thorough vestibular examination is key for establishing the location, pathogenesis, and severity of traumatic vestibular disorders. It is important to note that vestibular dysfunctions can cause people to adopt a more sedentary lifestyle in an effort to avoid dizziness symptoms, which then in turn results in decreased muscle strength/flexibility/endurance as well as increased balance deficits. The vestibular evaluation should include a comprehensive subjective interview of present symptoms including description of dizziness, duration, causes of symptoms, medications, significant past medical history, as well as subjective measures such as the Dizziness Handicap Inventory scale (DHI). The vestibular PT will then conduct a variety of observatory and clinically diagnostic tests, including postural control (i.e., eyes open and closed on varying surfaces), oculomotor tests (i.e., gaze stabilization and smooth pursuits), and vestibular positional test (i.e., Dix Hallpike and roll test). Diagnostic tools such as frenzel goggles may be used to help better

visualize ocular nystagmus and its direction to help determine the origin. Once the origin of the vestibulopathy is determined (peripheral vs. central vs. cervico-genic), the PT can then appropriately diagnose and treat the underlying dysfunction. The goal of VRT is to alleviate the primary and secondary problems caused by vestibular disorders by designing a customized exercise based program to reduce vertigo, dizziness, gaze instability, fall risk, and postural instability, while improving balance and functional mobility.

Peripheral

One of the most common peripheral vestibular dysfunctions is Benign Paroxysmal Positional Vertigo (BPPV) which is a sudden false sense of rotational movement triggered by specific head positions and is also one of the quicker dysfunctions to remedy. This occurs when the calcium carbonate crystals or otoconia dislodge and migrate to one or more of the three semicircular canals. The otoconia will interfere with the normal lymph fluid movement causing the inner ear to send false signals to the brain, which is perceived as a spinning sensation. After an in- depth evaluation of positional head changes utilizing tools such as videonystagmography (VNG) and concurrent observations of evoked nystagmus, the PT will determine which semicircular canal needs to be repositioned in order for the otoconia to be returned to the normal position. The testing for BPPV is extremely provocative as they are meant to recreate the spinning sensation and will commonly cause nausea and vomiting. The Epley Maneuver is commonly used by PTs to treat posterior canal BPPV (most common) in order to reposition the otoconia. Anterior canal and horizontal canal BPPV are less common but also have specific treatment maneuvers, such as the Rahko maneuver and the log roll maneuver, respectively. A follow-up visit with subjective reports and surveys (DHI) as well a Dix Hallpike retest with VNG will be performed.

Other common causes of peripheral vertigo are vestibular neuronitis: an infection and inflammation of the vestibular nerve and Meniere's Disease: a build-up of fluid within the labyrinth caused by possible conditions such as poor drainage, blows to the head, viral infections, and autoimmune responses. VRT may be appropriate for these conditions but referrals to appropriate ENT/audiologist are also necessary in these cases.

Central

Central vestibular dysfunctions are caused by disease or injury to the brain by conditions such as head injuries, brain tumors, strokes, illness or infections, migraines, vascular disorders and demyelinating diseases. The differentiating factor in central vs. peripheral vertigo is the duration of nystagmus and symptoms. Central origins of vertigo will cause nystagmus and vertigo for prolonged periods of time, will come on without warning or without a noticeable pattern, and will not subside when asked

to fixate on an object. Central vertigo will also clinically demonstrate lateropulsion, occulomotor abnormalities, and orientation to midline postural deficits or changes in subjective visual vertical. During the vestibular evaluation, the PT may notice dysfunctional oculomotor control such as retinal slipping with smooth pursuits and saccadic movements, gaze instability, VOR cancellation, or direction changing nystagmus. Trauma-induced dizziness can cause a decreased reabsorption of endolymph into the endolymphatic sacs resulting in swollen endolymph chambers and membrane perforation, producing symptoms of vertigo and hearing loss. Blows to the head can also cause temporal/parietal bone fractures with concurrent damage to the vestibular and cochlear nerve resulting in vestibular loss and/or hypofunction. Post-concussive syndrome (PCS) can also present with persistent symptoms of dizziness, but careful consideration and attention must be exercised in order to appropriately assess other contributing conditions such as anxiety and depression before determining the most appropriate treatment plan. The general prognosis for treatment of central vestibular lesions is longer and less effective than that for peripheral lesions. Habituation exercises aim at reducing the dizziness through repeated exposure to specific visual stimuli or movements that provoke the client's dizziness or, in other words, aims to increase the threshold tolerance of a specific stimuli by repeatedly and incrementally evoking the symptoms. Visual–vestibular interaction exercises are aimed at the remaining functioning vestibular system in an effort to promote adaptation to certain stimuli such as head movement. Substitution exercises are aimed at utilizing other sensory stimuli such as somatosensory input in order to compensate for the absent or hypofunctioning vestibular system.

Cervico-Genic

The pathophysiology of cervico-genic dizziness can arise from three possible different origins. (1) Posterior cervical sympathetic syndrome where an underlying cervical dysfunction such as arthritis can cause irritation of the sympathetic vertebral plexus and therefore constrict the internal auditory artery resulting in decreased perfusion of the labyrinth. (2) vertebrobasilar insufficiency, where the vertebral arteries are occluded, is usually a result of a variety of primary cervical issues including osteoarthritic spurs, occipitoatlantal instability, and atherosclerotic vascular disease. (3) Altered or inaccurate proprioceptive input from sensitized sensory receptors in the cervical spine can create a sensory mismatch between vestibular and proprioceptive inputs resulting in dizziness and altered postural control.

Case Study

JR is a 55-year-old male who was struck by a vehicle and diagnosed with left frontal intraparenchymal hemorrhage, anterior and posterior pelvic fractures, right sacral fracture, multiple rib fractures and L5 and T7 spinal fractures. JR underwent an

open reduction internal fixation of the pelvis 3 days after the initial injury and was non-weight bearing (NWB) on the right lower extremity (LE) for the next 6 weeks. JR also had a tracheostomy and PEG tube placement the following day. All other injuries were managed non-operatively.

After completion of inpatient acute rehabilitation, JR was recommended for outpatient physical therapy services and was evaluated 58 days post accident. JR presented with a thoraco-lumbo-sacral orthosis (TLSO), axillary crutches, and right LE NWB status. JR did not have any reports of dizziness or lightheadedness and subjectively reported 4/10 pain in right LE and pelvis and a feeling of numbness throughout the right LE. Objectively the client demonstrated within functional LE A/PROM and muscular strength as well as increased need of assistance for all transfers, ambulation, and stair negotiation. Due to the weight-bearing status, PT was unable to perform standardized tests to accurately gauge JR's balance, gait speed, and activity tolerance. Approximately 2 weeks after the IE, the WB status was changed to weight bearing as tolerated (WBAT) and the TLSO was discontinued. PT assessed the 10-meter-walk test with results of 1.11 m/s for self-selected walking speeds and 1.32 m/s for fast velocity, the DGI 22/24 and also noted mild antalgic gait deviations due to pelvic pain. JR's treatment plan consisted of 8 weeks of neuromuscular re-education, balance training, gait training, therapeutic exercise including core stabilization, pelvic control, UE/LE strengthening, and overall endurance training. Seven weeks into JR's treatment plan and preparation for discharge, JR reported significant c/o dizziness when rolling in bed and bending down. PT performed a vestibular evaluation including the Dix–Hall Pike maneuver with Frenzel goggles in order to better visualize nystagmus. Upon testing to the left, it was found that JR had torsional ocular nystagmus with a latency of 18 s, as well observable clinical signs of dizziness and subjective reports of spinning during the testing. It was concluded that JR had left posterior canal BPPV. JR was then treated on the same day in the clinic with canalith repositioning techniques and educated on self-maneuvers, HEP, and importance of avoiding extreme heads positions for the next 48 h. JR presented back to PT 4 days later with no subjective reports of dizziness or spinning. Diagnostic testing with Frenzel goggles was performed again and no ocular nystagmus was noted. JR and family were educated on the possibility of re-occurrence in the future and discharged home with recommendations to return to PLOF.

Concussion Return to Play Protocol

The American Academy of Neurology in 2013 defined concussion as a "clinical syndrome of biomechanically induced alteration of brain function typically affecting memory and orientation which may involve loss of consciousness" (Giza et al., 2013). According to the Consensus Statement of Concussion in Sport Zurich November 2012, a concussion may be caused by a direct blow to the head, neck, face, or elsewhere on the body with an impulsive force transmitted to the head. A concussion typically results in the rapid onset of short-lived impairment or

neurological function that resolves spontaneously and may evolve over a number of minutes to hours. A concussion may result in neuropathological changes, but the acute clinical symptoms largely reflect a functional disturbance rather than a structural injury, and as such, no abnormality is seen on standard structural neuroimaging studies. Resolution of the clinical and cognitive symptoms typically follow a sequential course; however, it is important to note that in some cases symptoms may be prolonged (Mccrory et al., 2013).

The role of a PT in the management and treatment of a client diagnosed with a concussion is dependent on the setting of which the PT works. A school PT will be more readily available for an on-field or on-site evaluation of the athlete. The on-site evaluation should include a Sport Concussion Assessment Tool (SCAT3). The SCAT3 is comprised of eight categories of assessment: Glascow Coma Scale, Maddocks score, symptom evaluation, cognitive assessment, neck and limb evaluation, balance examination, coordination examination, and a Standardized Assessment of Concussion (SAC) delayed recall. Any player suspected of having a concussion should be immediately removed from the field of play, medically assessed, monitored for deterioration, and should not return to participation on the same day of injury. Concussion signs and symptoms can include headache, dizziness, double/blurry vision, photophobia, phonophobia, concentration and memory problems, confusion, nausea, and vomiting. It is also important for the PT to recognize signs and symptoms that require immediate medical attention such as convulsions, loss of consciousness, slurred speech, weakness, one pupil larger than the other, and inability to recognize people or places.

Another role that a PT can play in concussion treatment and management is to get the athlete back to his/her sport by taking the student athlete through the return to play (RTP) protocol. Being able to manage and guide the safe recovery of individuals who participate in competitive or recreational activities is crucial in preventing second impact syndrome and prolonged recoveries. The RTP is a gradual five-step protocol to guide in the safe return to sport and is only to be initiated after the individual is symptom free and has completed physical and cognitive rest for a minimum of 24 h. The PT will utilize clinical judgment and subjective reports to progress the individual through the five stages: Light aerobic activity, Moderate activity, Heavy non-contact activity, Practice and full contact,. Return to competition. An athlete will not be able to progress to the next stage if any signs or symptoms appear during or within the 24 h following the current stage. If an athlete does experience signs or symptoms, then they will be required to have additional cognitive and physical rest without symptoms for a minimum of 24 h before they return to the previously asymptomatic stage. If symptoms worsen or do not go away in 10–14 days or if there is a history of multiple concussions or risk factors for prolonged recovery, the treating therapist should refer the client to an appropriate specialist. The CDC's HEADS UP initiative and the National Federation of State High School Associations (NFHS) have developed training programs for athletes, parents, coaches, and medical professionals in order to educate and train individuals on the impact, recognition, and management protocols of a suspected concussed athlete and to the safe return to sport. Below is a description of the Graduated Return to Play Protocol.

Stage 1: No activity. Short duration of complete physical and cognitive rest allowing for recovery.
Stage 2: Light aerobic exercise with no more than 70% HR max. No resistance training.
Stage 3: Sport-specific exercise with no head impact drills.
Stage 4: Non-contact, more complex training drills and progressive resistance training.
Stage 5: Full contact practice following medical clearance.
Stage 6: Return to play.

Mccrory et. al. (2009).

Common Findings in ABI

Pyramidal Strength Deficits and Hemiparesis

Upper motor neuron lesions or pyramidal insufficiency commonly results in a pattern of muscle weakness called pyramidal weakness. Pyramidal weakness refers to the resulting weakness of extensors (shoulder extension, elbow extension, and wrist extension) in the UE and flexors (hip flexion, knee flexion, and dorsiflexion) in the LE after a CVA or TBI. These weakness patterns are opposite of the synergistic patterns for the respective limbs. As we have already discussed, reducing and inhibiting synergies are vital goals for rehabilitation, but developing a comprehensive plan of care should include strengthening of the weak patterns out of the synergy.

Hemiparesis (HP) is weakness or inability to move that is restricted to one side of the body or face. HP is found in eight out of ten clients post stroke and can be a major contributing factor in gait abnormalities, loss of balance, muscle fatigue, coordination deficits, such as ataxia and apraxia and an overall decreased ability to perform all functional activities (National Stroke Association, 2018).

Coordination Deficits

Ataxia is described as a lack of muscle control or coordination due to cerebral and cerebellar infarcts or injury, which causes difficulties in regulating the force, range, direction, velocity, and rhythm of muscle contractions (Fredericks & Saladin, 1996).

Apraxia is the inability to carry out learned, skilled motor acts despite preserved motor and sensory systems, coordination, comprehension, and cooperation. Clients with motor apraxia show temporal and spatial errors affecting timing, sequencing, amplitude, configuration, and limb position in space which should be taken into consideration when creating a customized plan of care (Heilman, 2010) (Heilmann). Both Ataxia and Apraxia can make the rehabilitation process difficult and frustrating for both the client and PT. Adding resistance or weight to limbs during functional training or exercises can assist in providing increased feedback and help improve the accuracy and control of movements.

Balance Deficits

Having impaired balance can have various functional implications to a person's daily life. A balance impairment may require increased time during transfers and ambulation, increased assistance from others, and can lead to a more sedentary lifestyle. When a balance impairment is present, it is not uncommon to find that motivation and confidence to move is decreased. A person with a balance deficit may have a fear of falling, which can further impede the person's mobility. Impairments in balance can lead to falls not only during ambulation but also while performing seated tasks. Sitting, scooting, weight shifting, standing, leaning, turning, and walking are all motions involved in daily tasks from bed mobility to tasks centered around grooming, feeding, and leisure activities and all can be affected by balance impairments.

Pusher Syndrome

According to O'Sullivan sixth edition, "ipsilateral pushing known as pusher syndrome is an unusual motor behavior characterized by active pushing with the stronger extremities toward the hemiparetic side with a lateral postural imbalance" (O'Sullivan et al., 2014). A common finding of Pusher Syndrome is falling toward the hemiparetic side due to an altered perception of the body's orientation. The client perceives that his or her body is in midline, when it is actually shifted toward to hemiparetic side. "Ipsilateral pushing occurs in about 10% of clients with acute stroke and results from strokes affecting the posterolateral thalamus" (O'Sullivan et al., 2014). When performing functional tasks in sitting and standing, the client is unaware of the malalignment and increased weight shift toward the hemiparetic side. This can lead to injury of the hemiparetic arm in sitting or when navigating around in a wheelchair. The client will also demonstrate difficulty transferring toward the unaffected side. When standing with a client with pusher syndrome, the client will be unaware of their increased weight shift onto their weaker side, resulting in an unsustainable force onto their hemiparetic lower extremity. The client will be at a higher risk of falls although they will present fearless. Therapists must be cautious with their tactile and manual cues to this population, as the client will strongly resist any passive weight shifts to midline. When ambulating with this population, the client will demonstrate decreased stance time of the strong lower extremity due to the inability to transfer weight toward that side. Ambulation with a hemi-walker or hemi-cane tends to amplify the weight shift toward the hemiparetic side. "Pedersen et al demonstrated that clients with ipsilateral pushing behavior have poorer rehabilitation outcomes with longer hospital stays and prolonged recovery times. They also have significantly lower functional scores on admission and discharge with increased levels of dependence at discharge" (O'Sullivan et al., 2014).

Decreased Motor Processing Speed

Common findings in coordination typically stem from lesions in the cerebellum, basal ganglia, and dorsal column medial lemniscal pathway. "As the cerebellum, basal ganglia, and dorsal column medial lemniscal pathway provide input to, and act together with, the cortex in the production of coordinated movement, lesions in any of these areas affect higher level processing and execution of coordinated motor responses" (O'Sullivan et al., 2014). There are specific coordination deficits that stem from a cerebellar pathology. Ataxia is a very common term used to describe impairments in motor function. "Ataxia may affect gait, posture, and patterns of movement and is linked to difficulty initiating movement, as well as errors in the rate, rhythm, and timing of responses" (O'Sullivan et al., 2014). Another common coordination deficit with cerebellar pathology is dysdiadochokinesia, which presents as difficulty performing rapid alternating movements. When a client is asked to rapidly alternate between pronation and supination, the movement presents as asymmetrical and uncoordinated. These deficits are more pronounced with increased speed. Another typical impairment is dysmetria, which is "an inability to judge the distance or range of motion of a movement" (O'Sullivan et al., 2014). A client may over or under shoot when reaching for an object due to difficulty judging the proper distance. Clients may also present with dyssynergia, which is described as "a movement performed in a sequence of component parts rather than as a single, smooth activity" (O'Sullivan et al., 2014). Clients demonstrating coordination deficits may also present with gait ataxia. Typically a client would ambulate with a large base of support, poor dynamic balance, uncoordinated arm swing, and irregular stepping pattern with an ataxic lower extremity exaggerated step. The step may turn into a scissoring step with unpredictable patterns. The gait patterns are usually irregular, staggering, and demonstrate a veering or swaying to one side when ambulating forward.

Sensory Deficits

Having intact sensation is imperative for a client to be able to successfully negotiate any environment. It is known that sensory deficits commonly impact client's independence in activities of daily living, safety, and motor function (Pumpa, Cahill, & Carey, 2015).

When blood supply to a specific part of the brain is occluded or hemorrhages, a pattern (*see below*) of impairment can be predicted (Giles, 2015).

Anterior Cerebral Artery—*Contralateral lower extremity sensory involvement.*
Middle Cerebral Artery—*Sensory loss of the face and upper extremity and lesser involvement of the lower extremity.*
Posterior Cerebral Artery—*Contralateral pain and temperature sensory loss.*

Most clients will recover at least partially from their somatosensory deficits, with most recovery being in the first 3–6 months following stroke (Kessner, Bingel, & Thomalla, 2016).

Visual Deficits

Visual deficits can affect a person's ability to negotiate within their environment and interpret visual feedback. Visual disturbances commonly seen in neurological injury include disorders in conjugate eye movements, diplopia, ptosis, as well as other visual field deficits such as heminopsias (Fell et al., 2018). A visual deficit can interfere with a person's ability to see fine details, see with clarity and take in any aspect of their external environment. (Please see Dr. Han's Chap. 6 for a more detailed look into visual impairment in the client with ABI.)

Common Standardized Tests

Dynamic Gait Index (DGI)

The DGI examines a client's ability to safely attend to certain tasks while ambulating and modifying balance in the presence of external demands (O'Sullivan et al., 2014). This test is used to determine if a client is at risk of falling. The DGI uses a 0–3 point scale (0 being severely impaired) to grade the performance on eight different tasks. The test is scored out of 24 points. A client with a score of 19 or below is placed in the fall risk category.

Balance Evaluation System Test (BESTest)

The BESTest allows PTs to differentiate between six systems that affect balance. The systems are biomechanical constraints, limits of stability, postural responses, anticipatory postural adjustments, sensory orientation, and dynamic balance during gait. The test has 36 items and is scored using a 4-point scale with the best possible score being 108 points.

High-Level Mobility Assessment Tool (HiMat)

The HiMat is utilized in the clinic to assess high-level mobility and balance activities typically aimed toward younger adults recovering from a TBI. The HiMat includes 13 tasks performed at the safest maximal speed. Each task is based on a 5-point scale with the maximal score being 54.

Five Times Sit To Stand (5×STS)

The 5×STS test is a standardized objective measure used to determine a person's functional lower limb muscle strength and safety with transitional movements. A cutoff score of 12 s is discriminatory between healthy, elderly, and chronic stroke subjects (Mong, Teo, & Ng, 2010).

10 Meter Walk Test (10MWT)

The 10 MWT is a standardized objective instrument that measures a person's gait speed in meters per second over a short distance at a preferred walking velocity or fast velocity. Gait velocity is a powerful indicator of function and prognosis after a stroke. A person's walking speed will have significant implications for community functioning and is predictive of important outcomes such as social participation, fall risks, activities of daily living, and community safety (Schmid et al., 2007). Gait velocity can be categorized into clinically meaningful functional ambulation cutoff scores or classes as previously discussed in the "Community Mobility" section.

6 Minute Walk Test (6MWT)

The 6MWT is a sub-maximal exercise used to assess aerobic capacity and endurance by objectively measuring the distance walked over a 6-min time period. Objective gait deviations and use of assistive device or orthotic should be noted and documented during test administration in order to provide an accurate comparison to baseline upon re-testing. An increase distance of 34.4 m can lead to increased social activities, increased feeling of independence, and increased engagement with the environment resulting in less isolated lifestyles (Fulk & Echternach, 2008).

Function In Sitting Test (FIST)

The FIST is a 14-item bedside test that assesses a client's static sitting as well as reactive, motor, and dynamic function. The test is scored on 0–4 ordinal scale (0 being totally dependent and 4 being independent) with the maximal score being 56 (Function in Sitting Test, n.d.).

Berg Balance Scale (BBS)

The BBS is a 14-item objective measure designed to assess static balance activities, transitional movements, and the risk of fall. The BBS is scored on a 0–4 scale based on the ability to perform a specific activity with a maximum score of 56. A score of less than 49 is associated with increased fall risk. In the elderly population, a score of <45 indicates that an individual may be at a greater risk of falling, a score <40 is associated with almost 100% fall risk. (Berg Balance Scale, n.d.)

Timed Up and Go (TUG)

The TUG test is a quick screening tool used to assess a person's level of mobility and balance. An established cutoff score of greater than 14 s for older stroke clients is predictive of increased fall risk. (Timed Up and Go, n.d.)

Specific Treatment Techniques

Focal/Segmental Excitatory and Inhibitory Vibration Therapy

Segmental vibration therapy is a technique used to target a specific muscle, muscle group, or tendon via a handheld vibrator. High frequency vibration is applied to a muscle or tendon in order to elicit a reflex response referred to as the tonic vibratory response (TVR). Imparting a vibratory stimulus to a muscle or tendon stimulates the primary muscle spindle endings or proprioceptive stretch receptors, specifically Ia tonic and phasic receptors. Ia afferent impulses are transmitted via alpha motor neurons, where they activate monosynaptic and polysynaptic reflex arcs, producing involuntary contractions in the vibrated muscle. Tension within the muscles will increase slowly and progressively for 30–60 s and then plateau for the duration of the vibratory stimulus. At cessation of the input, the contractibility of the muscle is enhanced for approximately 3 min. These afferent impulses also alter the excitability of the corticospinal pathway by modulating of intracortical inhibiting and facilitating inputs to the primary motor cortex, conveying axial and limb motor control. Physical therapists utilize this reflex to enhance agonistic muscle contraction in hypotonic muscles or to inhibit hypertonic/spastic agonists via reciprocal inhibition. A study conducted by Steyvers, Levin, Baelen, and Swinnen (2003) determined that peripheral sensory stimulation can induce lasting increases in corticospinal excitability in the absence of actual movements by demonstrating AVR, which is considered to be of cortical origin, induces a delayed facilitation of musculature that is antagonistic to the site of the directly activated Ia afferent.

A study by Paoloni et al. 2009, aimed to assess whether segmental muscle vibration (SMV) to ankle dorsiflexor muscles of chronic stroke clients can improve walking. The study demonstrated that that there was a moderate improvement in mean gait speed, normal-side swing velocity, bilateral stride length, and normal-side toe-off percentages. It also demonstrated that a significant increase in bilateral ankle dorsiflexion at heel contact was associated with an increased maximum ankle dorsiflexion and plantar flexion during the swing phase of the paretic side. Surface EMG revealed that during the swing phase, there was a significant increase in the activation of the tibialis anterior muscle of the paretic side in the post-treatment assessment of the SMV experimental group.

Proprioceptive Neuromuscular Facilitation

After an ABI, functional movement patterns can become dysfunctional due to disrupted or altered communication within the neuromuscular system as well as an imbalance of mobility and stability throughout the body. Proprioceptive Neuromuscular Facilitation (PNF) is a treatment technique that is meant to improve and enhance the neuromuscular systems effectiveness in coordinating movements based on principles of irradiation, reciprocal innervation, and inhibition (Voss, Ionta, & Myers, 1985). PNF incorporates gross synergistic diagonal/spiral movement patterns that are typically found in everyday tasks such as dressing, throwing, rolling, kicking, and even walking. PNF is designed to have the maximum possible resistance throughout the range of motion of these primitive patterns. A PT may utilize special techniques, synergistic patterns, hand placements, and cues to facilitate neuromuscular re-education, strengthening, and stretching of isolated limb movements or gross body movements.

Two common forms of PNF utilized by PTs in the post ABI population are rhythmic stabilization training (RST) and combination of isotonic exercises (COI). RST is primarily used when weakness is the main deficit, such as hemiplegia s/p CVA, and it aims to facilitate and/or elicit isometric co-contractions of antagonistic patterns. These isometric co-contractions can be repeatedly broken or elicited by having the PT remove or add varying degrees of manual resistance. COI is commonly used to evaluate and develop the ability to perform controlled purposeful movements where strength and range of motion are deficient. COI involves alternating concentric, eccentric, and isometric muscle contractions (Voss et al., 1985). A combination of RST and COI techniques may commonly be used in the post ABI population to reduce/inhibit spasticity to enhance neuro-muscular coordination of proximal and distal segments of the body and to re-educate specific movement patterns during functional activities. It is important as a PT to understand and determine the goal of the PNF session, the movement patterns to be facilitated, and the relation of that movement pattern to the dysfunctional daily activity in order to customize and maximize treatment plans.

Neuro-Sensory Taping

Another adjunctive treatment tool for PTs to utilize in the neuro-rehabilitation setting is neuro-sensory taping. Our dermis and epidermis have multiple types of cutaneous receptors that respond or alter perceptions of stretch, light touch, deep pressure, vibrations, pain, temperature, tissue displacement, and proprioception. Utilizing neuro-sensory taping for pain mitigation, decompression, and neurosensory input will have a useful role in the recovery process. Neuropathic pain can be severely debilitating and limiting to functional movements and mobility. Nocioceptors (pain receptors) share pathways in the CNS with movement and proprioception and will bombard the brain with the perception of pain in the absence of a non-noxious stimuli. Applying neuro-sensory tape as a non-noxious stimulus to cutaneous receptors can alter the pain gate theory and downregulate the perception of pain. Subjective reports of decreased pain can assist PTs in improving ROM, movement patterns, and enhance overall rehab potentials (Callaghan, Mckie, Richardson, & Oldham, 2012). Neuro-sensory taping can also be useful for edema control and improved circulation by creating a mechanical decompression lifting effect to skin and underlying tissues. The lifting effect can more easily allow exudates to be removed from areas of convolutions that compress lymphatic vessels. It also allows for ecchymosis to be cleared more efficiently while reducing swelling. A good understanding of fascial anatomy and the desired outcome is key when determining the application technique and location of tape in clients whose movement patterns may be impaired from increased pain or edema.

Electrical Stimulation

Neuromuscular electrical stimulation (NMES) is one treatment approach used in outpatient neurological rehab to address spasticity, weakness, decreased range of motion, swelling, and to facilitate functional movement. NMES targets innervated muscles and intact peripheral nerves to excite a muscle contraction and response. When observing the effects of NMES on healthy individuals, studies have shown that NMES and exercise both show increases in strength, with no differences between the two groups. When combining NMES and strengthening, there was no additional benefit than just using them in isolation. Additional studies have shown that an increase in the intensity of NMES has directly improved strength gains, with 50–80% of maximal volitional isometric contraction (MVIC) yielding the largest increases in strength. There is also evidence regarding NMES and preventing atrophy in individuals with orthopedic and neurological impairments. During the initial phases of rehab, the use of NMES resulted in greater strength gains compared to progressive/traditional exercise. When treating clients who present with difficulty activating a MVIC or present with pain, NMES can help increase strength when the client is unable to volitionally perform a contraction. It is important to consider the

proper intensity (50–80% of MVIC) when utilizing NMES to increase motor unit recruitment when the aim is to improve muscle strength. During exercise, skeletal muscle is recruited in a predictable pattern of smaller to larger units as the forces required are increased. NMES, on the other hand, recruits more of a randomized pattern of activation. Therapists must consider a higher level of fatigue due to not selectively recruiting motor units and therefore must provide a longer off-time for recovery. The higher the frequency, the more fatigue the client will present with.

NMES can be utilized to address spasticity in the neurologic client population by being applied to either the agonistic or antagonistic muscle. When stimulating the agonist, the electric stimulation works to fatigue the spastic muscle or provide recurrent inhibition by stimulating Renshaw cells that inhibit spastic motor units. The Renshaw cells are inhibitory cells in the spinal cord that carry inhibitory information to the same muscle to turn off the signal. When the muscle is stimulated, it sends a message to the Renshaw cells in the spinal cord that will stimulate inhibition of the same muscle. NMES can also stimulate the antagonist muscle to decrease spasticity by reciprocal inhibition of the spastic muscle. Antagonist stimulation sends sensory information to the spinal cord when it excites an inhibitory neuron that will then travel along an alpha motor neuron to the spastic muscle to turn it off. This will reciprocally inhibit the motor neurons of the spastic muscle. For example, a therapist can place NMES on the quads to promote a contraction that will inhibit the spastic hamstrings. When utilizing NMES, therapists must always first assess for any precautions and contraindications prior to integrating this treatment approach.

Contraindications and precautions to e-stim consist of cardiac conditions, danger to hemorrhage, pacemaker, over superficial metal implants, DVT, malignancy, open wounds, and systemic infections. Precautions to electric stimulation consist of allergies to gels, sensory loss, severe edema, obesity, geriatric and pediatric populations, and disoriented clients (Bellew et al., 2016).

Biofeedback

Electromyographic Biofeedback (EMGBFB) is an intervention that can assist in the recruitment of weaker muscles, improve timing of muscle activation, decrease muscle spasm, and promote relaxation (Fell et al., 2018). The EMG signal picked up from an activated muscle is turned into either a visual or audible signal which can be used to give feedback to the client regarding voluntary contractions. EMGBFB studies focus mainly on use with stroke clients. The literature finds the effectiveness of EMGBFB to be variable. A systematic review performed by Stanton, Ada, Dean, and Preston (2011) analyzed the effectiveness of biofeedback using a signal of position, force, or EMG, via visual, auditory, or tactile sense. The review found that biofeedback demonstrated greater improvements of lower limb activities in the short-term and long-term time frames as compared to usual therapy and placebos. Moreland et al. (Moreland & Thomson, 1994; Moreland, Thomson, & Fuoco, 1998) and Schleenbaker and Mainous (1993) found EMGBFB to be an

effective adjunct to improve lower extremity recovery. Improvements were seen in active ROM, strength, and gait function of the ankle. In contrast, use of EMGBFB is not significantly supported in upper extremity recovery (Khadilkar & Phillips, 2006) and improvements made are similar to those found in conventional therapy (Rayegani et al., 2014). When EMGBFB is used in combination with NMES it is called EMG-triggered NMES. EMG-triggered NMES requires a client to voluntarily reach a certain threshold in order to activate the electrical stimulation. EMG-triggered NMES has been found to be more effective in increasing muscle activation than EMGBFB alone (Cozean, Pease, & Hubbell, 1988).

Technology and Virtual Reality

The use of technology in investigative studies and rehabilitation continues to grow each day. Although studies including the use of technology for recovery are not as abundant as those on some of the more traditional therapy techniques, there is evidence and growing support for the use of technology in treatment. Technology in the rehab world ranges to include robotics (refer to Chap. 5), virtual reality, and computerized devices. New technology is being developed for a multitude of alternative evaluative and treatment options to improve client engagement and/or assist clinicians with assessments. All forms of technology are developed with the mindset and hope that an environment or device is being created to promote new safe learning opportunities.

Virtual reality (VR) is a form of rehabilitative technology that allows a client to interact with simulated, multisensory, multidimensional environments in real time (Fell et al., 2018). VR can incorporate visual, auditory, haptic, or sensory inputs. In a review by Luque-Moreno et al. (2015), VR use in clients with a stroke concluded that "VR interventions may have a positive effect on balance and gait recovery, especially when combined with conventional therapy" (Fell et al., 2018). VR improves client engagement and should be considered as an adjunct to a framework established in traditional therapy.

Referrals and Alternative Therapies

Throughout a client's course of neurological rehabilitation, as clinicians, we may encounter situations where it is necessary to refer a client to appropriate specialists. Our referral decision will depend on the clinical signs, symptoms, and primary complaints the client may be presenting with. For example, a prosthetic and orthotic referral for further assessment and evaluation of a client with drop foot may be appropriate to decrease the fall risk and/or risk of ankle injury and to maximize mobility and safety. Another example of an appropriate referral is to Neuro-Optometry or Neuro-Opthamology due to complaints of vision changes, visual field

cuts, blurriness, or diplopia. Referrals to physiatry s/p ABI are common specifically due to increased muscle tone and spasticity or reports of increased neuropathic pain. Physical therapists frequently make recommendations for follow-up appointments with a neurologist when signs and symptoms do not align with the current diagnosis or comorbidities.

There are many alternative options to standard physical therapy rehabilitation. Acupuncture is one alternative option to therapy and can also be used in conjunction with standard physical therapy. According to Mayo Clinic, Acupuncture is a traditional Chinese medicine and is a specialized technique for balancing a person's flow of energy through meridians in the body. Needles are inserted into specific points along the meridians to decrease pain, improve overall wellness and stress management depending on the desired treatment goal. Wong et al. assessed electrical acupuncture on hemiplegic stroke clients, observing the effects of electrical acupuncture in rehab for clients with hemiplegia 2 weeks after onset of CVA as opposed to standard therapy alone. Clients who received both acupuncture in conjunction with standard therapy had a shorter hospital stay and better neurological and functional outcomes when compared to the control group, with significant improvements in self-care and ambulation (Wong et al., 1999).

Another alternative treatment option is hippotherapy. Hippotherapy is a form of physical, occupational and speech therapy in which a therapist uses the characteristic movements of a horse to provide carefully graded motor and sensory input (Tuba and Hilmi, 2015). Improvements can be seen in the client's motor coordination, muscle tone, postural alignment, stiffness/flexibility, and strength. These improvements can often allow for changes in other body systems such as respiration, cognition, sensory processing, balance, and speech/language.

Yoga is an increasingly popular alternative or adjunct to standard physical therapy. According to the *Journal of Yoga & Physical Therapy*, "Yoga has been introduced in physical therapy interventions to benefit postural misalignment, muscule imbalances, range of motion problems, and strength development of different muscle groups" (Shetty, 2016). The use of proper breathing techniques can help improve clients with respiratory and cardiac disorders, as well as controlling pain.

PT as Interdisciplinary Team Members in Neuro-Rehabilitation

No two clients with a neurological injury are the same nor do they follow the same path of recovery. The diverse presentation of clients with an ABI requires the skills and services of numerous healthcare providers. Physical therapists are essential members of the interdisciplinary team providing care to the client with an ABI. Through collaboration and communication with other care providers, the physical therapist is able to obtain a well-rounded understanding of the client's injury, limitations, prognosis, and potential for rehabilitation. This collaboration enhances the physical therapist's ability to provide client-centered care for maximal function, with the ultimate goal of enhancing their physical abilities therefore allowing them a more independent lifestyle.

References

Alhilali, L. M., Yaeger, K., Collins, M., & Fakhran, S. (2014). Detection of central white matter injury underlying vestibulopathy after mild traumatic brain injury. *Radiology, 272*(1), 224–232. https://doi.org/10.1148/radiol.14132670

Allison, L. (1999). Imbalance following traumatic brain injury in adults. *Neurology Report, 23*(1), 13–18. https://doi.org/10.1097/01253086-199923010-00010

Bellew, J. W., Michlovitz, S. L., & Nolan, T. P., Jr. (2016). *Michlovitz's modalities for therapeutic intervention*. Philadelphia, PA: F.A. Davis Company.

Berg Balance Scale. (n.d.). Retrieved January 27, 2018, from https://www.sralab.org/rehabilitation-measures/berg-balance-scale#stroke

Bowden, M. G., Balasubramanian, C. K., Behrman, A. L., & Kautz, S. A. (2008). Validation of a speed-based classification system using quantitative measures of walking performance poststroke. *Neurorehabilitation and Neural Repair, 22*(6), 672–675. https://doi.org/10.1177/1545968308318837

Brunnstrom, S. (1966). Motor testing procedures in hemiplegia: Based on sequential recovery stages. *Physical Therapy, 46*(4), 357–375. https://doi.org/10.1093/ptj/46.4.357

Bunton, E. E., Pitney, W. A., Cappaert, T. A., & Kane, A. W. (1993). The role of limb torque, muscle action and proprioception during closed kinetic chain rehabilitation of the lower extremity. *Journal of Athletic Training, 28*(1), 10.

Callaghan, M. J., Mckie, S., Richardson, P., & Oldham, J. A. (2012). Effects of patellar taping on brain activity during knee joint proprioception tests using functional magnetic resonance imaging. *Physical Therapy, 92*(6), 821–830. https://doi.org/10.2522/ptj.20110209

Chen, H., Schultz, A. B., Ashton-Miller, J. A., Giordani, B., Alexander, N. B., & Guire, K. E. (1996). Stepping over obstacles: Dividing attention impairs performance of old more than young adults. *The Journals of Gerontology Series A: Biological Sciences and Medical Sciences, 51A*(3), M116–M122. https://doi.org/10.1093/gerona/51a.3.m116

Cozean, C., Pease, W., & Hubbell, S. (1988). Biofeedback and functional electric stimulation in stroke rehabilitation [Abstract]. *Archives of Physical Medicine and Rehabilitation, 69*(6), 401–405. Retrieved December 14, 2018, from https://www.ncbi.nlm.nih.gov/pubmed/3288172

DeLisa, J. A. (1998). *Gait analysis in the science of rehabilitation*. Washington, DC: Dept. of Veterans Affairs, Veterans Health Administration, Rehabilitation Research and Development Service, Scientific and Technical Publications Section.

Fell, D. W., Lunnen, K. Y., & Rauk, R. P. (2018). *Lifespan neurorehabilitation: A patient-centered approach from examination to interventions and outcomes*. Philadelphia, PA: F.A. Davis Company.

Fredericks, C. M., & Saladin, L. K. (1996). *Pathophysiology of the motor systems: Principles and clinical presentations*. Philadelphia, PA: F.A. Davis Company. ISBN 0-8036-0093-3.

Fulk, G. D., & Echternach, J. L. (2008). Test-retest reliability and minimal detectable change of gait speed in individuals undergoing rehabilitation after stroke. *Journal of Neurologic Physical Therapy, 32*(1), 8–13. https://doi.org/10.1097/npt0b013e31816593c0

Function in Sitting Test. (n.d.). Retrieved January 28, 2018, from https://www.sralab.org/rehabilitation-measures/function-sitting-test

Ghadieh, A. S., & Saab, B. (2015). Evidence for exercise training in the management of hypertension in adults. *Canadian Family Physician, 61*(3), 233–239.

Giles, S. M. (2015). *PTEXAM: The complete study guide*. Scarborough, ME: Scorebuilders.

Giza, C. C., Kutcher, J. S., Ashwal, S., Barth, J., Getchius, T. S., Gioia, G. A., … Zafonte, R. (2013). Summary of evidence-based guideline update: Evaluation and management of concussion in sports: Report of the Guideline Development Subcommittee of the American Academy of Neurology. *Neurology, 80*(24), 2250–2257. https://doi.org/10.1212/wnl.0b013e31828d57dd

Heilman, K. M. (2010). Apraxia. *Continuum, 16*, 86–108. https://doi.org/10.1212/01.con.0000368262.53662.08

Hemiparesis. (2015, November 17). Retrieved July 12, 2018, from http://www.stroke.org/we-can-help/survivors/stroke-recovery/post-stroke-conditions/physical/hemiparesis

Joint Commission on Accreditation of Healthcare Organizations. (1996). Environmental safety and equipment management. In: Comprehensive Accreditation Manual for Home Care. Oakbrook Terrace, IL: Joint Commission on Accreditation of Healthcare Organizations.

Kessner, S. S., Bingel, U., & Thomalla, G. (2016). Somatosensory deficits after stroke: A scoping review. *Topics in Stroke Rehabilitation, 23*(2), 136–146. https://doi.org/10.1080/10749357.2015.1116822

Khadilkar, A., & Phillips, K. (2006). Ottawa panel evidence-based clinical practice guidelines for post-stroke rehabilitation. *Topics in Stroke Rehabilitation, 13*(2), 1–269. https://doi.org/10.1310/3tkx-7xec-2dtg-xqkh

Langhorne, P., Coupar, F., & Pollock, A. (2009). Motor recovery after stroke: A systematic review. *The Lancet Neurology, 8*(8), 741–754. https://doi.org/10.1016/s1474-4422(09)70150-4

Luque-Moreno, C., Ferragut-Garcías, A., Rodríguez-Blanco, C., Heredia-Rizo, A. M., Oliva-Pascual-Vaca, J., Kiper, P., & Oliva-Pascual-Vaca, Á. (2015). A decade of progress using virtual reality for post-stroke lower extremity rehabilitation: Systematic review of the intervention methods. *BioMed Research International, 2015*, 1–7. https://doi.org/10.1155/2015/342529

Mackintosh, S. F., Goldie, P., & Hill, K. (2005). Falls incidence and factors associated with falling in older, community-dwelling, chronic stroke survivors (1 year after stroke) and matched controls. *Aging Clinical and Experimental Research, 17*(2), 74–81. https://doi.org/10.1007/bf03324577

MayoClinic (n.d.). Acupuncture. Retrieved June 4, 2018, from https://www.mayoclinic.org/tests-procedures/acupuncture/about/pac-20392763

Mccrory, P., Meeuwisse, W., Aubry, M., Cantu, B., Dvořák, J., Echemendia, R., … Turner, M. (2013). Consensus statement on Concussion in Sport—The 4th International Conference on Concussion in Sport held in Zurich, November 2012. *Physical Therapy in Sport, 14*(2), e1–e13. https://doi.org/10.1016/j.ptsp.2013.03.002

Mccrory, P., Meeuwisse, W., Johnston, K., Dvorak, J., Aubry, M., Molloy, M., & Cantu, R. (2009). Consensus Statement on Concussion in Sport—The Third International Conference on Concussion in Sport Held in Zurich, November 2008. *The Physician and Sportsmedicine, 37*(2), 141–159. https://doi.org/10.3810/psm.2009.06.1721

Middleton, A., Fritz, S. L., & Lusardi, M. (2015). Walking speed: The functional vital sign. *Journal of Aging and Physical Activity, 23*(2), 314–322. https://doi.org/10.1123/japa.23.2.314

Mong, Y., Teo, T. W., & Ng, S. S. (2010). 5-Repetition sit-to-stand test in subjects with chronic stroke: Reliability and validity. *Archives of Physical Medicine and Rehabilitation, 91*(3), 407–413. https://doi.org/10.1016/j.apmr.2009.10.030

Moreland, J., & Thomson, M. A. (1994). Efficacy of electromyographic biofeedback compared with conventional physical therapy for upper-extremity function in patients following stroke: A research overview and meta-analysis. *Physical Therapy, 74*(6), 534–543. https://doi.org/10.1093/ptj/74.6.534

Moreland, J. D., Thomson, M. A., & Fuoco, A. R. (1998). Electromyographic biofeedback to improve lower extremity function after stroke: A meta-analysis. *Archives of Physical Medicine and Rehabilitation, 79*(2), 134–140. https://doi.org/10.1016/s0003-9993(98)90289-1

O'Sullivan, S. B., Schmitz, T. J., & Fulk, G. (2014). *Physical rehabilitation*. Philadelphia, PA: F.A. Davis Company.

Paoloni, M., Mangone, M., Scettri, P., Procaccianti, R., Cometa, A., & Santilli, V. (2009). Segmental muscle vibration improves walking in chronic stroke patients with foot drop: A randomized controlled trial. *Neurorehabilitation and Neural Repair, 24*(3), 254–262. https://doi.org/10.1177/1545968309349940

Pierson, F. M., & Fairchild, S. L. (2008). *Principles & techniques of patient care* (4th ed.). St. Louis, MO: Saunders Elsevier.

Pumpa, L. U., Cahill, L. S., & Carey, L. M. (2015). Somatosensory assessment and treatment after stroke: An evidence-practice gap. *Australian Occupational Therapy Journal, 62*(2), 93–104. https://doi.org/10.1111/1440-1630.12170

Rayegani, S. M., Raeissadat, S. A., Sedighipour, L., Rezazadeh, I. M., Bahrami, M. H., Eliaspour, D., & Khosrawi, S. (2014). Effect of neurofeedback and electromyographic-biofeedback

therapy on improving hand function in stroke patients. *Topics in Stroke Rehabilitation, 21*(2), 137–151. https://doi.org/10.1310/tsr2102-137

Reece, A. C., & Simpson, J. M. (1996). Preparing older people to cope after a fall. *Physiotherapy, 82*(4), 227–235. https://doi.org/10.1016/s0031-9406(05)66877-0

Salbach, N. M., Obrien, K., Brooks, D., Irvin, E., Martino, R., Takhar, P., … Howe, J. (2014). Speed and distance requirements for community ambulation: A systematic review. *Archives of Physical Medicine and Rehabilitation, 95*(1), 117–128.e11. https://doi.org/10.1016/j.apmr.2013.06.017

Scherr, J., Wolfarth, B., Christle, J. W., Pressler, A., Wagenpfeil, S., & Halle, M. (2012). Associations between Borg's rating of perceived exertion and physiological measures of exercise intensity. *European Journal of Applied Physiology, 113*(1), 147–155. https://doi.org/10.1007/s00421-012-2421-x

Schleenbaker, R. E., & Mainous, A. G. (1993). Electromyographic biofeedback for neuromuscular reeducation in the hemiplegic stroke patient: A meta-analysis. *Archives of Physical Medicine and Rehabilitation, 74*(12), 1301–1304. https://doi.org/10.1016/0003-9993(93)90083-m

Schmid, A., Duncan, P. W., Studenski, S., Lai, S. M., Richards, L., Perera, S., & Wu, S. S. (2007). Improvements in speed-based gait classifications are meaningful. *Stroke, 38*(7), 2096.

Shetty A (2016) Yoga as Physical Therapy Intervention and Future Direction for Yoga Research. J Yoga Phys Ther 6:e122. doi:10.4172/2157-7595.1000e122

Shumway-Cook, A., & Woollacott, M. H. (2012). *Motor control: Translating research into clinical practice*. Philadelphia, PA: Wolters Kluwer Health, Lippincott Williams & Wilkins.

Stanford Medicine. (n.d.). *Gait abnormalities*. Retrieved July 12, 2018, from https://stanfordmedicine25.stanford.edu/the25/gait.html

Stanton, R., Ada, L., Dean, C. M., & Preston, E. (2011). Biofeedback improves activities of the lower limb after stroke: A systematic review. *Journal of Physiotherapy, 57*(3), 145–155. https://doi.org/10.1016/s1836-9553(11)70035-2

Steyvers, M., Levin, O., Baelen, M. V., & Swinnen, S. P. (2003). Corticospinal excitability changes following prolonged muscle tendon vibration. *Neuroreport, 14*(15), 2001–2004. https://doi.org/10.1097/00001756-200310270-00004

Teasell, R., McRae, M., Foley, N., & Bharadwaj, A. (2002). The incidence and consequences of falls in stroke patients during inpatient rehabilitation: Factors associated with high risk. *Archives of Physical Medicine and Rehabilitation, 83*, 329–333.

Thomas, B. (2015, September 5). *Geriatrics*. Retrieved July 12, 2018, from http://rehab-insider.advanceweb.com/teaching-fall-recovery-skills-to-the-elderly/

Timed Up and Go. (n.d.). Retrieved January 27, 2018, from https://www.sralab.org/rehabilitation-measures/timed-and-go

Tuba Tulay Koca, Hilmi Ataseven. (2015). What is Hippotherapy? The indications and effectiveness of Hippotherapy. North Clin Istanb.; 2(3): 247–252

Voss, D. E., Ionta, M. K., & Myers, B. J. (1985). *Proprioceptive neuromuscular facilitation patterns and techniques*. Philadelphia, PA: Harper & Row.

Wong, A. M., Su, T. Y., Tang, F. T., Cheng, P. T., & Liaw, M. Y. (1999). Clinical trial of electrical acupuncture on hemiplegic stroke patients. *American Journal of Physical Medicine & Rehabilitation, 78*(2), 117–122.

Chapter 9
Rehabilitation of Speech, Language, and Swallowing Disorders in Clients with Acquired Brain Injury

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Introduction

Speech-language pathologists work to prevent, assess, diagnose, and treat speech, language, social communication, cognitive communication, and swallowing disorders in children and adults (ASHA, 2018a). The speech-language pathology team is a dynamic component of the interdisciplinary neurorehabilitation team and is involved in many aspects of care. The ultimate goal of this discipline is restoration of communication, resolution of dysphagia, and improvement of cognitive linguistic skills. The speech-language pathologist (SLP) works collaboratively alongside the individual with an acquired brain injury (ABI), his/her family, and a multidisciplinary team to create a comprehensive rehabilitation plan of care aimed at improving functional gains, independence, and quality of life.

Of critical importance in the neurorehabilitation process is the restoration of the client's communication skills. Without a means of communicating even the most basic needs, safety, and well-being can be compromised. Also of vital importance to the client is the restoration of swallowing function. Dysphagia, or difficulty swallowing, can compromise both the health and quality of life of individuals following an ABI and must be addressed early on in rehabilitation. The SLP also plays a major role in the rehabilitation of cognitive skills, a focus area that continues to grow in this field.

It is essential that the SLPs be aware of evidence-based practice and best-practice recommendations when evaluating and treating clients with ABI. As this is a rapidly advancing field, it is critical to remain up-to-date on research related to: cognitive rehabilitation approaches, pragmatic interventions, language and motor speech disorders, dysphagia, bilingualism, and new technological developments (e.g., neuromuscular electrical stimulation, augmentative-alternative communication).

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Evaluation

The evaluation process is initiated by a referral from the physician. SLPs begin their assessment when first greeting the client and his/her significant others and obtaining a case history. The combination of case history, coupled with objective measures, will provide the foundation for a comprehensive evaluation. The case history includes obtaining information about social, educational, vocational, and medical history. During the clinical interview, it is essential for the clinician to develop a comprehensive language profile for the client. This language profile should contain the client's educational level, reading and writing abilities in each language, as well as information relating to the client's preferred occupational and social language use (Rimikis, Smiljanic, & Calandruccio, 2013). Review of medical records is essential, as many clients with ABI are not able to report their history or current status accurately. Interviewing family members is also important, as they can report observations regarding changes in communication which are not reflected in the records, and of which the client may be unaware. Additional information is obtained regarding native language, handedness, need for eyeglasses or hearing aids, orientation, and present diet, including any restrictions or alternative consistencies for solids and liquids. Finally, eliciting comments from clients related to their primary complaints provide information about their level of insight. The person's diagnosis, age, severity of impairment, and observations made during the interview will determine which objective tests are chosen for the assessment (Chapey, 2001).

An audiologic screening can help identify individuals who have hearing impairments which would interfere with their communication function. Pure tone frequencies are presented bilaterally via earphones at 125, 250, 500, 1000, 2000, 3000, 4000, and 8000 Hz (ASHA, 2018b). If the screening is failed, the individual can be referred for a comprehensive audiologic evaluation by an ear, nose, and throat (ENT) specialist or audiologist. Follow-up testing will determine the severity of the hearing loss and the need for aural rehabilitation.

An oral peripheral examination is an essential part of the evaluation following an acquired brain injury. Range of movement and strength of structures, including the tongue, lips, and jaw, as well as velopharyngeal function, need to be assessed. For clients with known or suspected dysphagia, a swallowing evaluation will also be performed.

There are a number of evaluation tools that have been developed for the assessment of language/communication and cognition in the neurologically impaired individual. These include the Apraxia Battery for Adults, Second Edition (Dabul, 2000); Boston Diagnostic Aphasia Examination-3 (Goodglass, Kaplan, & Barresi, 2001); Brief Test of Head Injury (Estabrooks & Hotz, 1991); Cognitive Linguistic Quick Test Plus (Estabrooks, 2017); Frenchay Dysarthria Assessment-2 (Enderby & Palmer, 2008); Porch Index of Communicative Ability (Porch, 2001); Ross Information Processing Assessment, Second Edition (Ross-Swain, 1996); Scales of Cognitive Ability for Traumatic Brain Injury (Adamovich & Henderson, 1992); and Western Aphasia Battery (Kertesz, 2007).

The results of the evaluation will lead to the formulation of the functional diagnosis, severity of the impairment, goals, and treatment plan. Communication deficits post-ABI can result from impairments in both the motor aspects of speech and/or the ability to use and understand language. The former includes dysarthria and apraxia. The latter includes various types of aphasia, cognitive impairments (e.g., memory, problem solving), and impairments in social communication (e.g., pragmatics). Additionally, swallowing skills will be addressed during the evaluation and a treatment plan will be established as necessary.

Cognitive Communication

Cognitive communication abilities are the thought processes that allow people to function successfully and interact meaningfully with others. Cognitive communication disorders include difficulties in listening, speaking, reading, writing, conversation, and social interaction that result from underlying cognitive impairments (attention, memory, organization, processing information, problem solving, and executive functions) (MacDonald, 2017). The goal of cognitive rehabilitation is to improve injury-related deficits in order to maximize safety, daily functioning, independence, and quality of life (Haskins et al., 2014).

Evaluating and treating cognitive communication disorders is within the scope of practice for the SLP, who is uniquely trained to detect and remediate these disorders (MacDonald, 2017). Some areas of cognitive communication include speed of processing, attention, memory, executive functions, pragmatics, problem solving, and reasoning. Collaboration and sharing evaluation results related to cognition with the neurorehabilitation team, most notably between the neuropsychologist and occupational therapist, helps to develop a comprehensive understanding of the client's language, cognitive, and functional impairments and forms a basis for the development of a holistic treatment plan. SLPs participate in the rehabilitation of cognitive deficits by assisting the client to develop compensatory techniques and strategies to interact more effectively with those around them and to function more independently. One example of compensatory technique training includes teaching clients to use external aids or devices (e.g., daily planners, cell phone reminders/calendars) to facilitate the ability to organize and remember important information. There is evidence to support such strategy training (Cicerone et al., 2005) and that strategy use can be beneficial to individuals many years post-brain injury (Haskins et al., 2014). Internal memory strategies can also be taught to clients functioning at a higher level. A few examples of internal memory strategies include association techniques, visual imagery, and organizational techniques (Haskins et al., 2014). Because there is an interdependent relationship between cognitive and communication skills, it is important for an SLP to address cognitive deficits as these difficulties can interfere with the ability to use strategies to improve deficits in other areas including motor speech, swallowing, or language.

Case Study

AG is a 36-year-old single female; status: post-brain tumor (meningioma) removal resulting in a functional impact on her communication and cognitive skills. She presented at evaluation with aphasia and cognitive linguistic deficits judged as severe. Deficits were characterized by decreased orientation skills, difficulty formulating complete sentences, decreased confrontational naming, word finding, short-term memory, and problem solving skills. AG had decreased awareness and insight into her deficits when she first arrived at our facility. She needed supervision and help communicating and had to move back home with her parents for a few months.

When she started outpatient programming, she attended speech therapy three times a week. She was seen individually as well as in a group for memory. With consistent therapy, AG began to demonstrate increased awareness and insight into her deficits and showed improved communication skills.

At the 4 month post-surgery mark, she was able to speak in complete sentences, name common and uncommon objects, and demonstrated improved word finding in conversation. She still demonstrated moderate difficulties with short-term memory skills. These skills would be essential for her to return to living independently and to return to work as a teacher. With the gains in language skills noted, treatment shifted toward improving cognitive skills and learning compensatory strategies.

At 6 months post-surgery AG had learned internal memory strategies including making associations, using repetition and visualizing information being presented to her. Additionally, she executed external memory strategies, such as use of technology to input reminders for medication management and paying bills. The calendar application (app) was helpful for AG to schedule social events and family birthdays. The notes app was beneficial for her to document information received at doctors' appointments, as well as tracking her shopping lists. The use of these external memory strategies greatly helped AG to follow through on assignments in speech therapy, track social events, and organize work-related information (such as lesson planning and deadlines).

At 8 months post-injury, AG was able to use these strategies consistently, effectively, and independently at home and then successfully at work when she returned full time.

Pragmatics

Pragmatics, or social communication, is defined as the verbal and non-verbal aspects of language related to social interaction. Deficits in pragmatics are typically associated with, but not, limited to lesions or damage to the right hemisphere or frontal lobes of the brain (Channon & Watts, 2003; Dardier et al., 2011). Components of pragmatics include topic initiation and maintenance, inhibition, attention, eye contact, tone and prosody, and comprehension of non-literal language (e.g., humor,

sarcasm, proverbs) (Parola et al., 2016). Clients with impaired pragmatics can exhibit reduced ability to engage in successful conversation due to reduced topic initiation, maintenance, and inhibition, as well as increased tangential speech. Clients may also demonstrate difficulties maintaining attention and eye contact during conversational exchanges. Other areas that may be impaired include reduced or flat tone and prosody and inability to recognize and interpret social cues, humor, and non-literal language. Deficits in social communication can have significant negative impacts on clients' abilities to successfully build and maintain relationships with family, friends, coworkers, teachers, and others.

Assessment of Pragmatics

Assessment of social communication can be conducted formally and informally. Self-report questionnaires (e.g., La Trobe Communication Questionnaire [Douglas, O'Flaherty, & Snow, 2000]) are often administered to assess a client's pragmatic skills and their insight into deficits. The Assessment Battery for Communication (ABaCo) is one of the only formal assessments available to measure pragmatic language skills following an acquired brain injury (Sacco et al., 2008). Informally, pragmatics are assessed through observation during conversational exchanges, as well as client and caregiver reports. It should be noted that individuals with pragmatic language disorders often exhibit reduced insight into deficits (Ham et al., 2013). Furthermore, it is important to obtain a baseline for client's pragmatic skills, for example, a client may have been verbose and tangential prior to the injury, so there may not have been a change in social communication skills. Cultural differences should also be considered when assessing pragmatics. For example, in various cultures, eye contact and initiation may be reduced for females or young adults due to normative social constructs. These factors should be considered when developing individualized treatment plans to target social communication.

Treatment of Pragmatics

The goals of pragmatic intervention are to improve social communication skills as well as increase a client's insight into weaknesses. Pragmatic language can be targeted in both individual and group sessions. These skills are typically targeted in conjunction with other speech and language goals. Examples of pragmatic treatment goals include: "Client will improve pragmatic skills by maintaining conversational topic for 8/10 exchanges given min-mod assistance" or "Client will improve eye contact with less than 5 reminders within a 30-min session." Session activities may include watching video clips demonstrating both appropriate and inappropriate social interactions and having clients identify strengths/weaknesses during the exchanges. For more direct feedback, clinicians may videotape a client during social

interaction and have the client self-reflect after watching to help increase insight of pragmatic strengths and weaknesses. Role-playing can also be used during individual or group sessions to mimic common social interactions (e.g., interacting with a salesperson, interviewing for a job, meeting new people). Furthermore, group sessions give the opportunity to target pragmatic skills such as eye contact, turn taking, initiation, tangential speech, and reduced inhibition during conversations (Braden et al., 2010). Verbal and visual cues from clinicians can be used to alert clients to appropriate and inappropriate pragmatic language. Deficits in social communication are common following an acquired brain injury, and these impairments can significantly impact an individual's ability to build and maintain social relationships. Targeting social communication in individual and group therapy sessions via role-playing, self-reflection, and explicit instruction into appropriate pragmatics have been successful at improving weaknesses and improving insight (Gabbatore et al., 2015).

Aphasia

Aphasia is an acquired neurogenic language disorder in which symptomology varies greatly from person to person. Aphasia is caused by an acquired brain injury, most commonly a cerebral vascular accident (CVA) in the left hemisphere. Due to the multitude of language deficits affecting potentially all modalities (i.e., spoken expression, auditory comprehension, written expression, and reading comprehension) at varying degrees, there are numerous subtypes, as aphasia is not a single syndrome (ASHA, 2018c). An individual will often present with relatively intact cognitive skills, however, memory and executive function deficits may co-occur depending upon the site(s) of lesion. Aphasia can be extremely disruptive to social, vocational, and educational participation causing feelings of frustration, depression, and isolation.

The hallmark feature of aphasia is difficulty with word retrieval. When faced with word-finding difficulty, individuals may pause, abandon their utterance, or they may present with *paraphasias* and/or *neologisms* within their connected speech. *Paraphasias* are productions of unintended true words that are stated in error that have either a semantic connection (i.e., stating "spoon" for fork) or a phonemic connection (i.e., stating "feet" or "sheep" instead of sheet). *Neologism* is a novel word that is substituted (e.g., "froxil" instead of "finger"). The term *jargon* is used to describe a condition in which most of the client's speech is filled with paraphasias and neologisms.

When diagnosing an acquired language disorder, it is important for a clinician to incorporate knowledge from classification systems, lesion studies, and clinical experiences. Utilization of standardized assessment tools, client and family report, and clinical observations are essential to generate an accurate picture of a client's communication abilities and impairments. There are several language skills that a clinician can assess that will help identify communication strengths and weaknesses.

Tasks that assess verbal fluency, comprehension, naming, repetition, and characteristics of spontaneous speech will aid in formulating a communication profile. There are numerous diagnostic tools. The Boston Diagnostic Aphasia Examination (BDAE-3) (Goodglass et al., 2001) and the Western Aphasia Battery-Revised (WAB-R) (Kertesz, 2007) are two well-known batteries. The most commonly used aphasic subtypes come from the Boston Classification system: Broca's, transcortical motor, global, Wernicke's, transcortical sensory, conduction, mixed transcortical, and anomic aphasias (Goodglass et al., 2001). Once a communication profile is established, a diagnosis can be determined.

On either end of the aphasic spectrum lie the extreme syndromes of global and anomic aphasias. These are usually simpler to diagnose and differentiate between. An individual with global aphasia typically has severe deficits across all language skills and modalities. Global aphasia is usually caused by sizable damage to the perisylvian region (Ferreira de Oliveira & Damasceno, 2011). Meanwhile, an individual with anomic aphasia will present with intact linguistic skills across all tasks with an isolated deficit in naming. This aphasia type usually has the most variability in regard to lesion site. Anomic aphasia has been correlated to damage in the temporal parietal area and often associated with the angular gyrus (Ferreira de Oliveira & Damasceno, 2011). Another syndrome that is a rare type of aphasia but easier to diagnose due to its peculiar presentation is mixed transcortical. Mixed transcortical aphasia presents similarly to global aphasia with an isolated preservation in repetition. Interestingly, Broca's area, Wernicke's area, and the arcuate fasciculus are intact but isolated from the rest of the brain due to damage of the surrounding watershed areas. The ability to repeat is a skill that assesses this entire language circuit. In mixed transcortical aphasia, the individual has the ability to receive language in Wernicke's area and then communicate with Broca's area via the arcuate fasciculus in order to reproduce the original message, however, as a result of the inability to interact with the rest of the brain, this preserved repetition skill is not useful for meaningful communication. The remaining five subtypes are often more challenging to differentiate between, because there are greater overlapping features. Thus, it is important for a clinician to have strong understanding of the various aphasic features.

An important feature to assess when differentiating between aphasia types is fluency. Fluency is a multidimensional concept that refers to the fluid ability to formulate phrases of ranging length with a variety of grammatical structures (Goodglass et al., 2001; Gordon, 1998). Meanwhile, non-fluent would be characterized by effortful speech with restricted phrase lengths and a reduction in grammatical features use. If an individual presents with non-fluent speech, they could potentially have Broca's, global, mixed transcortical, or transcortical motor aphasia. By contrast, if their speech was fluent, a clinician could surmise it to be Wernicke's, conduction, anomic, or sensory transcortical aphasia. During the diagnostic procedure, aphasic classifications are commonly referred to as fluent or non-fluent or even receptive or expressive aphasias.

It is important to note that not all cases of aphasia fit nicely into a subtype, and a client can often have a mixed presentation. Following an ABI, individuals often

undergo multiple phases of recovery that will require several baseline assessments in order to properly provide treatment. Therefore, the exact subtype of aphasia is not valued as high as a person's overall communication profile and their functional communication abilities during their daily activities. It is also of equal importance for a clinician to be able to differentiate between aphasias and motor speech deficits (i.e., dysarthria and apraxia). Acquired speech and language rehabilitation warrants a dynamic approach. Whether a clinician is restoring and/or augmenting communication, generating a comprehensive and accurate language profile of strengths and weaknesses throughout the recovery process is an invaluable step to providing proper intervention.

Aphasia Treatment

Aphasia intervention is a multifaceted process that incorporates *education, client, and family goals, stimulating and restoring areas of linguistic deficit, learning compensatory, communication strategies, providing caregiver training*, and *facilitation of gains across all communicative settings* (Papathanasiou & Coppens, 2017). *Education* is important for persons with aphasia, their caregivers, and primary communication partners. Aphasic education should explain the nature of deficits, their impact across modalities, the usual preservation of intellect, the course of treatment, and include setting realistic expectations, as a recovery pattern is emerging. The clinical assessment provides a clear picture of communication strengths and weakness, but the understanding of the *clients' and caregiver's goals* will help a clinician shape functional and meaningful treatment plans.

The approach to *stimulating and restoring language abilities* will vary depending on the deficits being targeted. For example, a clinician may utilize a common treatment approach for word-finding deficits known as semantic feature analysis (SFA). Semantic features analysis treatment was initially developed by Ylvisaker and Szekeres in 1985 to provide a structured method of activating semantic networks (Boyle, 2010). This approach looks to elicit the target name and its semantic features when confronted with a picture. The six semantic features traditionally produced for each target are category, use, action, properties, location, and association (Boyle & Coelho, 1995; Peach & Reuter, 2010). A graphic organizer is used to facilitate the retrieval of the target name and each of its features. For example, a circle in the center of the page would be filled with either the picture or word of the target. As an example we will use the target "apple." Apple would be in the center with boxes surrounding it. The clinician and client would then fill in the various boxes with the semantic features for apple: fruit (*category*); food (*use*); cut or bite (*action*); round, peel, stem, seeds, core, and/or various colors (*properties*); fruit bowl, supermarket, kitchen counter, tree (*location*), and apple pie or The Big Apple (*association*). A client may have difficulties formulating sentence; therefore, a clinician may want to utilize an approach of Verb Network Strengthening Treatment (VNeST). VNeST promotes word retrieval and sentence formulation by targeting

the verb as the core of a sentence (Edmonds, Nadeau, & Kiran, 2009). This approach can target the word level. However, the nature of pairing content words to verbs assists with underlying skills needed in connected speech. VNeST bridges word finding (i.e., semantic) and sentence formulation (i.e., syntactic) in a systematic way. Clients with aphasia often express an interest in improving the ability to engage in monologues and dialogues. Fluent blocks of speech are often challenging to come by when aphasia is present. Script training is an approach that allows an individual to rehearse and learn scripts verbatim with the intended outcome of automaticity in functional daily discourse (Goldberg, Haley, & Jacks, 2012; Youmans, Holland, Muñoz, & Bourgeois, 2005). There are countless approaches that a clinician may incorporate into a treatment plan. The abovementioned are very structured means to target expressive skills. A clinician may also elect to shape sessions to be less structured and allow for more open-ended discourse with clinician support.

A clinician will often simultaneously address stimulation of impaired language skills while *incorporating compensatory strategies*. Compensatory strategies may be needed on a temporary or on-going basis depending on the client's recovery pattern. Multimodality strategy integration can vary from use of gestures (i.e., head nods, pointing), visuals (i.e., pictures/drawings), simplifying sentence structure, use of an alternate word, describing the intended word, and/or use of reading and writing. There are certain clients who will benefit from incorporation of alternative and augmentative communication (AAC) to support daily communicative needs. In addition to teaching the individual with aphasia ways to compensate for communication breakdowns, a clinician will provide *caregiver training*. Caregiver training will help the family and the main communication partners support the individual with aphasia. Training may address the need to simplify communication, provide choices, utilize yes/no questions, decrease rate of communication, and/or provide semantic or phonemic cues to assist an individual with word retrieval. When time and situation permits, it is helpful for a communication partner to assist the person with retrieving a word and not just give them the word or to speak for them. Each individual with aphasia will respond differently to cues. A clinician will be able to demonstrate and teach a caregiver which techniques they can use with the individual with aphasia in order to maximize communication effectiveness. In addition to therapeutic carryover tasks and exercises, having a supportive caregiver can assist with *facilitating the carryover of skills and strategies* outside of the therapy room.

Aphasic intervention can be delivered on an individual basis, in a group setting, or a combination of the two. The group setting is an opportunity for individuals with aphasia to socialize and communicate. Aside from the psychosocial positive aspects of group therapy, there is an opportunity for clinicians to assist with generalization of skills to a more natural conversational environment (Elman, 2007; van der Gaag et al., 2005).

Motor Speech Disorders

Motor speech disorders fall into two categories, dysarthria and apraxia. They are speech disorders caused by neurological impairments affecting the motor planning, programming, neuromuscular control, or execution of speech (Duffy, 2013). They result in disorders of the voice and speech production subsystems: respiration, phonation, articulation, resonance, and/or prosody. Dysarthria and apraxia can lead to decreased speech intelligibility, reduced ability to function in communicative situations, and feelings of social isolation.

These are a large and heterogeneous class of disorders, that is, clients vary greatly with age at onset, underlying pathology, neurological condition, and description of symptoms. A breakdown in any one of the subsystems constitutes a motor speech disorder. Motor speech disorders are much more prevalent than aphasia encompassing 25% of those with strokes, one-third of those with TBI, 60% of those with Parkinson's disease, and it is often the presenting sign/symptom of ALS. Overall, motor speech disorders make up 58% of acquired neurologic communication disorders (Duffy, 2013).

Dysarthria

Dysarthria refers to disruption of speech intelligibility due to "disturbances in muscle control over the speech mechanism due to damage of the central or peripheral nervous system" (Darley, Aronson, & Brown, 1969). Dysarthria is characterized by impairments in oral communication due to paralysis, weakness, or incoordination of the speech musculature. Speech muscles can be impaired with respect to sensation, range of motion, strength, steadiness, coordination, precision, tone, endurance, speed, direction, and/or timing. When one is diagnosed with dysarthria, the muscular deficits are always present across all communicative interactions; therefore, dysarthria is consistent in nature. Dysarthria can range from mild to profound in which case the individual may be non-verbal as a result.

The framework for identifying the now well-known seven different types of dysarthria was first developed by Darley et al. (1969). They outlined clusters of disordered speech that typified the individual, unique types based on their perceptual speech characteristics. This is a crucial diagnostic framework that is widely used today. The most common dysarthria type is mixed dysarthria (Freed, 2012), which is typically a combination of some of the other types based on the various neurological regions that are impaired. The SLP can determine the type and location of the impairment by listening to the client's speech throughout a variety of tasks and contexts beginning with producing vowels and words, then increasing in length up to complex conversation. In terms of articulation, imprecise consonant production is the most common deficit and often seen in all dysarthria types; therefore, articulation is never a distinguishing factor. A seasoned SLP can listen for other hallmarks

such as a breathy voice, hypernasality, or pitch breaks to make an accurate diagnosis.

Treatment of Dysarthria

Treatment of dysarthria focuses on restoration, compensatory strategy implementation—or, in the event of poor prognosis for speech recovery—assessment and development of an augmentative/alternative communication system. Of paramount importance is education related to the disorder to aid with insight into the deficit and the rationale behind treatment. A precursor to successful treatment is stimulability. Goals include improving awareness of speaking habits, posturing, breathing patterns, rate of speech, and prosody. Feedback is crucial to learning what does and does not work. Clinician-directed feedback is needed initially with the goal of client-centered self-awareness and self-monitoring. Making therapy meaningful and functional is key to aid in transitioning from conscious speech practice to the more automatic/subconscious control present in naturalistic communication.

Principles of Motor Learning

There are several principles that a SLP must employ/impart upon retraining motor speech production. Some important principles are as follows: *Improving speech requires speaking*—physical practice is best and treatment tasks should be relevant to those needed for speech situations. *Drill is essential*, involving systematic practice of specially selected and ordered exercises, and consistent and repetitive practice is necessary for motor learning. Clients should be given *specific instruction and feedback*. Clients should internalize that speech *accuracy is more important than speed* and develop the *ability to self-cue*, thus allowing for better long-term carryover (Freed, 2012).

Treatment sessions may include external tools such as prosthetic devices. For example, a palatal lift prosthesis to aid velopharyngeal closure, reduce hypernasality, and increase intraoral pressure. Pacing boards and metronomes are also external tools that can be utilized to slow speaking rate. Behavioral strategies/techniques may include positioning and pushing or bearing down to improve breath support and improving articulatory contact via over-enunciation. Word production drills, such as contrasting word pairs (e.g., bat, hat; map, lap; match, catch) and rapid changing word lists (e.g., may, me, my, mow, moo), are examples of additional treatments. As an example, using key words, such as "cook," facilitates the production of /k/ in initial and final position and aids in the kinesthetic awareness of tongue placement during phoneme production. Prosody improvements can be targeted by drills using contrasting stress to improve variation of intonation and rhythm—for example, "*I* am hungry"; "I *AM* hungry"; "I am *HUNGRY*!" (Chapey, 2001). Using

audio and visual recordings is an excellent form of feedback and often useful in improving awareness (Duffy, 2013).

Apraxia

The diagnosis of apraxia is most common in clients with left-hemisphere cerebrovascular involvement and common among those diagnosed with aphasia. There are two types of apraxia, oral and verbal. Conditions that cause verbal apraxia (also known as apraxia of speech (AOS)), include stroke (the most common, 49%), degenerative diseases, trauma, and tumor (Freed, 2012). AOS is a neurologic speech disorder that reflects an impaired capacity to plan or program sensorimotor commands necessary for directing movements that result in phonetically and prosodically normal speech (Duffy, 2013).

In oral apraxia, the client demonstrates groping behaviors that are not structurally related, as there is no weakness or slowness of movement of the articulators. Disruptions in the sequencing of oral movements are non-verbal including smiling, puckering, protruding the tongue, and biting the lower lip. Hesitations, revisions, and groping are displayed. The problem is in voluntary movement not automatic movement. For example, the client may not be able to *show* you how they would blow out a match, but when presented with one, will do so automatically.

Verbal apraxia is characterized by deficits in programming of sequential and volitional movement of the articulators for speech production (e.g., jaw, lips, tongue, cheeks) and is not caused by muscle weakness. Sequencing errors are displayed when saying multisyllabic words. Groping to position the articulators, transposition of syllables, and phoneme substitutions are evident. For example, "*tuptake*" for "*cupcake*." Articulation errors are greater in apraxia, with a slow rate and prolonged intervals due to groping. By contrast, involuntary, spontaneous, or automatic productions such as counting and reciting the days of the week, as well as, emotionally charged words are usually free of error. Speech is commonly slow and effortful however, unlike dysarthria, apraxic errors are inconsistent with trial and error prevalent in client responses. It is common for clients with mild to moderate apraxia to typify their frustration or difficulty expressing, i.e., "*my speech won't come out right even though I know what I want to say*." Struggling/groping, attempts at self-correction, and self-awareness of errors are all cardinal signs of verbal apraxia.

Treatment of Verbal Apraxia

Not all individuals with apraxia are appropriate for speech therapy treatment (Duffy, 2013). If coexisting aphasia is severe, treatment of language should precede treatment of apraxia. Patients and families need to understand the characteristics of apraxia and the rationale for treatment. For example, families need to understand

why treatment begins with syllables and words, why there is so much repetition during practice, and why progress may be slow (Freed, 2012).

Integral stimulation is a treatment procedure first developed in the 1950s as an articulation treatment for children. It was further developed by Rosenbek, Lemme, Ahern, Harris, and Wertz (1973). During integral stimulation, the clinician cues the client: "watch me" (visual), then "listen" (auditory), then simultaneous production, followed by repetition, then client-independent repetition without cue, and finally, client production in a role-play scenario.

Melodic Intonation Therapy (MIT) is classified as a rate-rhythm type of apraxia treatment (Helm-Estabrooks, Nicholas, & Morgan, 1989). Many individuals with apraxia can sing the words of a song better than they can say the same words in a conversation. Through singing, one can make use of the undamaged right hemisphere, thus, the right hemisphere facilitates the function of the damaged left hemisphere, resulting in better verbalizations in song rather than in conversation. Clinicians initially model the word or phrase in a specific intoned singing manner that resembles the normal intonation of the phrase (i.e., raising pitch song for questions, lowering pitch song for statements), then work toward fading cues to increase client independence. Targets increase in length and complexity as the client processes. The ultimate goal of MIT is modification and generalization to a more natural speaking intonation.

Script training is a therapeutic approach that is aimed at facilitating automatic natural language production of those who present with aphasia or apraxia of speech through the use of short self-chosen monologues and dialogues (Youmans et al., 2005). Speech is an automatic process that occurs rapidly and without conscious effort. Speech is no longer automatic and speaking becomes an effortful struggle for individuals with expressive language impairments and motor speech disorders (Youmans et al., 2005). During script training, functional scripts are identified by the client and clinician. Script topics should be important, relevant, functional, and personalized to each individual client. Scripts are practiced in natural, conversational contexts and are mastered through repetitive drilling until they are produced automatically, consistently, with few errors, and without apparent struggle (Youmans et al., 2005). Generalization is promoted when practicing the scripts in conversational context with multiple partners and in non-therapeutic environment (Youmans et al., 2005). Script training incorporates traditional therapeutic approaches. Client's practice script training in a repetitive cue-based and drill manner (Youmans, Youmans, & Hancock, 2011). Intensive practice leads to automatic and more accurate productions of speech within the script.

The PROMPT program, first developed by Square-Storer and Hayden in 1989, uses a combination of proprioceptive, pressure, and kinesthetic cues to show patients how to sequence their oral movements for speech. PROMPT involves touching the client's face manually to guide the articulators at points of contact around the mouth, chin, and neck. Thus, the SLP becomes the external motor speech programmer, providing sensory input like the manner, place, and voicing of the articulators. Treatment targets progress hierarchically from syllables to words to phrases to spontaneous production.

The ultimate goal for clients with motor speech disorders is to improve the overall speech intelligibility and/or production within the limits of the individual's neurological impairment to increase functional communication. In some cases, speech may not be a realistic goal. In cases of severe dysarthria or apraxia, in that of degenerative disease, or in that of clients with concomitant diagnoses such as severe co-occurring aphasia, speech may not be realistic as a primary means of communication. Therefore, the goal with these clients may be to establish a functional means of communication via augmentative/alternative communication.

Augmentative/Alternative Communication (AAC)

According to ASHA's practice standards (ASHA, 2018d), AAC is an area of clinical research and educational practice for SLPs and audiologists that attempts to compensate and facilitate temporarily or permanently for the impairment and disability patterns of individuals with severe expressive and/or language comprehension disorders. Individuals with ABI may have severe communication, motor, and cognitive impairments that could benefit from using AAC strategies (Fager, Hux, Beukelman, & Karantounis, 2006; Glennen & DeCoste, 1997). These individuals exhibit significant difficulty communicating effectively with their families, friends, and coworkers. The heterogeneous nature of this population requires the ongoing adjustment of evaluation and training procedures to meet the ultimate goal of communicative competence within a variety of functional contexts (Doyle & Fager, 2011). The goal of an AAC team working with an individual is to provide communication assistance so that he or she is able to maximize communication participation effectively. The focus should not only be placed on the ability to communicate basic wants and needs and engage in rehabilitation services but also the ability to engage in social interactions and improve quality of life (Beukelman & Mirenda, 2013; Fried-Oken, Beukelman, & Hux, 2011); therefore, this process is ongoing and dynamic in nature (Wallace, 2010).

AAC devices can be divided into two general classifications, manual or electronic. Manual devices include object boards, single switch communicators, picture communication boards, wordbooks, and letter boards. By contrast, electronic devices employ the use of computerized software programs displayed on dynamic screens. An electronic device can be dedicated computers or tablets with operating software designed solely for communication that may or may not interface with other technologies. More commonplace devices such as smartphones or tablets can utilize applications (apps) to assist with intervention and communication (Edwards & Dukhovny, 2017). Additionally, a clinician will need to take several features of an AAC system into consideration when selecting the best means of communication, such as *mode of access or selection* (i.e., manual or eye gaze), *language characteristics* (i.e., picture, symbol, and/or written word), and *output* (i.e., spoken or written forms) (Beukelman & Mirenda, 2013). For individuals with physical deficits, an

SLP often consults with a PT or OT to determine the best body positions, means of access, and/or positions of AAC on wheelchair.

Individuals who receive an AAC system and their caregivers will undergo an acceptance process. Individuals and families should be counseled that the use of an AAC system can be temporary or permanent. While serving to increase immediate functional communication, AAC can be temporarily used as a bridge to the re-acquisition of communication and/or be utilized as a permanent communication means when impairment will be long-term. It is vital that the individual and caregiver be actively involved in the selection of an AAC system following a comprehensive evaluation process. Consequently, they will be more likely to use the system effectively, feel a greater sense of commitment, and be less likely to abandon the device. In a study conducted by Fager et al. (2006), it was found that adults with traumatic brain injury generally accepted both low-tech and high-tech AAC devices and utilized systems for extended durations. However, abandonment of systems was more so due to the loss of facilitator support than rejection of the technology. Therefore, it is imperative to engage and train communicative partners (e.g., spouses, caregivers) in order to facilitate carryover and may increase long-term acceptance of the AAC device (Kent-Walsh, Murza, Malani, & Binger, 2015). SLPs should educate the family/caregiver on the benefits of AAC, operations of the system, and ways to support functional communication.

Case Study

MG is a 79-year-old male; status: post CVA resulting in moderate-severe expressive and receptive aphasia, as well as verbal apraxia. He was always polite but was limited to head nods, verbal perseverations, and would say "yes" in response to everything. He was cared for by his wife who was able to anticipate his needs; however, communication and conversation remained a rather significant challenge. His wife attended every session and would carry over exercises and activities regularly. Five months post CVA, MG made very little functional gains in his communication, as he still had significant difficulty comprehending session activities and his verbal output remained severely restricted. His SLP began to incorporate Script Training and an AAC device (which was previously too complicated for MG to use). Although still impaired, a major turning point in recovery occurred as MG began making positive expressive gains and increased the ability to repeat the clinician, spouse, and the AAC device. Rehearsal of his script training allowed the client to produce lengthier sentences with good speech production. The AAC device provided visual (pictures and words) supports for MG to comprehend and understand the clinician. The AAC device additionally provided MG and his spouse a tool to assist repair of communication breakdowns and expand his ability to express wants/needs that were not directly in his environment. Over the following months, MG was able to improve ability to follow instruction, comprehend questions, and make spontaneous comments and jokes. With his spouse well trained to provide supportive communication

and the AAC device, MG was able to have greater participation in social activities and communication.

Bilingual

As the prevalence of bilingual and multilingual speaking individuals in the United States increases, the need for tailored speech-language pathology services for these clients has become vital. Recent census data have shown that there are more than "350 languages spoken in US homes," and that "between 2012 and 2016, 21.1% of the population age 5 and older spoke a language other than English at home" (US Census Bureau, 2017). This number is only expected to rise in coming years. Due to language and cognitive processing differences in bilingual individuals compared with monolinguals, modifications need to be made in both assessment and treatment to provide adequate services (Jones et al., 2011; Marian & Spivey, 2003).

Assessment

When conducting an assessment for a bilingual individual, the tests should ideally be administered in the client's preferred language by a speech-language pathologist that is fluent in that language. If necessary, secondary methods for evaluation including the use of an interpreter or the use of a translation service via video or telephone should be utilized (ASHA-Bilingual Service Delivery: Key Issues, n.d.). Finally, if other options are not possible, the speech-language pathologist should have a family member/friend translate the evaluation materials into the client's preferred language. The Bilingual Aphasia Test, an aphasia battery produced in more than 58 different languages, can be utilized for this purpose (Paradis & Libben, 2014). While conducting a bilingual assessment, it is important to also consider any cultural or language differences that may occur. For example, during the confrontational naming section of the Boston Diagnostic Aphasia Examination, a client who recently emigrated from Russia may be unfamiliar with a "beaver" or a "cactus" (Roth, 2011). Inability to name these objects may not stem from word retrieval difficulties, but may occur due to unfamiliarity with these items. Items that may be foreign to these patients should be omitted during assessment.

During the clinical interview, it is essential for the clinician to develop a comprehensive language profile for the client. This language profile should contain the client's education level, reading, and writing abilities in each language, as well as information relating to the client's preferred occupational and social language use (Rimikis et al., 2013). Prior to the onset of therapy, the speech-language pathologist is charged with developing a comprehensive language profile according to results of both formal and informal assessment. After developing the language profile and analyzing the results of the assessments, the clinician is charged with determining if

language deficits are present in all languages. For example, there may be a parallel impairment in both languages or the level of impairment may differ for each language. Due to variation of impairment in each individual language, treatment goals should be developed that take into account both or all languages.

Treatment

The intervention plan for the bilingual client should be developed considering the client's preferences, performance on evaluation, and language proficiencies. Treatment plans can be created to target both languages simultaneously or sequentially. Research has shown that there is no advantage to simultaneous treatment of both languages over sequential treatment (Kurland & Falcon, 2011). Recent research has also explored recovery patterns for bilingual individuals and has found that recovery is not uniform; level and speed of recovery may fluctuate over the course of treatment for each of the languages (Green et al., 2010). Ideally, treatment should be provided by a bilingual therapist, with secondary and tertiary options consisting of an interpreter, translation service, or client family member/friend.

While creating an intervention plan for a bilingual client, it is important to note whether a speech and/or language disorder is present or if symptoms are merely reflective of a language difference. For example, syntactic errors in one language may not be a sign of aphasia or difficulty with sentence formulation but a reflection of crossover from the client's additional language. Phonological differences should also be taken into account in determining speech sound disorders and rating intelligibility as phonics are unique to each language. Furthermore, cultural differences should be taken into account when conducting therapy. For example, a client's food preferences/dietary restrictions due to cultural/religious reasons need to be taken into account during food trials. In conclusion, assessment and treatment of bilingual clients adds an additional layer of complexity, and consideration needs to be made during assessment and treatment for whichever language/s the client speaks.

Dysphagia

Dysphagia can be defined as when any one or more of the stages of swallowing becomes impaired due to changes in sensation, muscle strength, and coordination, whereby the client can no longer safely or efficiently swallow (Logemann, 1998). Dysphagia can range from mild to severe and can be caused by physical and/or cognitive impairments with implications including malnutrition, dehydration, and aspiration pneumonia. Aspiration refers to a food or liquid bolus falling below the level of the vocal folds into the airway. Aspiration can occur before, during, or after the pharyngeal swallow (Lazarus, 1989). Therefore, signs and symptoms of aspiration may be delayed or silent. *Silent aspiration* refers to aspiration of a bolus with

no overt behavioral signs or symptoms. According to estimates, silent aspiration may occur in up to 40% of clients with dysphagia, and it is *not* generally identifiable during the bedside swallow evaluation (Murry & Carrau, 2001).

Evaluation of Swallowing Disorders

Subjective Assessment Procedures

The *bedside clinical evaluation* assesses both structure and function of the swallow mechanism at the oral preparatory, oral, and pharyngeal phases of the swallow. This involves assessing sensation, strength, ROM (both volitional and reflexive responses) of the oral-pharyngeal structures. Assessment includes gathering a full medical history, noting pulmonary history, gastrointestinal history, nutrition and hydration status, current medications, and surgical history. During the bedside evaluation, the SLP may work with the occupational therapist to further assess a client's positioning and self-feeding abilities. The SLP assesses a variety of consistencies and textures with the aim of recommending the least restrictive safe and appropriate diet. The SLP assesses as many consistencies as possible given the client's current level of alertness, cognitive, and physical impairment. Ideally, an evaluation should be conducted within the context of a full meal, as opposed to an isolated event, in order to simulate the client's everyday naturalistic environment.

Objective Assessment Procedures: Imaging and Instrumentation

The SLP collaborates with a radiologist when performing a Modified Barium Swallow Study (also referred to as Videofluorographic study). The MBS is a comprehensive, dynamic evaluation of all phases of swallowing requiring clinical expertise to interpret results. The recommendation of an MBS often follows the identification of dysphagia risk factors found during the clinical bedside evaluation (Murry & Carrau, 2001). The client is presented with a variety of food and liquid consistencies, which have been impregnated with barium. "The modified barium swallow study is designed to assess not only whether the client is aspirating, but also why, so appropriate treatment can be initiated" (Logemann, 1998). Furthermore, it helps to determine the safest diet texture as well as compensatory techniques to facilitate the safest, most-efficient swallow. Another objective assessment tool is a Flexible Endoscopic Evaluation with Sensory Testing (FEESST), under the supervision of an otolaryngologist the SLP may perform a FEESST, which involves a fiber optic endoscope that passes through the nasal cavity to a position above the epiglottis. It serves to examine the anatomy and physiology of the oral cavity and the pharynx from above, before, and after the swallow (Aviv, 2000). The exam includes sensory testing via an air pulse presented above the level of the vocal folds. This

provides insight into the client's ability to reflexively protect their airway and thus prevent aspiration.

Treatment of Swallowing Disorders

A multidisciplinary approach is essential in order for the treatment plan to be carried out effectively. The team includes the SLP, occupational therapist, physician, family members, and/or caregivers. The SLP utilizes multiple approaches to swallow safety, including restorative therapy, compensatory techniques, and diet modification.

Restorative Therapy Techniques

Oral-pharyngeal exercises are widely used to improve awareness, strength, movement, coordination, and volitional control of the lips, tongue, cheeks, mandible, larynx, and vocal folds. Vocal fold closure is a key factor in preventing aspiration; therefore, vocal fold adduction exercises are important for clients whose vocal folds fail to close sufficiently. Bolus control and chewing exercises can also be used to improve fine motor coordination of the tongue. However, only limited data is available to demonstrate the efficacy of oral-pharyngeal exercises on positive clinical outcomes (i.e., weight gain, reduced aspiration), for clients with neurological impairments (Murry & Carrau, 2001). By contrast, the act of swallowing itself is the most efficacious and best therapy for the swallow. The client can also be trained to perform specific maneuvers that are employed to improve volitional control over various aspects of the pharyngeal phase of the swallow. These maneuvers require alertness, physical effort, and the ability to follow specific complex directions. Therefore, they may not be a feasible treatment modality for clients who exhibit significant cognitive-linguistic impairments.

The Deep Pharyngeal Neuromuscular Stimulation (DPNS), a dysphagia treatment that was first developed in 1993, represents an organic understanding of the swallow mechanism. DPNS is defined as a systemized therapeutic method for pharyngeal dysphagia that utilizes direct neuromuscular stimulation to the pharyngeal musculature to restore muscle strength, endurance, reflex response, and reflex coordination for a restored, coordinated swallow. The interaction of cold temperature, sour taste, and deep pressure applied by the SLP works to elicit sensory-motor responses (i.e., tongue base retraction, velopharyngeal closure, laryngeal elevation, pharyngeal wall constriction, vocal fold closure, swallow reflex trigger, and saliva production) (Stefakanos, 2005).

Neuromuscular Electrical Stimulation is a restorative treatment modality that had traditionally been used by physical and occupational therapists. This approach is now widely used and accepted for the treatment of swallowing disorders. NMES

is the use of electrical stimulation for the activation of muscles via stimulation of intact peripheral motor nerves through the skin (a transcutaneous medium). VitalStim® (Wijting & Freed, 2003) is the brand name of an FDA-cleared method to promote swallowing through the application of neuromuscular electrical stimulation. The major treatment goals are to strengthen weak muscles, maintain or gain ROM, facilitate voluntary motor control, and increase sensory awareness (Wijting & Freed, 2003). To date, there is significant research to support the use of VitalStim® with continued numerous ongoing studies. A meta-analysis by Chen et al. (2016) regarding NMES post stroke where all used VitalStim® compared the effects of traditional therapy alone, NMES alone, and the use of traditional dysphagia therapy plus concurrent NMES. The results found that traditional therapy plus the use of concurrent NMES was better than traditional therapy alone. For example, mild-moderate dysphagia clients benefit the most with over 80% discontinuing feeding tubes (Scarponi et al., 2015). Overall efficacy studies indicate that the use of electrical stimulation contributes significantly to improvement in swallowing function (Blumenfeld, Hahn, Lepage, Leonard, & Belafsky, 2006).

Another recent advancement involves interfacing NMES + sEMG. sEMG is a form of biofeedback that measures muscle activity non-invasively through surface electrodes. The signals measured and displayed on screen as biofeedback to the patient. Crary, Carnaby Mann, Groher, and Helseth (2004) found that a dysphagia therapy program supplemented with sEMG biofeedback improved functional swallowing status in the majority of stroke and head and neck cancer clients who had not improved with previous standard therapy programs. A study by Bogaardt, Grolman, and Fokkens, performed in 2009, suggests that sEMG is an effective adjunct to standard therapy for swallowing disorders finding that the use of sEMG as biofeedback in the treatment of chronic dysphagia after stroke produced significant improvement of swallowing function and feeding tube removal in 6–8 tube-dependent patients. Thus, the use of sEMG biofeedback in the treatment of swallowing disorders is an important advancement that engages a client with tangible biofeedback about their swallow performance (particularly useful for clients with sensation impairments) in the aim for positive treatment outcomes.

Compensatory Treatment Techniques

A compensatory strategy, such as the use of a head, neck, or body postural change, generally requires less physical effort on the part of the client and potentially temporarily changes the dimensions of the pharynx and the direction of food flow. Postural changes have been shown to improve oral-pharyngeal transit times, reduce the risk of aspiration, and decrease the amount of residue after the swallow (Logemann, 1998). Widely used postures include chin tuck and head rotation to the weaker side. In general practice, carryover of postural techniques may be compromised in clients with moderate–severe cognitive impairments. Further compensatory strategies for a client with oral and pharyngeal deficits may include an SLP's

Table 9.1 Diet consistencies

Least restrictive to most restrictive consistencies	
Food	Liquid
Unrestricted diet	Thin
Mechanically altered	Nectar
Ground	Honey
Puree	NPO
Therapeutic feeding by SLP only	
NPO	

recommendations to remain upright for 30 min post-meal to reduce risk of aspiration, take controlled bites and sips, alternate solids and liquids, cueing the client to perform multiple swallows, and training the client to clear or remove food pocketing in the mouth. These strategies also may help to reduce the risk of aspiration.

Diet modification is another component in the treatment of dysphagia; however, this should be considered as a last resort in treatment planning. Oral nutrition and hydration is the ultimate goal for clients with dysphagia. The National Dysphagia Diet (NDD), published in 2002 by the American Dietetic Association, aims to establish standard terminology and practice applications of dietary texture modification in dysphagia management (McCullough, Pelletier, & Steele, 2003). Even though there is variation, diets are typically developed in a stepwise progression of bolus consistencies. Table 9.1 demonstrates the typical progression of diet textures.

Since it is unsafe for certain clients to eat by mouth, it is determined that they must receive nutrition and hydration via alternative, non-oral means, a status known as NPO, or non-perioral. The decision of whether to have a feeding tube inserted for non-oral feeding is a crucial one for clients and families, and they often do not understand that feeding tubes can be temporary. In these cases, counseling should clearly emphasize the benefits to the client, i.e., good nutrition/hydration enabling them to do better in therapy. Clients and families may choose to go against the recommendations for a specific dysphagia diet, and it is the SLP's responsibility to educate them regarding the potential health risks of aspiration.

Case Study

HV is a 60-year-old male; status: post right-side CVA with left-sided weakness. Prior to the CVA, HV was independent and worked as a custodian. Moderate receptive and mild expressive language deficits included difficulty with auditory processing, word finding, and verbal fluency. Cognitive deficits were demonstrated in memory, attention, problem solving, and impulsivity. An MBS study revealed decreased oral motor control, premature spillage, delayed swallow trigger with silent penetration on all liquid consistencies. Silent aspiration with delayed cough was noted on thin liquids. Diet recommendations were for mechanical soft foods

with honey thick liquids. Compensatory strategies taught included remaining upright 30 min post-meal, small bites/sips, checking mouth for pocketing, and alternating solids/liquids. VitalStim® therapy was used in conjunction with traditional swallow exercises and food/liquid trials. HV demonstrated difficulty isolating tongue/jaw movements. During therapy, he required repetition, cues to decrease impulsivity, and increase attention. Cognition improved over time. Over time, HV also demonstrated increased tolerance for electrical stimulation but continued to demonstrate impaired sensation on his left side. He tolerated NMES for 50 min 3×/week, paired with an "effortful" swallow and head turn to the left with trials of thin liquids. Increased management of thin liquids was evident by treatments 9–12. Repeat MBS revealed an improvement in swallow function, with mild oral-pharyngeal; however, no penetration or aspiration was noted. Diet recommendations were regular foods cut up into small pieces and cup sips of thin liquids. Recommendations for compensatory techniques were modified to include no straws, remaining upright 30 min post-meal, dry swallows to clear any residue in oropharynx, and monitoring for signs/symptoms of aspiration. HV was discharged to home with recommendations to continue the above safe eating strategies.

Conclusion

As part of the comprehensive neurorehabilitation team, speech-language pathologists are responsible for evaluating, educating, and providing restorative or compensatory therapy for a broad spectrum of disorders. It is essential to involve the family/caregivers as partners in the therapy process, providing education and training to enable carryover into home-based and community-based settings. Once a client maximizes gains in the outpatient setting, post-rehabilitation programs and activities will likely be recommended in order to maintain gains, provide cognitive and communicative stimulation, as well as for the client's overall well-being. (For further information regarding post-discharge recommendations, refer to Chap. 14). Utilizing an interdisciplinary team approach that includes speech therapy, physical therapy, occupational therapy, neuropsychology, and outside medical professionals (such as neuropsychiatrists, ENTs, dieticians) will ensure the best functional outcome for the client.

References

Adamovich, B. B., & Henderson, J. (1992). *Scales of cognitive ability for traumatic brain injury (SCATBI)*. Austin, TX: Pro-Ed.

American Speech-Language-Hearing Association. (2018a). *Learn about the CSD profession*. Rockville, MD: Author. Retrieved July 15, 2018, from ASHA.org: https://www.asha.org/Students/Learn-About-the-CSD-Professions/

American Speech-Language-Hearing Association. (2018b). *The audiogram*. Rockville, MD: Author. Retrieved July 15, 2018, from ASHA.org: https://www.asha.org/public/hearing/Audiogram/

American Speech-Language-Hearing Association. (2018c). *Clinical topics: Aphasia*. Rockville, MD: Author. Retrieved April 24, 2018, from ASHA.org: http://www.asha.org/Practice-Portal/Clinical-Topics/aphasia/

American Speech-Language-Hearing Association. (2018d). *Augmentative and alternative communication (Practice Portal)*. Rockville, MD: Author. Retrieved May 8, 2018, from www.asha.org/Practice-Portal/Professional-Issues/Augmentative-and-Alternative-Communication/

ASHA. (n.d.). *Bilingual service delivery: Key issues*. Rockville, MD: Author. Retrieved from https://www.asha.org/PRPSpecificTopic.aspx?folderid=8589935225§ion=Key_Issues

Aviv, J. E. (2000). Prospective, randomized outcome study of endoscopy versus modified barium swallow in patients with dysphagia. *Laryngoscope, 110*, 563–574.

Beukelman, D. R., & Mirenda, P. (2013). *Augmentative & alternative communication* (4th ed.). Baltimore, MD: Paul H. Brookes Publishing.

Blumenfeld, L., Hahn, Y., Lepage, A., Leonard, R., & Belafsky, P. C. (2006). Transcutaneous electrical stimulation versus traditional dysphagia therapy: A nonconcurrent cohort study. *Otolaryngology-Head and Neck Surgery, 135*(5), 754–757. https://doi.org/10.1016/j.otohns.2006.04.016

Bogaardt, H., Grolman, W., & Fokkens, W. (2009). The use of biofeedback in the treatment of chronic dysphagia in stroke patients. *Folia Phoniatrica et Logopaedica, 61*(4), 200–205. https://doi.org/10.1159/000227997

Boyle, M. (2010). Semantic feature analysis treatment for aphasic word retrieval impairments: What's in a name? *Topics in Stroke Rehabilitation, 17*(6), 411–422.

Boyle, M., & Coelho, C. A. (1995). Application of semantic feature analysis as a treatment for aphasic dysnomia. *American Journal of Speech-Language Pathology, 4*, 94–98.

Braden, C., Hawley, L., Newman, J., Morey, C., Gerber, D., & Harrison-Felix, C. (2010). Social communication skills group treatment: A feasibility study for persons with traumatic brain injury and comorbid conditions. *Brain Injury, 24*(11), 1298–1310. https://doi.org/10.3109/02699052.2010.506859

Channon, S., & Watts, M. (2003). Pragmatic language interpretation after closed head injury: Relationship to executive functioning. *Cognitive Neuropsychiatry, 8*(4), 243–260. https://doi.org/10.1080/135468000344000002

Chapey, R. (2001). *Language intervention strategies in aphasia and related neurogenic communication disorders* (4th ed.). Baltimore, MD: Williams & Wilkins.

Chen, Y. W., Chang, K. H., Chen, H. C., Liang, W. M., Wang, Y. H., & Lin, Y. N. (2016). The effects of surface neuromuscular electrical stimulation on post-stroke dysphagia: A systemic review and meta-analysis. *Clinical Rehabilitation, 30*(1), 24–35. Retrieved from https://www.ncbi.nlm.nih.gov/pubmed/25697453

Cicerone, K. D., Dahlberg, C., Malec, J. F., Langenbahn, D. M., Felicetti, T., Kneipp, S., … Catanese, J. (2005). Evidence-based cognitive rehabilitation: Updated review of the literature from 1998 through 2002. *Archives of Physical Medicine and Rehabilitation, 86*, 1681–1691.

Crary, M., Carnaby Mann, G. C., Groher, M., & Helseth, E. (2004). Functional benefits of dysphagia therapy using adjunctive sEMG biofeedback. *Dysphagia, 19*(3), 160.

Dabul, B. L. (2000). *Apraxia battery for adults* (2nd ed.). Austin, TX: Pro-Ed.

Dardier, V., Bernicot, J., Delanoë, A., Vanberten, M., Fayada, C., Chevignard, M., … Dubois, B. (2011). Severe traumatic brain injury, frontal lesions, and social aspects of language use: A study of French-speaking adults. *Journal of Communication Disorders, 44*(3), 359–378. https://doi.org/10.1016/j.jcomdis.2011.02.001

Darley, F. L., Aronson, A. E., & Brown, J. R. (1969). Differential diagnostic patterns of Dysarthria. *Journal of Speech and Hearing Research, 12*, 246.

Douglas, J. M., O'Flaherty, C. A., & Snow, P. C. (2000). Measuring perception of communicative ability: The development and evaluation of the La Trobe Communication Questionnaire. *Aphasiology, 14*(3), 251–268.

Doyle, M., & Fager, S. (2011). Traumatic brain injury and AAC: Supporting communication through recovery. *The ASHA Leader, 16*(2). https://doi.org/10.1044/leader.FTR8.16022011.np. Online only. Retrieved from http://leader.pubs.asha.org/article.aspx?articleid=2278988

Duffy, J. R. (2013). *Motor speech disorders: Substrates, differential diagnosis, and management*. St. Louis, MO: Elsevier.

Edmonds, L. A., Nadeau, S. E., & Kiran, S. (2009). Effect of verb network strengthening treatment (VNeST) on lexical retrieval of content words in sentences in persons with aphasia. *Aphasiology, 23*(3), 402–424.

Edwards, J., & Dukhovny, E. (2017). Technology training in speech-language pathology: A focus on tablets and apps. *Perspect ASHA SIGs, 2*(SIG 10), 33–48. https://doi.org/10.1044/persp2.SIG10.33

Elman, R. J. (2007). *Group treatment of neurogenic communication disorders: The expert clinician's approach* (2nd ed.). San Diego, CA: Plural Publishing.

Enderby, P., & Palmer, R. (2008). *Frenchay dysarthria assessment* (2nd ed.). Austin, TX: Pro-Ed.

Estabrooks, N. (2017). *Cognitive linguistic quick test plus (CLQT+)*. Bloomington, MN: Pearson/PsychCorp.

Estabrooks, N., & Hotz, G. (1991). *Brief test of head injury (BTHI)*. Austin, TX: Pro-Ed.

Fager, S., Hux, K., Beukelman, D. R., & Karantounis, R. (2006). Augmentative and alternative communication use and acceptance by adults with traumatic brain injury. *Augmentative and Alternative Communication, 22*(1), 37–47.

Ferreira de Oliveira, F., & Damasceno, B. P. (2011). A topographic study on the evaluation of speech and language in the acute phase of a first stroke. *Arquivos de Neuro-Psiquiatria, 69*(5), 790–798.

Freed, D. B. (2012). *Motor speech disorders: Diagnosis and treatment* (2nd ed.). Clifton Park, NY: Delmar, Cengage Learning.

Fried-Oken, M., Beukelman, D., & Hux, K. (2011). Current and future AAC research considerations for adults with acquired cognitive and communication impairments. *Assistive Technology, 24*, 56–66.

Gabbatore, I., Sacco, K., Angeleri, R., Zettin, M., Bara, B. G., & Bosco, F. M. (2015). Cognitive pragmatic treatment. *The Journal of Head Trauma Rehabilitation, 30*(5), E15. https://doi.org/10.1097/htr.0000000000000087

Glennen, S., & DeCoste, D. C. (1997). *The handbook of augmentative and alternative communication*. Clifton Park, NY: Thomson/Delmar Learning.

Goldberg, S., Haley, K. L., & Jacks, A. (2012). Script-training and generalization for people with aphasia. *American Journal of Speech-Language Pathology, 21*(3), 222–238. https://doi.org/10.1044/1058-0360(2012/11-0056)

Goodglass, H., Kaplan, E., & Barresi, B. (2001). *Boston diagnostic aphasia examination* (3rd ed.). Baltimore, MD: Lippincott Williams & Wilkins.

Gordon, J. K. (1998). The fluency dimension in aphasia. *Aphasiology, 12*(7/8), 673–688.

Green, D. W., Grogan, A., Crinion, J., Ali, N., Sutton, C., & Price, C. J. (2010). Language control and parallel recovery of language in individuals with aphasia. *Aphasiology, 24*(2), 188–209. https://doi.org/10.1080/02687030902958316

Ham, T. E., Bonnelle, V., Hellyer, P., Jilka, S., Robertson, I. H., Leech, R., & Sharp, D. J. (2013). The neural basis of impaired self-awareness after traumatic brain injury. *Brain, 137*(2), 586–597. https://doi.org/10.1093/brain/awt350

Haskins, E. C., Cicerone, K., Dams-O'Connor, K., Eberle, R., Langenbahn, D., Shapiro-Rosenbaum, A., & Trexler, L. E. (2014). *Cognitive rehabilitation manual. Translating evidence-based recommendations into practice* (1st ed.). Reston, VA: ACRM Publishing.

Helm-Estabrooks, N., Nicholas, M., & Morgan, A. R. (1989). *Melodic intonation therapy (manual)*. San Antonio, TX: Special Press.

Jones, O. P., Green, D. W., Grogan, A., Pliatsikas, C., Filippopolitis, K., Ali, N., & Price, C. J. (2011). Where, when and why brain activation differs for bilinguals and monolinguals during picture naming and reading aloud. *Cerebral Cortex, 22*(4), 892–902. https://doi.org/10.1093/cercor/bhr161

Kent-Walsh, J., Murza, K. A., Malani, M. D., & Binger, C. (2015). Effects of communication partner instruction on the communication of individuals using AAC: A meta-analysis. *Augmentative and Alternative Communication, 31*(4), 271–284.

Kertesz, A. (2007). *Western aphasia battery-Revised*. San Antonio, TX: Pearson.

Kurland, J., & Falcon, M. (2011). Effects of cognate status and language of therapy during intensive semantic naming treatment in a case of severe nonfluent bilingual aphasia. *Clinical Linguistics & Phonetics, 25*(6-7), 584–600. https://doi.org/10.3109/02699206.2011.565398

Lazarus, C. L. (1989). Swallowing disorders after traumatic brain injury. *The Journal of Head Trauma Rehabilitation, 4*(4), 34–41.

Logemann, J. A. (1998). *Evaluation and treatment of swallowing disorders*. Austin, TX: Pro-Ed.

MacDonald, S. (2017). Introducing the model of cognitive-communication competence: A model to guide evidence-based communication interventions after brain injury. *Brain Injury, 31*, 1760–1780.

Marian, V., & Spivey, M. (2003). Competing activation in bilingual language processing: Within- and between-language competition. *Bilingualism: Language and Cognition, 6*(2), 97–115. https://doi.org/10.1017/s1366728903001068

McCullough, G., Pelletier, C., & Steele, C. (November, 2003). National dysphagia diet: What to swallow? *The ASHA Leader, 8*, 16–27. https://doi.org/10.1044/leader.FTR3.08202003.16

Murry, T., & Carrau, R. L. (2001). *Clinical manual for swallowing disorders*. San Diego, CA: Singular.

Papathanasiou, I., & Coppens, P. (2017). *Aphasia and related neurogenic communication disorders* (2nd ed.). Burlington, MA: Jones & Bartlett Learning.

Paradis, M., & Libben, G. (2014). *The assessment of bilingual aphasia*. London: Psychology Press.

Parola, A., Gabbatore, I., Bosco, F. M., Bara, B. G., Cossa, F. M., Gindri, P., & Sacco, K. (2016). Assessment of pragmatic impairment in right hemisphere damage. *Journal of Neurolinguistics, 39*, 10–25. https://doi.org/10.1016/j.jneuroling.2015.12.003

Peach, R. K., & Reuter, K. A. (2010). A discourse-based approach to semantic feature analysis for the treatment of aphasic word retrieval failures. *Aphasiology, 24*(9), 971–990.

Porch, B. E. (2001). *Porch index of communicative ability–Revised (PICA-R)*. Albuquerque: PICA Programs.

Rimikis, S., Smiljanic, R., & Calandruccio, L. (2013). Nonnative English speaker performance on the basic English lexicon (BEL) sentences. *Journal of Speech Language and Hearing Research, 56*(3), 792. https://doi.org/10.1044/1092-4388(2012/12-0178)

Rosenbek, J. C., Lemme, M. L., Ahern, M. B., Harris, E. H., & Wertz, R. T. (1973). A treatment of apraxia of speech in adults. *Journal of Speech and Hearing Disorders, 38*, 462–472. https://doi.org/10.1044/jshd.3804.462

Ross-Swain, D. G. (1996). *Ross information processing assessment* (2nd ed.). Austin, TX: Pro-Ed.

Roth, C. (2011). Boston diagnostic aphasia examination. In *Encyclopedia of clinical neuropsychology* (pp. 428–430). New York, NY: Springer.

Sacco, K., Angeleri, R., Bosco, F. M., Colle, L., Mate, D., & Bara, B. G. (2008). Assessment battery for communication — ABaCo: A new instrument for the evaluation of pragmatic abilities. *Journal of Cognitive Science, 9*(2), 111–157. https://doi.org/10.17791/jcs.2008.9.2.111

Scarponi, L., Mozzanica, F., Cristofaro, V. D., Ginocchio, D., Pizzorni, N., Bottero, A., & Schindler, A. (2015). Neuromuscular electrical stimulation for treatment-refractory chronic dysphagia in tube-fed patients: A prospective case series. *Folia Phoniatrica et Logopaedica, 67*(6), 308–314. https://doi.org/10.1159/000443499

Square-Storer, P., & Hayden, D. (1989). PROMPT treatment. In P. Square-Storer (Ed.), *Acquired apraxia of speech in aphasic adults* (pp. 165–189). London: Taylor & Frances.

Stefakanos, K. H. (2005). *Comprehensive DPNS: A Dysphagia Workshop on Deep Pharyngeal Neuromuscular Stimulation. Resource text*. Dover, FL: The Speech Team, Inc.

US Census Bureau. (December 07, 2017). *Commuting times, median rents and language other than English use*. Washington, DC: Author. Retrieved from https://www.census.gov/newsroom/press-releases/2017/acs-5yr.html

Van der Gaag, A., Smith, L., Davis, S., Moss, B., Cornelius, V., Laing, S., & Mowles, C. (2005). Therapy and support services for people with long-term stroke and aphasia and their relatives: A six-month follow-up study. *Clinical Rehabilitation, 19*, 372–380.

Wallace, S. E. (2010). AAC use by people with TBI: Affects of cognitive communication. *Perspectives on Augmentative and Alternative Communication., 19*(3), 79–86.

Wijting, Y., & Freed, M. L. (2003). *VitalStim® therapy manual*. Hixson, TN: Chattanooga Group.

Youmans, G., Holland, A., Muñoz, M. I., & Bourgeois, M. (2005). Script training and automaticity in two individuals with aphasia. *Aphasiology, 19*, 435–450.

Youmans, G., Youmans, S. R., & Hancock, A. B. (2011). Script training for adults with apraxia of speech. *American Journal of Speech-Language Pathology, 20*, 23–27.

Chapter 10
Neuropsychiatry and Traumatic Brain Injury

Angela Scicutella

Introduction

Although the earliest descriptions of brain injuries date back to the ancient Egyptians (1700 BC) where 27 cases of head trauma are recorded in *The Edwin Smith Surgical Papyrus*, the neuropsychiatric concept that behavioral sequelae can result from brain injury was not understood, as this culture believed that the heart was the seat of emotion and thinking (Finger, 2000). Later on, in the historical timeline, there appears to be some hint of recognition that the brain and human behavior may be linked when in 400 BC, the Greek physician Hippocrates wrote *On Injuries of the Head* and described a patient with head trauma who subsequently experienced delirium and seizures (Hippocrates). More recently, in 1848, the now well-known case of Phineas Gage, who suffered destruction of the left frontal lobe of his brain while at his job laying down track for a new railroad, was documented. Subsequent to his injury, he evidenced a change in personality marked by impulsivity and poor judgment. He was unable to resume his occupation where he had previously been highly regarded, as his colleagues noted, "He was no longer Gage." This landmark case in our modern era linked Gage's brain trauma as being etiologically responsible for his emotional changes (Barker, 1995). Subsequently, others such as Adolf Meyer in 1904, proposed that brain trauma from a variety of causes could lead to neuropsychiatric syndromes such as delirium, psychosis, memory problems, and mania, and he introduced the nosology "post-traumatic insanity" to try to define this phenomenon more precisely (Meyer, 1904). More recently, in 1972, the neuropsychologist Dr. Luria described the case of a Russian soldier, Leva Zasetsky, who had suffered a bullet wound to the left parieto-occipital area of his brain during combat

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in World War II. Dr. Luria worked with him for 25 years and recorded this patient's courageous struggle to recover some of his ability to function despite cognitive deficits and frightening hallucinations (Luria, 1972).

In the United States, according to an updated report by the Centers for Disease Control, about 2.8 million patients sustain head trauma each year due to falls, vehicular accidents, violence, or sports injuries (Taylor, Bell, Breiding, & Xu, 2017). Due to improved acute trauma care in hospitals, many patients survive the physical ravages of traumatic brain injury (TBI), but many subsequently experience neuropsychiatric disorders such as those described in the patients above. In this chapter, the modern neuropsychiatrist's role in the diagnosis and treatment of the psychiatric consequences of TBI such as mood and anxiety disorders, psychosis, agitation, arousal and attention, dementia, and sexual dysfunction will be reviewed.

Depression

Hopelessness and sadness are characteristic of the emotional state known as depression, which is a commonly observed neuropsychiatric condition after TBI. The psychiatrist's handbook, known as the *Diagnostic and Statistical Manual Text* (DSM-5) (American Psychiatric Association, 2013), outlines the necessary symptoms which a patient must experience to be diagnosed with major depression. These include a depressed mood or loss of pleasure for 2 weeks, as well as the presence of four or more of the following symptoms: change in appetite or weight loss, insomnia or hypersomnia, fatigue or loss of energy, being restless or slowed down to a degree that is observable by others, feeling worthless, being unable to concentrate, or having suicidal thoughts, plan, or attempt. However, patients suffering from various medical conditions such as TBI can have a prominent disturbance in their mood marked by many of the above characteristics and yet not fulfill the criteria for major depression. Such a patient would be categorized by DSM-5 (American Psychiatric Association, 2013) criteria as having depressive disorder due to another general medical condition (TBI). In using this symptom profile, a study of 666 outpatients with TBI found that the three symptoms which most differentiated depressed from nondepressed patients were feeling hopeless, feeling worthless, and having difficulty enjoying activities (Seel et al., 2003). In a literature review of clinical considerations for making a diagnosis of depression in TBI patients, the same author noted that irritability, anger, and aggression rather than sadness along with features of rumination and guilt, are more characteristic of this population (Seel, Macciocchi, & Kreutzer, 2010).

The critical symptom in the diagnostic criteria regarding suicidality must be emphasized given the increased awareness of its association with TBI (Dreer et al., 2018). In reviewing the literature, it is important to note that in addition to suicide itself, some studies have reviewed suicidal-related behaviors such as suicidal attempts and suicidal ideation. An early study examined post-injury suicide attempts in 172 outpatients and reported a rate of 17.4% in this group followed over a 5-year period; an increased risk was observed in those patients who demonstrated hopelessness,

prior suicidal attempts, and suicidal ideation (Simpson & Tate, 2002), whereas two recent research studies analyzing suicidal ideation, which was defined as thoughts about killing oneself with or without a plan, but with no attempt, reported similar rates of this behavior, 28% (Tsaousides, Cantor, & Gordon, 2011) and 25% (Mackelprang et al., 2014).

The issue of death by suicide of TBI patients was reported as between 2.7 and 4.1 times that of the general population in an earlier epidemiological study (Teasdale & Engberg, 2001). The risk of suicide remained constant over the 15-year period that the patients in that study were followed, highlighting the fact that suicide is not just an acute problem in the first few months subsequent to a devastating injury. Several recent studies have highlighted similar findings of the chronic risk of suicide in patients with TBI. In a Swedish longitudinal population study spanning 41 years, the mortality rates of 218,300 TBI patients, who survived beyond the first 6 months after sustaining their injury, were compared to a population of controls who were age- and sex-matched totaling 2,163,190 individuals, with the conclusion that even 5 years after the diagnosis of TBI there was an elevated risk of suicide with an adjusted odds ratio of 2.7 (Fazel, Wolf, Pillas, Lichtenstein, & Langstrom, 2014). In the Traumatic Brain Injury Model Systems (TBMS), a TBI population studied over a 20-year period, suicidal ideation ranged from 7% to 10% and suicidal attempts ranged from 0.8% to 1.7% (Fisher et al., 2016). A large retrospective cohort study performed in Denmark involved 567,823 TBI patients who had any medical contact, which included emergency room visit, hospitalization, or outpatient treatment, over the period from 1980 to 2014. Of this TBI population, the rate of suicide was double that of a population of patients who committed suicide without TBI history. Those with more severe TBI and skull fractures, increased number of TBIs (three or more), and psychological illness either before or after TBI had higher suicide risk than those with mild TBI (Madsen et al., 2018). However, despite the association of suicide with more severe TBI, a just published study involving data extracted from cohort, cross-sectional, and case control studies found a twofold increased risk of suicide behavior, which includes suicidal ideation and attempts even among those experiencing one concussion or mild TBI compared to individuals without such a history (Fralick et al., 2018).

Risk factors for suicidal behavior highlighted in one study of patients who had a history of TBI and a diagnosis of either major depression or bipolar disorder, noted that the patients were more likely to be males, to have a history of substance abuse, to be aggressive and hostile, and to have been diagnosed with narcissistic, borderline, antisocial, or histrionic personality disorders (Oquendo et al., 2004). In addition to the above factors, recent research on suicidal ideation also found correlations with a diagnosis of anxiety, prior suicidal attempts, having Medicaid insurance, and less than high school education (Mackelprang et al., 2014). By contrast, a young woman, in a recent case report with no history of psychiatric illness or substance abuse, died by suicide while being treated in the hospital 2 weeks after sustaining diffuse subarachnoid hemorrhage and contusions of the left frontal lobe in a motor vehicle accident (Rocca, Caputo, Frigiolini, Verde, & Ventura, 2018). This report emphasizes that despite the fact that most suicides after TBI occur months to years

after the injury and with premorbid risk factors, patients in the post-traumatic delirium phase can also be at risk for this devastating consequence of TBI.

Executive dysfunction on neuropsychological testing as well as frontal lobe white matter damage in TBI patients also appear to be correlated with suicidal behavior (Brenner et al., 2015; Yurgelun-Todd et al., 2011). Furthermore, there has been some evidence to suggest that suicidal behavior is related to immune dysregulation as inflammatory factors, such as interleukins and tumor necrosis factor (TNF) which are activated in TBI, can worsen neurodegeneration (Brundin, Bryleva, & Rajamani, 2017); TNF levels in TBI were found to be elevated and correlated positively with disinhibited, impulsive behaviors and suicidal ideation in TBI patients in one study (Juengst, Kumar, Arenth, & Wagner, 2014).

Epidemiology and Risk Factors

The occurrence of depression after TBI has been estimated to be between 6% and 77% (Jorge & Robinson, 2003). Various methodological factors have been suggested to explain the wide range in statistics, including how the sample was chosen (i.e., referral to a specialty TBI clinic or a community population study); size of the sample (small or large); what subgroup of patients was assessed (i.e., mild, moderate, or severe TBI patients or some combination thereof); when studies were done in relation to injury (i.e., in the first few months after the incident or years or even decades later); what type of assessment tool was utilized (patient self-report questionnaire, family's report of the patient, or a clinician's structured diagnostic interview) (Newburn, 1998); and the medication status of patients at the time of the assessments (i.e., the effect narcotics, steroids, or benzodiazepines may have had on the rating of symptoms) (O'Donnell, Creamer, Bryant, Schnyder, & Shalev, 2003). Moreover, the diagnostic process is further complicated by the overlap between the two syndromes in that certain symptoms such as sleep and appetite changes as well as psychomotor agitation have been argued to be the neurological sequelae of the TBI itself, rather than the result of depression as a primary mood disturbance (Babin, 2003; Moldover, Goldberg, & Prout, 2004).

Some recent studies have looked at TBI populations over shorter time frames; a cohort of 559 patients with complicated mild-to-severe TBI followed for 1 year revealed a rate of major depression as 53.1% (Bombardier et al., 2010), while a 2-year cohort study (SHEFBIT—Sheffield Brain Injury After Trauma) noted a diagnosis of depression of 56.3% in 774 patients with all levels of severity of TBI (Singh, Mason, Lecky, & Dawson, 2018). Other studies over the past decade have recorded a range of depression rates: 11.6% in a study by Bryant et al. (2010) in patients with mild traumatic brain injury and a new diagnosis of depression at 12 months; 18.7–28.3% in patients with moderate-to-severe TBI by Alway, Gould, Johnston, McKenzie, and Ponsford (2016); 45% in patients 6 months to 5.5 years after TBI with the injury severity in the study ranging from mild to severe (Whelan-Goodinson, Ponsford, Johnston, & Grant, 2009); and Ouellet et al. (2018) recorded a rate of 29% over 12 months.

Despite these issues in research protocols, what is significant in terms of clinical outcomes is that the risk of depression has been reported to remain elevated for decades following brain injury, as has been highlighted in the studies. The lifetime prevalence of major depression 50 years after closed-head injury for 520 veterans who had sustained TBI during World War II was noted to be 18.5%, while a current diagnosis of major depression was recorded in 11.2% of those same veterans (Holsinger et al., 2002). A second report of 60 patients who had been followed for 30 years post TBI recorded a lifetime rate of major depression of 26.7%, with 10% having current illness at the time of the study (Koponen et al., 2002). However, in another study of TBI patients, a decline in the frequency of psychiatric disorders was noted over time, challenging the conclusion that the rate of psychiatric diagnoses, including depression, remains elevated years later. The researchers suggest that using cross-sectional, longitudinal, and cross-sequential assessments, where age and time after injury are controlled for at enrolment to the study, may help to improve the accuracy of the epidemiological data in future studies with this population of patients (Ashman et al., 2004). There is variability about the outcomes in that some studies report decrease in rates over time and others note stability, or in the case of Ouellet's study, some patients with milder depression episodes at the study outset worsened with a diagnosis of major depression later on. Given the variability in trajectories over time, it is important to carefully monitor these patients to optimize their outcomes (Osborn, Mathias, Fairweather-Schmidt, & Anstey, 2018).

Since major depression among the survivors of TBI is associated with diminished quality of life and poorer psychosocial functioning in studies which have examined patients in both the acute and chronic phases of TBI, the need for early recognition and treatment interventions in this patient population is a pressing one (Hibbard et al., 2004; Rapoport, McCullagh, Streiner, & Feinstein, 2003; Underhill et al., 2003). A few studies have approached this problem by trying to identify relatively homogeneous subgroups within the inherent heterogeneity of TBI patients with the technique of growth mixture modeling; demographic, clinical history, and psychiatric and substance use history variables are recorded at or soon after TBI and used to classify patients into depression trajectories (low, high, delayed, recovery, and persistent). In one of these studies, for example, the high depression group revealed that they had more severe TBI, where female had a shorter hospital stay; using this model in the future to predict target patients and intervene with treatment earlier may improve prognosis (Bombardier, Hoekstra, Dikmen, & Fann, 2016; Gomez, Skilbeck, Thomas, & Slatyer, 2017).

Several factors which have been suggested as being correlated with the development of depression after TBI include lesion location (left dorsal lateral frontal lesions and/or left basal ganglia lesions, as well as possibly parietal–occipital and right hemispheric lesions), poor social functioning (less than high school education, unstable employment, and relationship difficulties), and previous psychiatric history (depression and substance abuse) (Dikmen, Bombardier, Machamer, Fann, & Temkin, 2004; Fann et al., 2004; Federoff et al., 1992; Gomez-Hernandez, Max, Kosier, Paradiso, & Robinson, 1997; Jorge et al., 2005). In a recent meta-analysis of 26 studies spanning from 1992 to 2016, with outcomes measured between 1 month and 6 years, depression was associated with female gender, preinjury depression,

postinjury unemployment, and lower brain volume (Cnossen et al., 2017). Some research indicates that neuroanatomical markers may predict those who may be at greater risk for major depression after their injuries, and these factors include reduced hippocampal volumes, white matter abnormalities in frontotemporal regions, and in one study, damage to the white matter tracts such as right arcuate fasciculus, right inferior longitudinal fasciculus, anterior facets of the corpus callosum, and left and right fronto-occipital longitudinal fasciculi (Jorge, Acion, Starkstein & Magnotta, 2007; Rao et al., 2012; Spitz, Alway, Gould, & Ponsford, 2017). Additionally, in recent sophisticated imaging, functional MRI research has examined depression in relation to brain regions involved in emotional processing, and TBI patients were found to have altered the integrity of functional networks that included the insula, thalamus, and anterior cingulate cortex (Moreno-Lopez, Sahakian, Manktelow, Menon, & Stamatakis, 2016). As noted in the above section on suicide, dysregulation of the immune system may be another associated factor to be explored in future studies, since elevated cytokines which are signaling proteins involved in inflammation are aberrantly elevated in both TBI and depression leading to a potential common pathway (Bodnar, Morganti & Bachstetter, 2018).

Assessment

Assessing a patient begins with the taking of a thorough history to explore the details of the TBI incident and the subsequent treatment and hospital course which took place in the acute care setting. Past and present medical history other than TBI should be reviewed, as the clinician must consider the possibility that medical comorbidities such as epilepsy, stroke, brain tumors, infections, systemic neoplasms, cardiac or renal disease, and endocrinological disorders (thyroid, adrenal, and pituitary) may be relevant and play a contributory role in the patient's presentation of depression. For example, a common comorbid medical problem in TBI survivors which impacts on mood is hypopituitarism, especially growth hormone deficiency. In a recent update in the literature on this topic, the authors cite possible factors involved, including severe TBI, diffuse axonal injury, basal skull fracture, blast TBI, and autoimmune dysfunction (antibodies against the hypothalamus and pituitary); direct trauma to the pituitary, its stalk, or the vasculature (hypophyseal portal veins) can lead to ischemia of the gland, and then deficiencies in growth hormone or other gonadotropins (luteinizing or follicle-stimulating hormones) may occur. Recommendations for screening in the acute and chronic phases of TBI for dysfunction in the endocrinological hormones involved in the hypothalamic–pituitary–adrenal axis are provided to identify those patients who can benefit from hormone replacement therapy (Tan et al., 2017), as significant improvements in depression, anxiety, and fatigue have been observed when replacement was administered (Popovic, Aimaretti, Casanueva, & Ghigo, 2005).

A review of the patient's and his/her family's psychiatric history is the key area to explore, since other psychiatric disorders such as bipolar illness, anxiety syndromes,

adjustment disorders, and substance abuse/dependence can either produce overlapping symptoms or be comorbid with depression (Seel et al., 2010). This has been observed in several studies: 76.7% of patients with TBI and depression met criteria for a comorbid anxiety disorder (Jorge et al., 2004) as did 60% of the patients, in the study by Bombardier et al. (2010), who had TBI severity ranging from complicated mild to severe. Pain and sleep problems (see detailed section below) are often present in TBI patients and can greatly affect mood, so a thorough assessment of these issues should also be explored when considering a possible diagnosis of depression (Branca & Lake, 2004; Ouellet, Savard, & Morin, 2004). The current medications taken by the patient must be reviewed, since many pharmacological agents such as anticonvulsants, cardiac medications, steroids, hormones, and psychiatric medications can cause depression as a side effect. Of paramount importance to the history is an in-depth review of the patient's use of alcohol or illicit substances, as they too can play a role in the manifestation of depressive symptoms. Questions about psychosocial factors such as education, occupation, sexual history, current relationships, and avocations help the clinician to have a more complete portrait of the person as a human being, and not just a patient with TBI. To be thorough, the clinician should speak to the patient's family or friends to corroborate the history provided by the patient, so that the most accurate information guides the workup and treatment (Sadock, Sadock, & Ruiz, 2015). Additionally, during the initial assessment process, the neuropsychiatrist should seek to communicate with other members of the treating team to discuss their observations of the patient as this may help the clinician to clarify diagnostic issues. Furthermore, this liaison approach is important in promoting a dialog between the disciplines to enhance the patient's treatment as he or she progresses in the recovery process.

Subsequent to the history, the clinician performs a complete physical and neurological exam, including a cognitive assessment which reviews orientation, attention, memory, language skills, visuospatial abilities, praxis, and frontal lobe tasks. During the psychiatric mental status exam, appearance, attitude, speech, motoric abnormalities (such as tremor), mood, psychotic symptoms (such as paranoia and hallucinations), homicidality (aggressive tendencies), and suicidality are assessed. After this thorough evaluation, the neuropsychiatrist may order appropriate lab tests based on his/her findings, such as a complete blood count (CBC), electrolytes, endocrine panel, electrocardiogram (EKG), electroencephalogram (EEG), and brain imaging to help clarify the diagnosis of depression.

Differential Diagnosis

Pertinent to the differential diagnosis of depression in TBI patients is the syndrome of apathy which is frequently reported in patients after TBI and greatly impacts on the rehabilitative efforts as well as the patient's function and relationships with the caregivers (Andersson, Gundersen, & Finset, 1999; Marin & Wilkosz, 2005). The significance of this symptom was recently highlighted in a study that reported

apathy present at 3 months was associated with poor outcome at 1 year after injury (DeBoussard, Lannsjo, Stenberg, Stalnacke, & Godbolt, 2017). Updated research on diagnostic criteria for apathy has attempted to hone the definition of this entity as it is not a category in DSM-5. In one model, it is defined as: (1) lack of motivation and diminished goal-directed behavior (lack of energy and need for prompts to structure activity); (2) diminished goal-directed cognition (diminished interest in learning new things and lack of concern about one's problems); and (3) diminished response to either positive or negative events as evidenced by an unchanging or flat affect (Starkstein & Leentjens, 2008). To help differentiate between a mood disorder (depression) and apathy (motivational syndrome), there are several rating scales used for a variety of neurological disorders, but the Apathy Evaluation Scale (AES) is most often used in the assessment in TBI (Marin, Biedrzycki, & Firinciogullaari, 1991). The Neuropsychiatric Inventory (NPI) (Cummings, Mega, Gray, Rosemberg-Thompson, & Gornbein, 1994) is another scale used to evaluate behavioral, emotional, and motivational symptoms in patients with neurological illness, and in a study of 120 severe TBI patients it was noted that apathy was exhibited in 42% of the cases (Ciurli, Formisano, Bivona, Cantagallo, & Angelelli, 2011). Despite being frequent sequelae of TBI, the frequency of this problem is not well categorized due to difficulties in the study methodologies. Ranges in the literature for apathy are reported as low as 10.84% in a study of 59 TBI patients with apathy, but without concomitant depression (Kant, Duffy, & Pivovarnik, 1998), whereas in two studies by Andersson and colleagues (Andersson & Bergedalen, 2002; Andersson, Gundersen, & Finset, 1999; Andersson, Krogstad, & Finset, 1999), the prevalence of apathy in TBI patients using AES was greater than 60%. Even across cultures, the concept of motivational deficits has been found to be a relevant construct, as was demonstrated in a study of 80 TBI patients in a nonindustrialized country where the incidence of apathy, as measured by AES, was reported to be 20% (Al-Adawi et al., 2004). In a review of the literature, the overall point prevalence of apathy was cited as 47.3% in patients with TBI (Arnould, Rochat, Azouvi, & Van der Linden, 2013).

Anatomically, apathy has been associated with dysfunctional activity in subcortical–frontal circuits which involve the basal ganglia, limbic structures, anterior cingulate, and prefrontal cortex (Masterman & Cummings, 1997). Neuroimaging studies involving lesion mapping in patients with TBI validate the association of apathy symptoms with damage to the anterior cingulate and frontal lobe regions and, in addition, the insula, supplementary motor area and white matter in the corona radiata and corpus callosum (Knutson et al., 2014). Using diffusion tensor imaging (DTT), a case report concluded that a 46-year-old female with a current clinical presentation of apathy in the context of a mild TBI but with a past history of left putamen intracranial hemorrhage from which she completely recovered suffered traumatic axonal injury to the prefrontocaudate white matter tract which accounted for her symptoms (Jang & Kwon, 2017). Neuropsychological testing of patients with apathy in recent reports describe deficits in multitasking exercises as well as memory (Arnould, Rochat, Azouvi, & Van der Linden, 2018; Arnould, Rochat, Dromer, Azouvi, & Van der Linden, 2018).

In contrast to apathy, which is marked by emotional indifference, the clinician who is assessing for mood disorders should also be aware of the patients with TBI who can exhibit sudden uncontrollable outbursts of laughing or crying, which are usually brief, stereotyped in nature and can occur multiple times per day; this disorder is referred to as pseudobulbar affect (PBA). These involuntary episodes are triggered by trivial stimuli which ordinarily would not result in such an extreme affective response (Zeilig, Drubach, Katz-Zeilig, & Karatinos, 1996). Since the patient's prevailing mood is neither one of depression nor euphoria, these incongruent responses can be a source of embarrassment. Epidemiologically, the prevalence of PBA was recorded as 10.9% during the first year after TBI (Tateno, Jorge, & Robinson, 2004), but a more recent study recorded the prevalence to be as high as 48.2% (Work, Colamonico, Bradley, & Kaye, 2011). Neuroanatomically, while brain stem nuclei mediate the acts of laughing and crying by integrating facial and respiratory functions, the motor cortex exerts control over the expression of these emotions. Therefore, a lesion along the pyramidal tracts between these brain regions can result in PBA (Wilson, 1924). An alternative hypothesis to account for this clinical entity is that there is disruption of the cerebro-ponto-cerebellar pathways (Parvizi, Anderson, Martin, Damasio, & Damasio, 2001).

Sleep disturbances in TBI have become much more recognized sequelae, with about 50% of TBI patients with the gamut of mild-to-severe TBI complain of sleep problems (Mathias & Alvaro, 2012). This can interfere with the rehabilitation efforts in the early post-TBI period and quality of life more chronically (Theadom, Cropley, Parmar, Barker-Collo, Starkey, Jones, Feigin, and on behalf of the BIONIC Group, 2015). One study indicated that TBI patients with early sleep problems after injury had symptoms of depression, anxiety, and apathy 12 months post injury (Rao, McCann, Han, Bergey, & Smith, 2014). Cognitive issues can be exacerbated by sleep difficulties also (Duclos, Beauregard, Bottari, Ouellet, & Gosselin, 2015). The sleep problem can be present as a symptom of depression or anxiety, but alternatively it can be due to a lesion in the neuronal pathways involved in regulating the sleep–wake cycle, a result of pain from the injury or a medication side effect. In a meta-analysis review of 1706 TBI patients, a variety of subcategories of sleep problems were documented, and included insomnia—29% (getting and staying asleep), hypersomnia—28% (need for an increased amount of sleep and excessive daytime sleepiness), obstructive sleep apnea—25%, periodic leg movements of sleep—19%, and narcolepsy—4%. Circadian rhythm disorder is another sleep disturbance observed in TBI which can be present in the acute and chronic phases of TBI. This phenomenon occurs when the environmental 24-h day clock is not in synchrony with the biological clock in the hypothalamus, specifically the suprachiasmatic nucleus, causing the disruption of times of wakefulness/sleep and wreaks havoc with the individual's function (Duclos et al., 2014; Grima, Ponsford, & Pase, 2017). In addition, some patients have parasomnias such as REM behavior disorder in which dreams are re-enacted because atonia is lost which can be dangerous to the bed partner when there is thrashing and kicking as part of the dream state. A thorough evaluation of sleep issues is needed to determine the diagnostic sleep entity. The clinical evaluation should include the history about the type and severity of

injury (blast or blunt trauma), mood and anxiety disorders, pain syndromes, and maladaptive sleep behaviors (lack of routine in sleep/wake times, use of alcohol and caffeine in relation to bedtime, television, and electronic devices in the bedroom), all of which can impact sleep disturbances (Wickwire et al., 2018). Tools to aid in the diagnosis of sleep disorders can include sleep questionnaires such as the Epworth Sleepiness Scale (assesses falling asleep in different daytime situations) and Pittsburgh Sleep Quality Index (measures sleep quality, latency, duration, efficiency, disturbance of sleep, medications, and dysfunction in the daytime), both of which have been validated in TBI, a sleep diary recorded by the patient, actigraph (device that is like a watch which measures motor activity during sleep/wake periods), and polysomnography (an objective evaluation of sleep with the measurement of brain waves with EEG, muscle tone with EMG, and eye movements with electro-oculogram coupled with cardiac and respiratory evaluations) (Buysse, Reynolds III, Monk, Berman, & Kupfer, 1989; Duclos et al., 2014; Johns, 1991; Ouellet, Beaulieu-Bonneau, & Morin, 2015).

Understanding the pathophysiology of TBI sleep problems is an ongoing area of research which will assist in informing better treatment modalities in the future. Conceptually, damage as a result of TBI to brain areas such as the hypothalamus which is involved in promoting normal sleep/wake rhythms would then subsequently alter the balance of hormones which play a role in these physiological states and would include hypocretin (orexin), which is involved in maintaining an individual's state of wakefulness, and melatonin (Grima et al., 2017).

Treatment

Drug treatment for depression in TBI patients is based on good clinical judgment, experience with psychotropic medications in other neurological disorders, as well as limited studies and case reports on these agents in the TBI population. Pharmacotherapy should be administered at the lowest effective doses initially with gradual increases as clinically indicated, with the goal being to ameliorate target symptoms and to minimize troublesome side effects which can interfere with rehabilitation efforts and wreak havoc on the patient's quality of life.

The class of antidepressant medications known as SSRIs, which selectively inhibit serotonin reuptake by presynaptic neurons and include fluoxetine (Prozac), sertraline (Zoloft), citalopram (Celexa), escitalopram (Lexapro), and paroxetine (Paxil), is generally the choice of treatment for TBI patients due to the lack of cardiac side effects, a lower risk of inducing seizures, and fewer anticholinergic side effects. There are open-label studies and case reports of TBI patients whose depression improved when they were treated with the first three agents (Cassidy, 1989; Fann, Uomoto, & Katon, 2000; Horsfield et al., 2002; Perino, Rago, Cicolin, Torta, & Monaco, 2001; Rapoport et al., 2008; Turner-Stokes, Nibras, Pierce, & Clegg, 2002). Subsequently, a 4-week double-blind parallel group trial with ten patients in each arm of the study was conducted involving sertraline, the stimulant methylphe-

nidate (Ritalin) (see section below), and placebo, which indicated that depressive symptoms in TBI patients improved significantly with either agent as compared to the placebo group (Lee et al., 2005). An updated study by Ashman et al. (2009) was a double-blind, controlled study of 52 patients with TBI ranging from mild to severe who were administered sertraline which showed no statistically significant difference between the drug and placebo groups; both groups improved on the scores of depression, anxiety, and quality of life. In newer studies utilizing sertraline in TBI patients, work by Jorge, Acion, Burin, and Robinson (2016) found in a double-blind placebo-controlled, parallel group, randomized trial with 46 placebo and 48 treatment TBI patients that this agent was efficacious in preventing the onset of depressive disorders, while a study by Fann et al. (2017), in a similarly designed trial with 62 participants who were within 1 year of TBI, showed that there was no significant difference between placebo and treatment groups, as both improved. The side effects of SSRIs include diarrhea, nausea, vomiting, insomnia, sedation, tremors, and sexual dysfunction.

Venlafaxine (Effexor), which inhibits both serotonin and norepinephrine reuptake, has been reported anecdotally to be useful in treating depression in TBI patients. Nausea, constipation, dry mouth, and hypertension can be observed as side effects (Rao & Lyketsos, 2002). In one study, there was limited evidence to recommend the treatment of TBI patients with phenelzine (Nardil), a member of the monoamine oxidase inhibitors (MAO-Is), a class of antidepressants which blocks the catabolism of norepinephrine and serotonin (Saran, 1985). However, with the risk of a hypertensive crisis, if dietary sources of tyramine in foods such as cheese and wine are consumed, these agents are best avoided in TBI patients. Another agent, bupropion (Wellbutrin), which acts to increase the efficiency of the noradrenergic neurotransmitter systems, may be utilized in patients who have depression marked by apathy, but the risk of seizures at higher dosages of this medication makes it a less attractive choice in TBI patients (Shaughnessy, 1995).

Historically older agents, the tricyclic antidepressants (TCAs), which block the reuptake of norepinephrine and serotonin into the presynaptic neuron, were previously more frequently used in the treatment of patients with depression and TBI. Examples of these drugs include amitriptyline (Elavil), nortriptyline (Pamelor), and desipramine (Norpramin). The latter drug was utilized in a small blinded, randomized, placebo-lead-in study and showed efficacy in improving the symptoms of depression in ten patients with TBI (Wroblewski, Joseph, & Cornblatt, 1996). Side effects of the TCAs include cardiac arrhythmias, sedation, and anticholinergic effects, such as dry mouth, confusion, and urinary retention. There is a potential for these medications to lower the seizure threshold, and therefore, in patients with TBI who are already at risk for this complication, vigilance in using the lowest doses possible should be the rule, although at the present time these agents are rarely utilized.

Several recent meta-analyses of antidepressant treatment in TBI revealed varying opinions about efficacy, with one stating there was no significant benefit (Kreitzer et al., 2019) and the other two noting possible, but not robust evidence of improvement (Paraschakis & Katsanos, 2017; Salter, McClure, Foley, Sequeira, & Teasell, 2016). Given the significant problem that depression post TBI poses on the

return to function on this population, the need for a well-designed placebo-controlled trial is imperative.

In patients with PBA which can mimic depression, the SSRI class of medications has also been used successfully to treat this disorder (Muller, Murai, Bauer-Wittmund, & Von Cramon, 1999; Tateno et al., 2004). However, a newer agent was approved specifically for this disorder which is a combination of dextromethorphan (DM) and quinidine. The pharmacology is complex, with the former component (DM) having antagonist properties at the *N*-methyl-D-aspartate and nicotinic receptor sites, agonist properties at the sigma 1 receptor sites, and also serves as a serotonin–norepinephrine reuptake inhibitor, while the quinidine component of the compound helps to make DM more bioavailable in the central nervous system. In one group of patients in the open-label study of PBA in the Pseudobulbar Affect Registry Investigating Symptom Management II (PRISM II), 120 TBI patients demonstrated a reduction in PBA episodes compared to their baseline with this medication (Hammond, Sauve, Ledon, David, & Formella, 2018). Common side effects of this medication are diarrhea and headache.

In patients with a predominant clinical picture of apathy rather than depression, medications which target the dopamine pathways in the brain should be utilized, since a disruption of dopamine transmission is implicated in the etiology of amotivation. Psychostimulants, which cause the release of catecholamines such as dopamine and norepinephrine from presynaptic neurons, have demonstrated benefits with regard to mood, cognition, and motivation in TBI patients, as has been noted in placebo-controlled studies of methylphenidate (Gualtieri & Evans, 1988; Plenger et al., 1996), as well as a chart review of dextroamphetamine (Cylert) (Hornstein, Lennihan, Seliger, Lichtman, & Schroeder, 1996). Side effects can include psychosis, anxiety, irritability, insomnia, and increases in heart rate and blood pressure. The potential for an increased rate of seizures, while present, has been uncommon clinically. Other agents which augment dopaminergic transmission and can be used in this population of patients include amantadine (Symmetrel) (Kraus & Maki, 1997; Nickels, Schneider, Dombovy, & Wong, 1994; van Reekum et al., 1995), levodopa/carbidopa (Sinemet) (Lal, Merbtiz, & Grip, 1988), bromocriptine (Parlodel) (Muller & Von Cramon, 1994; Powell, Al-Adawi, Morgan, & Greenwood, 1996), and selegiline (Eldepryl) (Moutaouakil, El Otmani, Fadel, & Slassi, 2009; Newburn & Newburn, 2005). Psychosis, gastrointestinal side effects, and orthostatic hypotension can occur with these medications. Another option for patients with TBI and apathy is modafinil (Provigil), which promotes wakefulness and is approved for narcolepsy, but whose exact pharmacological mechanism of action is unknown. It has shown the potential for increasing alertness and attention and improving cognition in an open-label trial of ten TBI patients (Teitelman, 2001). The most common side effect of modafinil is headache, but nausea, vomiting, and anxiety can also occur.

In addition to the disruption of dopamine transmission in apathy, there is evidence to suggest that dysfunction of the cholinergic system can also lead to amotivation. This is based on the research done in Alzheimer's dementia (AD): patients with AD are often apathetic, while biochemically they suffer from a deficiency of the neu-

rotransmitter acetylcholine. Neuroanatomically, in both TBI and AD, cholinergic limbic–neocortical connections which are damaged can cause interference in the integration of cognitive and emotional processes (Cummings & Back, 1998). Therefore, cholinergic agents, such as acetylcholinesterase inhibitors (AchE-Is) which temporarily disrupt the hydrolysis of acetylcholine and thus increase its concentration in the synapse, have been shown to be beneficial in improving apathy in AD patients (Cummings, 2000). With this rationale, it was demonstrated that in one uncontrolled trial of four TBI patients in which AchE-I donepezil (Aricept) was used, apathy scores on a structured rating scale improved (Griffin, van Reekum, & Masanic, 2003). Side effects of AchE-Is include nausea, vomiting, and diarrhea.

In the pharmacological treatment of depression, sleep disturbances must be addressed as noted above. Treatment of sleep problems post TBI can employ both pharmacological and nonpharmacological strategies. Due to the dearth of rigorous studies evaluating sleep medications and TBI, clinical choices are predicated on analogous sleep issues in other disorders, sleep pathophysiology, and avoidance of side-effects which will worsen cognition. If the sleep disturbance is due to depression, then sleep will likely improve when the patient is treated with one of the above-discussed antidepressant agents. For circadian rhythm disturbance, melatonin as an over-the-counter supplement, or ramelteon, a prescription medication that functions at the melatonin receptor may be tried (Kemp, Biswas, Neumann, & Coughlan, 2004; Lequerica, Jasey, Portelli Tremont, & Chiaravalloti, 2015). A trial of an agent such as trazodone (Desyrel), a relatively specific inhibitor of serotonin reuptake, can be used as it is sedating, but there are no studies in the TBI population. An important side effect to monitor with this medication is orthostatic hypotension. Hypnotics such as benzodiazepines (BZDs) (e.g., lorazepam [Ativan]), which broadly enhance gamma-aminobutyric acid transmission, and nonbenzodiazepines (e.g., zolpidem [Ambien]), which are selective BZD agonists, are best avoided due to the side effects such as confusion, sedation, unsteady gait, and dependence issues (Ouellet et al., 2004). Modafanil can be tried for narcolepsy and daytime sleepiness (Kaiser et al., 2010). Prazosin, which is an alpha-1 receptor adrenergic blocker, was found to help patients suffering from TBI and post-traumatic stress disorder (Ruff, Ruff, & Wang, 2009). Side effects of this agent can include dizziness, drowsiness, or headache. Although no studies have been specifically reported, the theoretical use of suvorexant which blocks orexin could be a potential agent in treating insomnia, but caution is needed due to side effects that can include mood changes and problems with coordination (Viola-Saltzman & Musleh, 2016). However, if sleep problems persist, it is recommended that adjustment of the patient's environment and sleep patterns be implemented with sleep hygiene techniques. A recent pilot study in TBI patients used warm footbaths 30 min before bedtime to help the chronic insomnia of TBI patients as a way to relax patients for sleep (Chiu, Lin, Chiu, & Chen, 2017). Nonpharmacological treatments also specifically include cognitive behavior therapy (CBT) for insomnia to help in the restructure of negative thinking with regard to the issues of falling/staying asleep, blue light therapy (Sinclair, Ponsford, Taffe, Lockley, & Rajaratnam, 2014), continuous positive airway pressure (CPAP) devices, or oral appliances for sleep apnea (Wickwire et al., 2018).

If medications are not successful in treating depression, then electroconvulsive therapy (ECT), a nonpharmacological option, is an alternative, as has been shown in a few case reports of TBI patients. Although the precise mechanism to explain the reason for ECT's effectiveness is not known, hypotheses include neurogenesis, release of trophic factors, and promotion of neuroplasticity by resetting disrupted functional brain networks (Srienc, Narang, Sarai, Xiong, & Lippman, 2018). ECT does result in a seizure to produce efficacy; so, caution is warranted in its use in a population of patients with higher risk of seizures at baseline (Ferguson et al., 2010). Another concern with this therapy is that it can cause cognitive side effects, as was recently shown in a case report of a patient who suffered subarachnoid hemorrhage, diffuse axonal injury, and bilateral subdural hygromas. After failing multiple trials of medications, an initial positive response to ECT for depression was followed by relapse of mood symptoms; a second trial of ECT was halted due to cognitive deterioration (Horinkova, Bartecek, & Fedorova, 2017). However, the use of unilateral rather than bilateral electrodes may help to diminish this side effect (Crow, Meller, Christenson, & Mackenzie, 1996; Ruedrich, Chu, & Moore, 1983).

An additional potential nonpharmacological treatment for TBI patients is repetitive transcranial magnetic stimulation (rTMS), which utilizes the application of an electromagnetic field to depolarize neurons underneath the scalp where the transducer is placed; the effectiveness of this treatment is hypothesized to be the modulation of the activity of synapses in brain regions involved in fronto-subcortical-limbic connected circuitry, areas which are thought to be involved in the manifestation of depression (Reti, Schwarz, Bower, Tibbs, & Rao, 2015). Case reports highlighting its use in TBI depression are sparse (Cavinato, Iaia, & Piccione, 2012; Fitzgerald et al., 2011); a recent one involved improvement in depression symptoms in a 37-year-old male who suffered diffuse axonal injury in a fall in the context of a suicide attempt (Iliceto, Seiler, & Sarkar, 2018). Similiar to ECT, modifications to prevent seizures, which are also potential side effects of rTMS, are suggested and include using low-frequency pulses over the right hemisphere rather than high-frequency pulses over the left hemisphere (Fitzgerald et al., 2003).

Since TBI patients require support and education to help them cope with their injuries, psychotherapy as a treatment option for patients with depression and TBI cannot be overemphasized (Prigatano, 1991). A recent report of 77 depressed TBI patients who were randomized to receive either supportive psychotherapy or cognitive behavioral therapy which uses the concept of altering automatic negative thought patterns noted that both therapies were effective in ameliorating symptoms of depression (Ashman, Cantor, Tsaousides, Spielman, & Gordon, 2014). A study which evaluated new onset depression in 103 TBI patients without any history of depression prior to injury noted that the patients' depression at 3, 6, and 12 months after injury was associated with their perceived decreased post-TBI social functioning rather than a veridical change in their social networks. This highlights the important role that psychotherapeutic techniques can have in the treatment of these patients to help them to change their cognitive distortions to more realistic perceptions (Roy, Koliatsos, Vaishnavi, Han, & Rao, 2018). In addition, one study using a cognitive

behavioral therapy program to treat hopelessness provides some evidence for the usefulness of this method for suicide prevention (Simpson, Tate, Whiting, & Cotter, 2011). Bombardier et al. (2017) also noted preliminary evidence for improvement in depressive symptoms when TBI patients were engaged in meaningful, enjoyable social and occupational roles as measured by a questionnaire, the Environmental Reward Observation Scale (Armento & Hopko, 2007). Physical exercise in a recent meta-analysis supports its use as a treatment paradigm in reducing depressive symptoms in TBI patients (Perry, Coetzer, & Saville, 2018). Comprehensive neuropsychological rehabilitation programs which provide psychotherapy and cognitive remediation help to decrease the symptoms of depression and anxiety in TBI patients, as has been demonstrated in a single-blind randomized controlled study (Tiersky et al., 2005). This topic is discussed in depth in the chapter on counseling patients with brain injury.

As noted in the discussion regarding suicide and TBI, the issue of inflammation as a pathogenic etiology for depression has gained more attention by researchers in the field. This is relevant for treatment as neuroimmune modulation may have a role in the treatment of neuropsychiatric manifestations of TBI (Bodnar et al., 2018).

Vignette

A 53-year-old female was found unconscious at the bottom of a staircase in her home. In the emergency department, a head computerized tomography (CT) revealed an epidural hematoma of the right frontal-temporal region as well as bilateral frontal contusions. Several months later, during her rehabilitation at a TBI unit, she was noted by the therapists working with her to have frequent crying spells and to be despondent over her cognitive deficits. During sessions, she was often amotivated, displayed poor self-esteem, and complained of low energy and difficulty with concentration.

After neuropsychiatric assessment, our patient was diagnosed with depression secondary to TBI, as the rest of the medical workup was negative. She was treated with an SSRI with improvement of her tearfulness, overall mood, and a notable increase in her participation in her rehabilitation classes.

Mania

After TBI, patients can experience an elevated mood state which is referred to as mania. In DSM-5 (American Psychiatric Association, 2013) nosology, this is defined as an elevated, expansive, or irritable mood which lasts at least 1 week. When the patient has an elevated mood, three of the following symptoms must also be present to make the diagnosis of mania, while when an irritable mood predominates, four additional symptoms are required. These include inflated self-esteem,

decreased sleep, pressured speech, racing thoughts, poor attention, an increase in goal-related activity, and excessive involvement in pleasurable activities (e.g., spending sprees or sexual indiscretions), which could have painful repercussions. DSM-5 (American Psychiatric Association, 2013) diagnostic categorization would classify such a patient as having bipolar I disorder. However, patients suffering from various medical conditions such as TBI can have an expansive or irritable mood and yet not meet the full criteria for a manic episode or bipolar disorder. In such a case, bipolar and related disorder due to another medical condition would be the diagnosis given. Secondary mania is a concept similar to the preceding one and was first described by Krauthammer and Klerman (1978), who observed patients without previous psychiatric history who developed a psychotic disorder after a medical illness. Their definition of this syndrome included an elated or irritable mood in addition to only two of the above-listed criteria.

Epidemiology

The occurrence of mania after TBI has been estimated to be far less frequent than depression, in the range of 1.6–9% (Jorge et al., 1993; Silver, Kramer, Greenwald, & Weissman, 2001). A study of 60 patients followed 30 years post TBI revealed only one patient (1.7%) with a diagnosis of bipolar II disorder (Koponen et al., 2002), which by definition is an episode of depression and, at some time, an episode of hypomania (same symptoms as mania, but the duration is at least 4 days but less than 1 week) (American Psychiatric Association, 2013). Although it is less likely to occur, some TBI patients have also been observed to experience rapid-cycling bipolar disorder in which at least four manic, hypomanic, or depressive episodes occur within 12 months (Monji, Yoshida, Koga, Tashiro, & Tashiro, 1999; Murai & Fujimoto, 2003). Anatomically, an association has been made in TBI patients suffering from symptoms of mania and lesions which occur mainly in the right basotemporal or orbitofrontal regions (Jorge et al., 1993; Starkstein, Boston, & Robinson, 1988). However, several new case studies have noted TBI patients suffering manic episodes after left frontal or bilateral temporal lesions (Heinrich & Junig, 2004; Mustafa, Evrim, & Sari, 2005). There is also an inverse relationship that has been reported in some studies noting that a diagnosis of bipolar disorder increases the subsequent risk of incurring a TBI, which may be due to impulsive behavior, self-harm, homelessness, comorbid substance abuse, or the side-effects of medications used to treat bipolar illness (Bacciardi et al., 2017; Huang, Su, et al, 2018). Conversely, a recent study examining the clinical characteristics of patients suffering from bipolar illness found that 37 of 505 individuals who had a premorbid TBI history before the onset of affective symptoms were more likely to have a comorbid diagnosis of migraine and to have higher scores on a mania rating scale for disruptive symptoms (Drange et al., 2018).

Assessment

The clinician's observations and findings after taking a careful history and neuropsychiatric evaluation (as outlined in the previous section on depression in this chapter) will guide the ordering of appropriate tests such as brain imaging, lab tests, or EEG to further clarify the diagnosis.

Obtaining a list of medications, including over-the-counter preparations is essential, since agents such as antidepressants, steroids, and herbal preparations such as St. John's Wort and ginkgo biloba can precipitate manic symptoms, as was reported in a recent case study of a TBI patient (Spinella & Eaton, 2002). A recent example of this issue is a case report of a 17-year-old male who had sustained a TBI in a motor vehicle accident with damage to the right frontal lobe, left temporal lobe, and corpus callosum and was prescribed methylphenidate when he subsequently developed attentional problems. Manic symptoms of grandiose delusions with religious content, irritable mood, and sleeplessness subsequently occurred after 1 week of treatment with this stimulant (Ekinci, Direk, Ekinci, & Okuyaz, 2016). The clinician's observations and findings after taking a careful history and neuropsychiatric evaluation (as outlined in the previous section on depression) will guide the ordering of appropriate tests such as brain imaging, lab tests, or EEG to further clarify the diagnosis.

Differential Diagnosis

When obtaining the history, the clinician should consider other medical conditions (in addition to TBI) which could present with the symptoms of mania, such as epilepsy, brain tumors, central nervous system infections, thyroid disease, renal disease, and vitamin deficiencies. Other psychiatric illnesses to be screened for include substance abuse, since manic symptoms can be observed in patients who use opioids, hallucinogens, and cocaine. Diagnoses such as borderline or antisocial personality disorders also need to be considered since overlapping features of these syndromes such as irritability, aggressiveness, and impulsivity can mimic the manic state (Sadock et al., 2015).

Treatment

With the completion of this workup, the neuropsychiatrist is left with the decision of choosing an appropriate agent to treat the manic symptoms experienced by TBI patients. The literature in this area is sparse as there are no double-blind randomized placebo-controlled studies. Instead, clinical judgment is guided by the treatment of classical bipolar disorder and case reports of patients with TBI (Kennedy, Burnett,

& Greenwald, 2001). Anticonvulsants such as valproic acid (Depakote) and carbamazepine (Tegretol) have been used successfully in the treatment of the TBI manic patient and may be particularly good options if there is the presence of a seizure disorder as well (Kim & Humaran, 2002; Monji et al., 1999; Pope et al., 1988; Sayal, Ford, & Pipe, 2000; Stewart & Hemsath, 1988). Side effects of the former agent include weight gain, thrombocytopenia (low platelet count), and hepatic dysfunction while the latter can cause hyponatremia (low serum sodium) and hematological dysfunction. Other anticonvulsants such as lamotrigine (Lamictal) and topiramate (Topamax) have also been shown to successfully treat bipolar disorder, but there have been no studies which have used these agents in patients with TBI and mania (Calabrese, Bowden, McElroy, & Cookson, 1999; Marcotte, 1998). Side effects of lamotrigine include dizziness, sedation, ataxia, and most importantly, rash, which can then evolve into the potentially fatal condition known as Stevens–Johnson syndrome. Adverse effects of topiramate include sedation, decreased appetite, speech disorders, and cognitive impairment. Gabapentin (Neurontin) may also be a reasonable choice to treat manic symptoms based on its use in agitation with other types of neurologically impaired patients (Roane et al., 2000). However, as with the other pharmacological agents already described, there are no controlled studies of its use with TBI patients. Common side effects of gabapentin include fatigue and dizziness. Although the mood stabilizer lithium (Eskalith) is used in classical bipolar disorder with much success and has been recommended for mania in relation to TBI, much caution must be utilized with this agent, as it can cause neurological side effects such as tremor, ataxia, and confusion, which can worsen the condition of a TBI patient (Hale & Donaldson, 1982). In a placebo-controlled trial of one patient who suffered bilateral orbitofrontal and right temporal–parietal contusions, clonidine, an alpha-adrenergic receptor agonist which reduces the firing rate of noradrenergic neurons, successfully reversed the patient's manic symptoms (Bakchine et al., 1989). Side effects to monitor include dry mouth and eyes, sedation, hypotension, and constipation. The FDA (Food and Drug Administration) has approved the use of some second-generation antipsychotic medications (described in more detail in the section on psychosis in this chapter) for the treatment of bipolar disease. There are a few case reports in which medications from this class such as quetiapine and olanzapine have been used to treat mania due to TBI. These two medications have been cited as adjunctive therapy to antiepileptic drugs such as valproic acid and carbamazepine (Daniels & Felde, 2008; Oster, Anderson, Filley, Wortzel, & Aciniegas, 2007; Zincir, Izci, & Acar, 2014), and more recently olanzapine was used as monotherapy to treat a patient with mild TBI who developed manic symptoms 5 months after his injury (Cittolin-Santos, Fredeen, & Cotes, 2017). Ariprazole, another second-generation antipsychotic was used successfully as an adjunctive treatment to lithium for a 45-year-old delusional, hypersexual man with a history of bipolar disease and bifrontal encephalomalacia sustained after a prior fall 15 years before (Moreira, Khemiri, & Runeson, 2011). ECT is another treatment modality which may be beneficial in patients with manic symptoms after TBI, as was reported in another patient where medications were ineffective (Clark & Davison, 1987). An important treatment-related caveat is that patients with TBI who

experience affective symptoms or exhibit behavioral difficulties are often treated with antidepressants. In certain predisposed individuals, these medications can then induce manic symptoms, so caution must be exercised (Handel, Ovitt, Spiro, & Rao, 2007; Ummar, Kumar, & Ramanathan, 2016).

Vignette

A 76-year-old male falls outside his internist's office and loses consciousness. The patient is rushed to the emergency room where a head CT reveals a right-frontal–temporal–parietal hemorrhage. A craniotomy is performed to evacuate the hemorrhage, and, postoperatively, the patient does well. At home, a few weeks later, he becomes agitated and accuses his wife of having affairs with several men in their apartment building. His wife noted that prior to this incident he had not been sleeping well for several nights. The patient is loud, irritable, and argumentative, and his speech is difficult to interrupt. His thoughts race from one topic to another, and he grandiosely proclaims to anyone who will listen that his doctor actually hit him over the head to cause the head injury to rob his money.

After a neuropsychiatric evaluation, the patient was treated with valproic acid, which resulted in a decrease in his irritability and agitation.

Anxiety

Anxiety refers to a state of apprehension, uneasiness, or dread that occurs in anticipation of either internal or external threats which are perceived as unpredictable or uncontrollable. The subcategories of anxiety in the DSM-5 (American Psychiatric Association, 2013) include panic disorder (PD), generalized anxiety disorder (GAD), and social phobia. When features of anxiety syndromes are present secondary to medical conditions such as in TBI, then anxiety disorder due to another medical condition is the DSM-5 diagnosis to be used, as often the patient may have characteristics of several different types of anxiety syndromes present simultaneously (Hiott & Labbate, 2002). Since anxiety has a tremendous impact on the social and occupational functioning of patients after TBI, it is important to understand the diagnostic issues involved, and thus each subcategory of anxiety will be described.

Panic attacks are defined as discrete periods of intense fear which develop abruptly and reach a peak within 10 min with the presence of at least four of the following 13 symptoms: palpitations, sweating, trembling, shortness of breath, the sensation of choking, chest pain, abdominal discomfort, dizziness, derealization, chills, paresthesia, and fear of loss of control, or death. To qualify for a diagnosis of PD, the patient must have recurrent panic attacks and be worried about having further episodes or be concerned about the consequences of an attack for at least 1 month after the initial panic attack. In some patients, the fear of having a panic

attack in a situation or place from which they cannot escape creates marked discomfort and avoidant behavior known as agoraphobia (American Psychiatric Association, 2013).

GAD is a syndrome marked by excessive worry and anxiety about a number of issues that occur almost daily for at least 6 months. The patient is unable to control the worry and experiences at least three of six somatic symptoms which include restlessness, being easily fatigued, diminished concentration, irritability, muscle tension, or sleep disturbance (American Psychiatric Association, 2013). The symptoms of anxiety experienced by patients with TBI are usually attributed to the loss that patients feel in terms of their independence as well as the relation to their prior level of high functioning and can compromise rehabilitative efforts if not addressed.

Social phobia is defined as marked fear or anxiety about one or more social situations in which the individual is exposed to possible scrutiny by others, whereas specific phobia refers to fear about a specific object or situation. In both cases, the fear or anxiety is persistent lasting 6 months or more and is out of proportion to the actual danger or threat posed by the circumstance, and the individual typically avoids the object or situation (American Psychiatric Association, 2013).

Epidemiology

As with other epidemiological studies on TBI, the prevalence rates regarding anxiety are variable due to the population studied and the definition of the syndrome utilized; in some studies which use self-report questionnaires, there is a more broad conception focused on physical symptoms such as tenseness or cognitive issues such as rumination and worry. In those studies, over-reporting of symptoms can occur, and some of the identified symptoms may be better explained by the TBI itself rather than anxiety. Some recent research has attempted to review studies of anxiety in the TBI literature to assess overall pooled anxiety prevalence rates, and these have been reported as between 37% and 38% (Osborn, Mathias, & Fairweather-Schmidt, 2016; Scholten et al., 2016). Some risk factors for the development of anxiety identified in studies were female sex, unemployment, those with prior psychiatric history before their TBI (Scholten et al., 2016) and black race, more than one TBI, and low socioeconomic status (Hart et al., 2016). In earlier studies which reported a diagnosis of GAD, the rates have been reported as low as 1.7% to as high as 44% (Fann, Katon, Uomoto, & Esselman, 1995; Hibbard, Uysal, Kepler, Bogdany, & Silver, 1998; Hoofien, Gilboa, Vakil, & Donovick, 2001; Koponen et al., 2002; Van Reekum, Bolago, Finlayson, Garner, & Links, 1996). More recent epidemiological studies have tried to hone the prevalence figures using structured clinical interviews. In a prospective study of mild TBI patients who were followed for 12 months after their injury, 9% were newly diagnosed with GAD (Bryant et al., 2010), while a retrospective study of 100 patients with TBI, which ranged from mild to severe and who were from 6 months to 5 years out from their injuries, revealed 17% had a diagnosis of GAD (Whelan-Goodinson et al., 2009). In a longer 5-year

prospective study of moderate-to-severe TBI patients, the rate of GAD was 3%, while in a 6-year study of severe TBI patients, the rate was 6% (Alway, Gould, Johnston, et al., 2016; O'Donnell et al., 2016).

There are a limited number of studies where TBI and PD have been evaluated, and often this anxiety diagnosis is comorbid with depression and alcohol dependence, as was observed by Deb and colleagues (Deb, Lyons, Koutzoukis, Ali, & McCarthy, 1999), who reported a 9% rate of PD in 120 patients aged 18–64 years 1 year after TBI. In several other epidemiological studies which examined the frequency of anxiety diagnoses in patients with a TBI history, the rates of PD have been reported as 3.2%, 8.3%, and 11%, respectively, depending on whether lifetime prevalence (former) or post-TBI onset (latter two) was recorded (Hibbard et al., 1998; Koponen et al., 2002; Silver et al., 2001). The recent prospective study by Alway, Gould, Johnston, et al. (2016), recorded a rate of 2.0–3.3% for this disorder, which was similar to the finding of Bryant et al. (2010) who reported a new diagnosis of PD after TBI to be 3.5%.

Although there are fewer reports in the TBI literature regarding social and specific phobias, recent epidemiological research of the former diagnosis cites rates of 4.5% (Gould, Ponsford, & Spitz, 2014), 5% (Bryant et al., 2010), and 6% (Whelan-Goodinson et al., 2009), while the latter disorder has been reported as 7% in the study by Whelan-Goodinson et al. (2009) and 6.1% by Gould et al. (2014). A salient point to bear in mind in TBI patients with social anxiety is the fact that they may be avoiding social engagement with others due to cognitive deficits which impinge on their level of confidence (Mallya, Sutherland, Pongracic, Mainland, & Ornstein, 2015). An example of specific phobia is that of fear of falling which can interfere with rehabilitation efforts (Collicutt McGrath, 2008).

Neuroanatomically, the brain stem, limbic system, basal ganglia, and prefrontal cortex have been implicated in the etiology of anxiety likely due to the compromise of the neurocircuitry involved in the emotional regulation of responses to environmental stimuli which can provoke anxiety (Rauch, Shin, & Phelps, 2006; Scheutzow & Wiercisiewski, 1999). An increased risk for the development of an anxiety disorder after TBI has also been linked with cognitive impairment; in one study, deficits in attention, working memory, information processing, and executive function, especially processing speed, were significantly associated with anxiety disorders (Gould et al., 2014). The unifying hypothesis connecting the later development of anxiety with cognitive deficits which occur soon after TBI may also be explained by damage to frontolimbic systems. Etiologically, although the precise mechanism of anxiety is not known, inflammation is thought to play a role. In non-TBI individuals who have been diagnosed with PD and also PTSD (see below), proinflammatory cytokines which are proteins involved in mediating immunological responses to injury, stress and infection, have been demonstrated to be elevated (Hoge et al., 2009). An example of a possible role of inflammation in TBI patients with anxiety was reported in one study examining 3822 patients with TBI who were age- and gender-matched and had a diagnosis of pre-existing hyperlipidemia; the latter has been associated with markers of systemic inflammation and dysfunction of the endothelium of blood vessels. Hyperlipidemia was found to be an independent risk

factor for new-onset anxiety disorders in TBI patients with a 1.60-fold incidence rate compared to TBI patients without hyperlipidemia (Ho et al., 2014). Childhood traumatic brain injury may also be a risk factor for anxiety as reported in a recent study examining the long-term psychiatric outcomes of patients who were at least 5 years post injury; as compared to controls, those with orthopedic injuries, were found to have higher rates of anxiety, specific phobias, and panic disorders even as long as 13 years post-injury (Albicini & McKinlay, 2018).

Assessment and Differential Diagnosis

During the evaluation of a patient with symptoms of anxiety, the neuropsychiatrist first obtains a thorough history from the patient and his family about the various situations in which apprehension is experienced, any pattern of avoidance behavior, and accompanying physical symptoms of anxiety such as those listed above in the descriptions of anxiety disorders. As there are many medical imposters of anxiety, such as cardiac, pulmonary, and endocrinological disorders, the neuropsychiatrist must differentiate between these diagnostic challenges. In this population of patients with TBI, seizures are a particular concern for the clinician, since the presentation of seizures can mimic anxiety syndromes. For example, during ictus, intense fear and dread can be the sole expression of a simple partial seizure or the aura of a complex partial seizure (Scicutella, 2001). From the standpoint of the psychiatric differential diagnosis, the clinician must consider that the patient is suffering from more than one anxiety disorder or depression. In GAD and PTSD, since there is the presence of autonomic hyperarousal, the clinician must consider the possibility of the abuse of stimulants or withdrawal from alcohol and sedatives. After a careful neuropsychiatric evaluation, the physician may also need to perform laboratory tests, including a CBC, metabolic studies, an endocrinological screen, EKG, EEG, and brain imaging, if warranted, to rule out the medical etiologies of anxiety (Sadock et al., 2015).

Treatment

Once the neuropsychiatrist has determined the type of anxiety that the patient is suffering from, the issue of treatment must be addressed. Because no randomized placebo-controlled studies of anxiety disorders in TBI patients have been done, the general pharmacological principles for treating anxiety disorders in patients without neurological compromise are used, with attention to dosing regimens, side effect profiles, and drug–drug interactions. The TCAs and SSRIs have been shown to be efficacious in the treatment of anxiety disorders (Janicak, Davis, Preskorn, & Ayd, 1993). In the case reports of patients who have suffered TBI and anxiety, the successful use of SSRIs such as sertraline in the treatment of panic attacks

(Scheutzow & Wiercisiewski, 1999) has been demonstrated. A case report of an 18-year-old male who sustained a minor head injury developed social anxiety disorder 1 year later; brain imaging revealed a rare benign reactive process known as intradiploic hematoma that compressed the left frontal lobe. After neurosurgical decompression, his residual symptoms were treated with citalopram with improvement (Chaves et al., 2012). Venlafaxine has also been shown to be effective in the treatment of GAD (Derivan, Haskins, Rudolph, Pallay, & Aguiar, 1998). The side effects of these medications have been reviewed previously. Additionally, TBI patients with GAD may respond to the treatment with buspirone (Buspar), a partial serotonin (1A) agonist, whose side effects include nausea, dry mouth, dizziness, and nervousness (Gualtieri, 1991a, 1991b). Propranolol (Inderal), a beta-blocker, which reduces adrenergic receptor activation, can also be utilized in treating patients with GAD; its adverse reactions include weakness, hypotension, nausea, and depression (Emilien & Maloteaux, 1998). The BZD class of medications, of which lorazepam (Ativan) is an example, can be useful for treating PD and GAD, but the potential for tolerance, dependence, sedation, ataxia, memory disturbances, and occasional paradoxical disinhibition makes this class less attractive for treating patients with TBI (Spier, Tesar, Rosenbaum, & Woods, 1986). On occasions, antiepileptic drugs have been used to treat anxiety, but these are not first-line treatments, and there are no studies using these agents in TBI patients specifically. For example, valproic acid has been used to treat PD (Woodman & Noyes, 1994). Cognitive behavior therapy has been beneficial in the treatment of GAD, PD, and specific phobias (Arch & Craske, 2009), but how many sessions are needed to be effective has not been studied in a rigorous research paradigm (Waldron, Casserly, & O'Sullivan, 2013). This is likely due to the fact that depending on the severity of TBI, cognitive prowess of the individual can be a factor limiting the benefit of the treatment. Other promising therapies for these disorders are acceptance and commitment therapy (the practice of awareness of one's thoughts and somatic sensations to reduce fear-related avoidant behaviors) and mindfulness training, but no studies have been performed to validate these approaches in the TBI population (Mallya et al., 2015).

Vignette

A 70-year-old man fell off a 10-ft ladder while working at home and sustained a right temporal hemorrhagic contusion. A few months later, his family notes that he cannot stay in a closed room for any length of time. He becomes shaky, restless, and short of breath and needs to get out of the room urgently or he becomes agitated and will yell at his family. He also reports excessive worry about whether his grandchildren are safe, and he fears that they may hurt themselves.

The patient is referred to the neuropsychiatrist for assessment, and due to the symptoms of both PD and GAD, he was treated with a member of the SSRI class with some improvement of his symptoms.

Obsessive–Compulsive Disorder and Related Disorders

In the updated DSM-5 nomenclature (American Psychiatric Association, 2013), obsessive–compulsive disorder (OCD) and related disorders due to another medical condition were classified separately from the anxiety disorders discussed in the previous section due to the fact that these disorders appear to share the aspects of phenomenology, comorbidity, neurochemistry, and neuroanatomy as well as genetics and treatment responses which differ from anxiety syndromes (Hollander, Braun, & Simeon, 2008). OCD is characterized by recurrent obsessions or compulsions or both, which are time-consuming (more than 1 h/day) and cause distress in social, occupational, or other areas of function. Obsessions are defined by recurrent and persistent thoughts, urges, or images that are experienced as intrusive and unwanted and which an individual attempts to ignore, suppress, or neutralize via performance of compulsions. The latter are repetitive behaviors (checking, ordering) or mental acts (praying, counting) that the patient feels driven to perform excessively to reduce the distress of the obsession. This can be a challenging diagnosis in TBI patients since this population frequently suffers from cognitive problems including memory issues. For this reason, there is some debate in the literature that the checking behaviors are actually compensatory strategies to prevent forgetting something the patient deems important to remember, or the perseverative acts are due to a dysexecutive function secondary to frontal lobe damage as a result of the accident (Coetzer, 2011; Rydon-Grange & Coetzer, 2015). In contrast, in patients without brain injury, the observed repetitive behaviors have no rational relationship to being able to eliminate the unwanted thoughts or urges.

Epidemiological studies have reported rates of OCD from as low as 4.7% to as high as 14% in patients who had sustained TBI (Hibbard et al., 1998; Silver et al., 2001). In a more recent prospective study of 161 individuals with moderate to severe brain injury, and who were predominantly male (78.3%), the diagnosis of OCD was relatively uncommon over the 5-year period studied (0.0–2.6%) (Alway, Gould, Johnston, et al., 2016). Patients can experience obsessions and compulsions during the early postacute phase of injury or years later, as noted in the TBI case reports (Arauco, Grados, & Vizcarra, 2008; Coetzer, 2011; Williams, Evans, & Fleminger, 2003). Anatomically, the etiology of OCD has been linked to the disruption of cortico-striatal-thalamo-cortical circuits, and the TBI case reports of patients with these symptoms have provided additional support for this hypothesis (Baxter et al., 1992; Grados, 2003; Maia, Cooney, & Peterson, 2008). A study of 10 patients with TBI and OCD observed that they exhibited a high frequency of obsessions which involved contamination and sexual themes as well as the need for symmetry and exactness. In addition to compulsive exercising, these patients also displayed cleaning/washing, checking, and repeating compulsions. Comorbid psychiatric diagnoses such as depression and other anxiety disorders were common, while on neuropsychological testing, these patients showed poor performance on general intelligence, attention, learning, memory, word retrieval, and executive functions (Berthier, Kulisevsky, Gironell, & Lopez, 2001).

Assessment

As with the assessment of TBI patients with anxiety symptoms due to other etiologies, the history and physical exam and laboratory data would need to be obtained. In this patient population with TBI, OCD symptoms such as perseverative thoughts (forced thinking) can be experienced as the aura of a seizure (Scicutella, 2001). Therefore, an EEG should be considered as part of the evaluation. The differential diagnosis can include a personality disorder such as obsessive–compulsive personality disorder in which preoccupation with details or inflexibility about task completion may overlap with OCD. In one Finnish study of 38 TBI patients, 10.5% were given this diagnosis (Koponen, Taiminen, Hiekkanen, & Tenovuo, 2011).

Treatment

In case reports of patients who have suffered TBI and anxiety, the successful use of SSRIs such as fluoxetine to treat OCD (Hofer et al., 2013; Stengler-Wenzke & Muller, 2002) has been demonstrated. Venlafaxine produced almost complete remission of compulsions in one patient with OCD after an epidural hematoma (Khouzam & Donnelly, 1998), and the antiepileptic drug carbamazepine was successfully used to treat OCD (Koopowitz & Berk, 1997). Also important in the treatment paradigm for patients with obsessions and compulsions is the use of cognitive behavioral strategies focused on awareness training and self-regulation, employing exposure and response prevention techniques, goal setting, and coping strategies, as has been shown in several case studies of TBI patients and OCD symptoms (Arco, 2008; Hofer et al., 2013; Williams et al., 2003).

PTSD

As with OCD, post-traumatic stress disorder was also categorized separately from other anxiety disorders in DSM-5 (American Psychiatric Association, 2013). The criteria for this disorder include exposure to actual or threatened death, serious injury or sexual violence, and the presence of one or more intrusion symptoms such as distressing memories or dreams of the event, and dissociative reactions such as flashbacks or physiological or psychological reactions to external or internal cues which resemble the aspects of the event. In addition, there is avoidance of stimuli associated with the event as well as negative alterations in cognitions and mood and marked alterations in arousal such as hypervigilance or exaggerated startle response; the disturbances are greater than 1 month in duration (American Psychiatric Association, 2013). A similar entity is acute stress disorder in which diagnostic criteria overlap with PTSD, but the duration of symptoms is 3 days to 1 month

(American Psychiatric Association, 2013). However, unlike other DSM-5 diagnoses, there is no PTSD or related disorder due to another medical condition. The overlap of symptoms of these two entities has an impact on treatment, since anxiety, irritable mood, cognitive, and sleep issues can be experienced in both disorders; thus, being able to more reliably differentiate them is desirable from a treatment perspective (Hendrickson, Schindler, & Pagulayan, 2018; Vasterling, Jacob, & Rasmusson, 2018).

In relation to TBI, PTSD is an entity about which there has been much debate, since one of the essential criteria is that the patient who has been exposed to a threatening event must display re-experiencing symptoms, such as intrusive memories or distress when reminded about the particular trauma, recurrent dreams of the event, or the feeling that the trauma is recurring. Given that many TBI patients do not recall the event due to post-traumatic amnesia, which is a short interval after injury during which the capacity to store and retrieve new information is lacking, one can argue that theoretically PTSD cannot occur in these patients. In an older study by Warden and colleagues who reviewed 47 active-duty service members who had sustained moderate TBI with amnesia for the event and in which strict PTSD criteria were used, none of those patients qualified for the diagnosis, since no patient reported re-experiencing phenomena. However, when that part of the criteria was eliminated, then six patients (12.7%) received a diagnosis of PTSD (Warden et al., 1997). In 100 patients involved in traffic accidents who sustained head injury with a definite loss of consciousness, 48% reported PTSD at 3 months after the incident, and 33% suffered with this disorder 1 year later (Mayou, Black, & Bryant, 2000). In a recent 4-year prospective study of 85 moderate-to-severe TBI patients who had event-related amnesia, 17.6% still developed PTSD with vivid re-experiencing phenomenon. This study also looked at the subthreshold variants of PTSD which then escalated the statistics of a PTSD diagnosis to 22.4% of their study population (Alway, Gould, McKay, et al., 2016).

Some mechanisms to explain PTSD when there is a lack of recall of the traumatic event itself include: (1) recall of other distressing experiences associated with the event which occurred either before or after the period of amnesia that then serves as the "trauma"; (2) traumatic experiences may be processed by the limbic area of the brain at an implicit level outside awareness; (3) if loss of consciousness and post-traumatic amnesia are brief, there may be a partial or full encoding of the event; (4) learning of the traumatic event as told by others helps the patient to reconstruct the memory (Bontke, Rattok, & Boake, 1996; Bryant, 2001; McNeil & Greenwood, 1996).

Epidemiologically, there is a vast array of reported rates of PTSD in relation to TBI which depends on several factors, namely whether the studied population is civilian or military, what degree of TBI (mild/moderate/severe) has been sustained, and what evaluation tools are utilized. Given these limitations, the reported range of PTSD in civilian populations is as low as 3% for severe TBI to as high as 30% in mild TBI, whereas in military populations, the range is more widespread from 12% to 89% (Bahraini et al., 2014). From a prognostic viewpoint, a trajectory of the severity of the condition has been reported in one prospective study of 1084 patients

(40% of whom had mild TBI) over 6 years of follow-up; 4% maintained chronic PTSD symptoms, while 10% actually suffered worsening symptoms over the course of the study (Bryant et al., 2015). Of note is that two recent epidemiological reports noted similar rates of PTSD despite the length of the study and severity of patients' injuries; in a 12-month study of mild TBI patients, the rate was 7% and in a study evaluated 6 years after severe TBI, the rate was recorded as 6% (Bryant et al., 2010; O'Donnell et al., 2016). Being able to identify factors which place certain individuals at higher risk can be helpful to target treatment as early as possible.

Factors associated with patients suffering from PTSD and TBI include: female gender, prior psychiatric history including anxiety, depression, substance abuse, and having an acute stress disorder soon after the trauma (Bryant, 2011; Cnossen et al., 2017; Scofield, Proctor, Kardouni, Hill, & McKinnon, 2017). Of note, acute stress disorder after mild TBI has ranged from 4.6% to 21.2%; therefore, there is a risk for certain individuals who will transition to PTSD (Ponsford, Alway, & Gould, 2018). In military population, blasts, explosions, and multiple TBIs can also put patients at risk for the concurrent PTSD diagnosis (Lindquist, Love, & Elbogen, 2017).

There are debates in the literature about the exact neuroanatomic pathology that can explain the deficits seen in PTSD, with the hypotheses focusing on damage to the frontal–subcortical systems which include the amygdala, hippocampus, and ventromedial prefrontal cortex, which then create an imbalance in the network of inhibitory and excitatory responses of these structures in dealing with the memory of the events and subsequent fear responses (Bahraini et al., 2014; Rosso, Crowley, Silveri, Rauch, & Jensen, 2017). Additionally, there has been a focus in more recent research to attempt to understand the overlap in the symptoms of TBI and PTSD using more sophisticated imaging techniques as well as genetic studies and neuropsychological profiles to try to decipher the nuances and better define these disorders.

A recent study of 39 patients with mild TBI and either no PTSD or significant PTSD symptoms was performed with brain MRI to assess the volume of regions of interest, diffusion tensor imaging (DTI) to analyze white matter abnormalities, and neuropsychological testing to determine if any patterns could be identified which would differentiate the study groups. Compared to mild TBI patients, those with both mild TBI and PTSD evidenced an enlarged volume of the entorhinal cortex, an area of the brain involved in memory retrieval; in PTSD, this finding could hypothetically contribute to an overactive retrieval of trauma memories and subsequent intrusive thoughts and re-experiencing symptoms. The mild TBI/PTSD group also demonstrated reduced structural integrity of the right cingulum white matter as assessed by DTI. In the normal brain, the right dorsolateral and prefrontal cortices limit the encoding and retrieval of unpleasant memories by the hippocampus, and therefore a damaged right cingulum white matter tract which connects the right frontal brain regions and hippocampus would compromise this normal memory suppression network. Finally, the mild TBI/PTSD group evidenced deficits in their neuropsychological profile compared to the mild TBI group in processing speed as well as encoding and retrieving tasks (Lopez et al., 2017). Another research tool to try to help in the differentiation of PTSD alone versus combined TBI and PTSD is

magnetic resonance spectroscopic imaging which measures the ratio of *N*-acetyl aspartate (NAA) to choline; NAA decreases are found in neuronal loss, and choline has been shown in axonal injuries to be increased. In one study of veterans with blast injuries, the group with dual diagnosis of TBI/PTSD showed a lower ratio of NAA/choline in the left hippocampus, which is implicated in memory compared with the veterans with PTSD alone (Kontos et al., 2017). In another study using functional MRI, patients with mild TBI and more PTSD symptoms compared to patients with mild TBI and fewer PTSD symptoms had lower connectivity of the network involving anterior prefrontal cortex which is involved in the regulation of attention, emotion, and memory coding and connected to the anterior cingulate cortex which is related to emotional symptoms; if the network is damaged, then the disrupted connections can play a role in how memories are appropriately processed and the responses to them (Nathan et al., 2017). Genetics may also have a role in the future of predicting the likelihood of PTSD in the context of TBI. Brain-derived neurotropic factor (BDNF) is important in long-term learning and memory; in some studies, the Met allele of the BDNF gene has been associated with mood disorders and hyperactivity of the amygdala. In one recent study, the Met/Met allele was associated with both sustaining TBI and having traumatic stress symptoms (Dretsch et al., 2016). Neuropsychological profiles in general indicate that the patients with chronic cognitive deficits such as learning and memory, and executive function in those patients with both mild TBI and PTSD symptoms, are usually more related to PTSD, sleep issues, or depression which are more enduring, rather than TBI (Merz, Roskos, Gfeller, & Bucholz, 2017; Vasterling, Brailey, Proctor, Kane, & Heeren, 2012).

Assessment

As with the assessment of TBI patients with anxiety symptoms due to other etiologies, the history and physical exam and laboratory data would need to be obtained. Medical issues such as intoxication with or withdrawal from illicit substances should be considered, as the clinical picture may be similar. In patients with PTSD and TBI, endocrine function should also be evaluated. In one case in the literature, a patient who was suffering from PTSD symptoms including traumatic memories, hypervigilance, and avoidance behaviors was found to have hypopituitarism, and replacement of low testosterone resulted in improvement of his symptoms, which pharmacotherapy and psychotherapy were unable to ameliorate (Isaacs & Geracioti, 2015). Other psychiatric diagnoses such as PD, GAD, or certain personality disorders should be considered in the differential diagnosis as well (Sadock et al., 2015). Of note, the patient experiencing flashbacks may believe that they are not recalling the trauma, but actually re-experiencing it, as they are not able to reality test at the time and can appear psychotic, a symptom the physician needs to differentiate (Meyer & Writer, 2015).

Treatment

In general, there is no robust evidence to support medications in the treatment of PTSD, but they may help to ameliorate some symptoms of depression and panic. Antidepressants such as SSRIs or serotonin and norepinephrine reuptake inhibitors (SNRIs) such as venlafaxine are usually the first choice in this population (Hoskins et al., 2015). Other options for treatment can include the antidepressant mirtazapine which blocks serotonin at lower levels of the medication and at higher doses blocks the reuptake of norepinephrine. Side effects can include weight gain and drowsiness (Schneier et al., 2015). Prazosin which is a noradrenergic alpha-1 receptor inhibitor can be useful if there are sleep problems and associated nightmares due to the trauma episode. Clonidine and propranolol which can modulate catecholamine transmission may also help to diminish the hyperarousal symptoms of PTSD (Hendrickson & Raskind, 2016). MAO-I antidepressants have been of benefit in treating PTSD in patients without TBI. In addition to the potential for a hypertensive crisis, as discussed earlier, more common side effects of these medications include orthostatic hypotension, edema, weight gain, insomnia, and sexual dysfunction (Sheehan, Ballenger, & Jacobsen, 1980). On occasion, antiepileptic drugs have been used to treat anxiety, but these are not first-line treatments, and there are no studies using these agents in TBI patients specifically. For example, studies with lamotrigine (Hertzberg et al., 1999) and gabapentin (Hamner, Brodrick, & Labbate, 2001) have indicated some benefit in those patients suffering from PTSD. Side effects of these agents have been discussed previously. The neuropsychiatrist should also emphasize the beneficial role of psychotherapy, in particular CBT, biofeedback, and support groups for TBI patients with anxiety to help them to better cope with their symptoms (Holland, Witty, Lawler, & Lanzisera, 1999; Mallya et al., 2015; Vasterling et al., 2018).

Psychosis

Psychosis is defined as the inability to distinguish reality from fantasy; or to put it another way, the psychotic patient demonstrates impaired reality testing. Clinically, one can observe that patients have a thought disorder, or they may experience perceptual disturbances such as hallucinations, delusions, or paranoid ideation (Ardila, 2018; Sadock et al., 2015). In DSM-5 (American Psychiatric Association, 2013), patients suffering from these symptoms after TBI and who are experiencing distress and impairment in their ability to function because of these disturbances would be given a diagnosis of psychotic disorder due to another medical condition. Psychosis can be a feature of a variety of other psychiatric disorders, including delirium and schizophrenia, which are particularly germane to a discussion of head trauma, as will be discussed below.

Due to the methodological problems in the research of psychosis and TBI, including the type of population of patients used in the studies (e.g., adults, children, open or closed head injuries) as well as the lack of standardized diagnostic criteria and variable periods of follow-up, it is difficult to assess the precise incidence and prevalence rates for psychosis and TBI (Arciniegas, Harris, & Brousseau, 2003). An often-quoted study is that of Davison and Bagley, who in 1969 reviewed medical reports published between 1917 and 1960 and recorded that the rates of psychosis in these studies ranged from 0.07% to 9.8% (Davison & Bagley, 1969). Of interest are the disparate rates of psychosis recorded in two studies where follow-up had been lengthy; in a 10–15-year study, a rate of 20% was noted and in a longer 30-year study, 1.7% was recorded (Koponen et al., 2002; Thomsen, 1984). A more recent review reported that the incidence of psychotic symptoms is between 3% and 8% (Bhalerao et al., 2013). There is no absolute correlation with regard to TBI and the onset of psychosis. In one case report of a 13-year-old boy who sustained a head injury at age 11 and developed psychosis 11 months later, there was a clear temporal relation to the symptoms (Bennouna-Greene, Frank, Kremer, Bursztejn, & Foucher, 2010), whereas long latency periods where the psychosis first develops 10 years or more after TBI have been reported in war injuries and in a recent report of a middle-aged patient with first onset psychosis 19 years after his motor cycle accident in the context of polysubstance abuse (Achte, Hillbom, & Aalberg, 1969; Sami, Piggott, Coysh, & Fialho, 2015).

Risk factors predictive for the development of psychosis in TBI patients include premorbid neurological or neurodevelopmental disorders as well as having sustained a head injury before adolescence (Fujii & Ahmed, 2001). A family history of psychosis in first-degree relatives and duration of loss of consciousness were also significantly associated with psychosis post TBI (Sachdev, Smith, & Cathcart, 2001). In a review of 69 published case studies of psychosis after TBI, certain features emerge which appear to be typical for this phenomenon: (1) it is more commonly observed in males; (2) persecutory or paranoid delusions are the most common type of psychotic symptoms, but auditory hallucinations are also frequently observed; (3) approximately 50% of patients demonstrate symptoms before the second year after TBI, while about 75% evidence psychosis within the first 4 years after TBI; and (4) abnormalities as recorded by EEG were most commonly temporal slowing, whereas brain imaging demonstrated frontal lobe lesions most often, but temporal lobe lesions were also observed (Fujii & Ahmed, 2002). Cognitively, patients with TBI and psychosis demonstrate impairments on neuropsychological testing in general intelligence, verbal memory, executive function, and vocabulary (Fujii, Ahmed, & Hishinuma, 2004). A more recent report compared TBI patients with psychosis to three control groups (TBI without psychosis, schizophrenia patients, and healthy controls) using a standardized instrument, the Repeatable Battery for Assessment of Neuropsychological Status (RBANS) (Randolph, Tierney, Mohr, & Chase, 1998), which assesses attention, language, visuospatial, and immediate and delayed memory. The results indicated that 70% of those patients suffering TBI and psychosis demonstrated significant impairment, scoring extremely low or low average compared to controls validating prior research (Batty et al., 2016).

In an updated analysis of their prior study, these authors reviewed 64 cases in the literature of TBI and psychosis that were recorded after the year 2000 to replicate the prior findings and to try to differentiate this entity from schizophrenia. Important findings include: (1) TBI psychosis can occur with both mild and moderate–severe TBI; (2) a bimodal distribution was noted in that 38% developed psychosis within the first year after TBI, whereas 36% developed this problem after 5 years; (3) seizure disorder was found to be more common in TBI patients with psychosis (23.6%) as compared to 7% of patients with TBI alone; (4) as in the prior study, male gender and family history were significant risk factors in the development of TBI psychosis; (5) 77% of patients manifested persecutory delusions and 92% had auditory hallucinations, whereas only 37% demonstrated negative symptoms (avolition, apathy, and poverty of speech/thought); and (6) EEG and brain imaging demonstrated focal abnormalities in the frontal and temporal areas. This study highlighted that patients with schizophrenia have a different pattern of neuropathology with global cortical atrophy, enlarged ventricles, hippocampal atrophy, and EEGs show general slowing; clinically there is a greater likelihood of negative symptoms and more global cognitive deficits in this population (Fujii & Fujii, 2012).

Assessment

During the assessment of the patient with psychosis, the neuropsychiatrist once again explores the recent TBI incident and the course of events during the acute hospitalization, including episodes of delirium, the latter of which is a period of acute disturbance in consciousness marked by attentional and cognitive deficits, as well as perceptual disturbances such as delusions or hallucinations (American Psychiatric Association, 2013). The patient's medical history is reviewed for other potential etiologies of psychosis such as prior head injuries, infections, vitamin deficiencies, metabolic disease, strokes, or tumors. Particularly relevant in this differential diagnosis is post-traumatic epilepsy, which is often observed as a sequelae of TBI. Moreover, a frequent complication of temporal lobe epilepsy is psychosis, which can occur prior to (aura), during (ictally), or after the seizure (postictally, either peri-ictally or interictally) (Trimble, 1991). The patient's medications should be reviewed since some agents such as steroids and anticholinergic drugs (e.g., tricyclic antidepressants) can cause psychotic symptoms. A recent case report documented an unusual clinical scenario of precipitated psychosis in two TBI patients treated with higher doses of bupropion, indicating that at risk patients can have unexpected manifestations of medications (Barman, Kumar, Pagadala, & Detweiler, 2017).

Psychiatric diagnoses to consider in the psychotic patient with TBI include substance or alcohol abuse/dependence which warrants highlighting as several case reports in recent years document the development of psychosis in patients with TBI in the context of cannabis use (Rabner, Gottlieb, Lazdowsky, & LeBel, 2016). This phenomenon may be caused by the disruption of the endocannabinoid system as the cannabinoid 1 (CB1) receptor of this system has a role in neural cell

development and synaptic pruning; agonists to the CB1 receptor such as THC (delta 9-tetrahydrocannibinol), the psychoactive component in cannabis, can have damaging effects in brain development but also can be neurotoxic in the context of TBI and contribute to cognitive dysfunction (Rabner et al., 2016). Other diagnoses in the differential include mood disorders with psychotic features, dementia with hallucinations or delusions, and personality disorders such as paranoid type.

Another important differential diagnostic entity is schizophrenia, which is defined in DSM-5 (American Psychiatric Association, 2013) by a period of at least 6 months of social or occupational dysfunction in which there is a 1 month period in which two or more of the following symptoms are present: (1) delusions; (2) hallucinations; (3) disorganized speech; (4) disorganized behavior; or (5) lack of affect and avolition; at least one of the symptoms must be among the first three listed. In the context of TBI, there may be an overlap with schizophrenia, since patients with the latter disorder may have sustained undocumented head injuries, or, conversely, patients with schizophrenia may have cognitive deficits which make them more prone to sustain head injury. In these cases, it may be difficult to assess whether the head injury or schizophrenia is the etiology of the psychosis (Malaspina et al., 2001). Despite more recent reviews of the literature, there is no consensus that TBI is a risk factor for schizophrenia (David & Prince, 2005; Hesdorffer, Rauch, & Tamminga, 2009; Kim, 2008); however, there appears to be a strong association in those TBI patients with a family history of schizophrenia, suggesting the possibility of interaction between genetics and the environment (Molloy, Conroy, Cotter, & Cannon, 2011).

After a thorough history, the neuropsychiatrist proceeds with the physical, neurological, cognitive, and mental status evaluation. Appropriate laboratory tests to perform include a CBC, metabolic panel, urine toxicology for substances, and when clinically indicated, EEG and brain imaging as well. If other etiologies cannot explain the patient's symptoms and it appears that TBI is the cause of the psychosis, then the DSM-5 diagnosis of psychotic disorder due to another medical condition would be given.

Treatment

From a treatment standpoint, psychotic symptoms are treated with antipsychotic medications, also known as neuroleptics. As there are no randomized placebo-controlled studies of the treatment of psychotic syndromes occurring in the context of TBI, more general pharmacological principles utilized in treating psychosis are employed. Typical antipsychotics (dopamine receptor antagonists), also known as first-generation antipsychotics, such as haloperidol (Haldol) have traditionally been the drugs of choice. However, there is some controversy about using haloperidol in TBI patients due to a few reports that it negatively impacts on post-traumatic amnesia duration and cognition (Rao, Jellinek, & Woolston, 1985; Stanislav, 1997). Side effects to be aware of include extrapyramidal symptoms (EPS) (tremor,

cogwheeling, and bradykinesia), dystonia (slow, sustained muscular contractions), akathisia (restlessness) (Sadock et al., 2015), and the rarer but more serious outcome, neuroleptic malignant syndrome (NMS), which is marked by hyperthermia, rigidity, autonomic instability, and confusion (Kadyan, Colachis, Depalma, Sanderson, & Mysiw, 2003). Tardive dyskinesia (TD) presents with involuntary movements of the head, limbs, and trunk and can be observed as a delayed side effect of these medications usually only after years of treatment. Another concern is the fact that neuroleptics can lower the seizure threshold, making TBI patients potentially more prone to sustaining a seizure (Sadock et al., 2015). If low-potency typical antipsychotics such as chlorpromazine (Thorazine) or thioridazine (Mellaril) are utilized, EPS is less of an issue, but anticholinergic side effects are more problematic as they too can exacerbate cognitive deficits which may already be present in the traumatic brain injury population (Stanislav, 1997).

The atypical antipsychotics (serotonin–dopamine antagonists), also known as second-generation antipsychotics, which have less potential to cause EPS symptoms, are more frequently used in treating psychosis in this population of patients (Elovic, Lansang, Li, & Ricker, 2003). The common choices are risperidone (Risperdal), olanzapine (Zyprexa), quetiapine (Seroquel), and clozapine (Clozaril). Beneficial use of these agents has been recorded in a few case reports of TBI patients (Butler, 2000; Guerreiro, Navarro, Silva, Carvalho, & Gois, 2009; Michals, Crismon, Roberts, & Childs, 1993; Schreiber, Klag, Gross, Segman, & Pick, 1998; Tremeau et al., 2011; Turkalj et al., 2012; Viana, Prais, Nicolato, & Caramelli, 2010). A recent case report of a 58-year-old male with a history of TBI and paranoia was cited as the first patient to be successfully treated with a long-acting injectable antipsychotic, paliperidone, a major active metabolite of risperidone (Douglass & Smyth, 2018). Side effects to monitor with these medications include orthostatic hypotension, sedation, weight gain, hyperlipidemia, and impaired glucose tolerance. With clozapine, in particular, the increased risk of seizures and agranulocytosis make it a less attractive choice (Labbate & Warden, 2000; Michals et al., 1993; Shaughnessy, 1995). More recently, there has been an association of an increased risk of stroke in patients who were treated with these medications for behavioral problems in dementia (Herrmann & Lanctot, 2005). Since patients with TBI may eventually go on to develop dementia over time, further research will be needed to guide the prescribing practice of these agents in this subset of patients. An alternative to medications is the option of ECT which was reported in one patient with psychosis and TBI who was successfully treated with this modality (Johnson & Ward, 2017).

Vignette

A 39-year-old male suffered traumatic brain injury as a result of a motor vehicle accident with brain damage in the right-frontal–temporal brain regions. Subsequently, he developed delusions about being attacked by sharks and believed that he was no longer on earth but resided on Mars. These perceptions

caused his attention to wander during therapy sessions, and so a neuropsychiatry consult was sought.

After a thorough evaluation, the patient was prescribed an atypical antipsychotic with a subsequent decrease in his delusional thinking and improvement in his ability to participate in his rehabilitation program.

Agitation and Aggression

Agitation is a frequent behavioral problem associated with TBI patients and has been a source of debate in the field due to the lack of agreement about a standardized clinical definition. Since DSM-5 (American Psychiatric Association, 2013) lacks a specific category for agitation, the closest approximation being personality change due to another medical condition (aggressive type), a proposal has been made to create a new diagnostic label, that of aggression, which could be subdivided into acute and chronic types. The acute stage would be defined as lasting from a few weeks up to a few months and be essentially synonymous with delirium (other terms in the literature for this entity include post-traumatic amnesia and post-traumatic confusional state) which occurs most frequently as patients may be transitioning from a coma or stuporous state to that of wakefulness after the injury (De Guzman & Ament, 2017; McAllister, 2011). This syndrome can occur in about 70% of TBI patients at this acute phase (Vaishnavi, Rao, & Fann, 2009) and is marked by disturbances in attention, awareness, mood, and cognition including memory, language, and perception. In addition, decreased daytime arousal, nighttime sleep disturbance, and psychotic symptoms are also characteristic of this syndrome (Sherer, Yablon, & Nick, 2014). Patients can have hypoactive or hyperactive delirium as well as mixed presentations. Agitation in these cases can manifest as psychomotor activity such as restlessness, trying to pull out lines and tubes, and attempting to get out of bed or elope from the unit (Luaute et al., 2016). Patients can also manifest motor symptoms such as tremor, asterixis, and myoclonus. Although the pathogenesis of post-traumatic delirium is not fully understood, the possible mechanisms cited include neuroinflammation, neurotransmitter imbalance, and structural damage with dysregulation of neuronal networks (Hughes, Patel, & Pandharipande, 2012). The enormity of this problem is emphasized in a study by Bogner and colleagues, who reported that the presence of agitation in TBI patients receiving treatment in an acute rehabilitation center was predictive of a longer length of stay and a decrease in functional independence from a cognitive standpoint at discharge (Bogner, Corrigan, Fugate, Mysiw, & Clinchot, 2001).

The chronic stage of post-traumatic aggression would refer to the persistence of inappropriate verbal or physical behaviors beyond the 2-month time frame (Silver, Yudofsky, & Anderson, 2005). It is important to highlight that this post-traumatic aggression is not purposeful and does not reflect forethought to act in a violent or destructive fashion for a particular end result. Rather, it is more impulsive and reflects the inability on an emotional and cognitive level to appropriately discern a

situation or conflict and respond accordingly (Wortzel & Arciniegas, 2013). Patients can be aggressive in their speech or be physically aggressive with others. From the physiatry literature, an interdisciplinary definition has been suggested that would incorporate the elements of delirium, post-traumatic amnesia, and excesses of behavior that include some combination of aggression (verbal or physical), akathisia, disinhibition, and emotional lability (Sandel & Mysiw, 1996).

One way to help standardize the definition of agitation would be the utilization of valid and reliable scales, an example of which is the Agitated Behavior Scale (ABS) (Corrigan, 1989). This instrument includes 14 items which rate the patient's behavior in a variety of areas such as attention, impulsivity, irritability, violence, anger, wandering, pulling at tubes, and self-stimulating or self-abusing actions. Each observable behavior is rated from 1 to 4 (absent, slight, moderate, or extreme) with a cumulative score greater than 36 considered to be in the severe range of agitation. Another of these instruments is the Overt Agitation Scale (OAS), which measures verbal aggression as well as physical aggression to self, objects, and people. Each of these four areas is rated in a range from mild to severe (Brooke, Questad, Patterson, & Bashak, 1992). A recent article reviewing validated aggression scales used in assessing TBI from 1950 to 2012 (Cusimano, Holmes, Sawicki, & Topolovec-Vranic, 2014) concluded that despite the prevalence of this problem, there was only one study of the 32, which met the inclusion criteria, that demonstrated statistical validation of aggression. The study by Johansson, Jamora, Ruff, and Pack (2008) of 67 patients with mild-to-severe TBI evaluated with the Ruff Neurobehavioral Inventory (Ruff & Hibbard, 2003), a 243-item questionnaire with an anger subscale, which allows patients to assess pre- and postmorbid factors, demonstrated the strongest evidence as a measure of aggression.

The incidence of agitation has been reported to be from 35% to 96% (Levin & Grossman, 1978; Rao et al., 1985) in the acute recovery period after TBI, and from 31% to 71% in patients who were followed 1–15 years after sustaining TBI (McKinlay, Brooks, Bond, Martinage, & Marshall, 1981; Oddy, Coughlan, Tyerman, & Jenkins, 1985). Subsequently, a study of 158 subjects in an acute-care rehabilitation setting, most of whom had severe TBI, demonstrated that approximately 50% of these patients had post-traumatic agitation as measured by the ABS; this study noted that there were no statistically significant differences as regards to gender in terms of the frequency, duration or presentation, and extent of the post-TBI agitation (Kadyan et al., 2004). A newer, large study of 507 patients with severe TBI and aggression that occurred within 6 months of injury reported the prevalence rate to be 31.9% with the use of the Overt Behaviour Scale (OBS), a clinician-rated instrument which evaluates challenging behaviors such as verbal and physical aggression in TBI patients (Kelly, Todd, Simpson, Kremer, & Martin, 2006). Verbal rather than physical aggression was more predominant, and an important finding was that increased care needs and decreased participation in life roles were significantly associated with challenging behaviors (Sabaz et al., 2014). This study is similar to an older one by Tateno and colleagues (Tateno, Jorge, & Robinson, 2003) that demonstrated 33.7% of 89 patients manifested significant aggressive behavior when measured with the OAS 6 months after their injury. Furthermore, the aggressive

behavior was significantly associated with major depression, a history of alcohol or drug abuse, frontal lobe lesions, and poorer social functioning.

The ability to anticipate factors associated with subsequent aggression would be helpful in guiding treatment in TBI patients, as evidence of agitation at 3 months predicts worse outcome at 1 year (DeBoussard et al., 2017). In one prospective study of 146 TBI admissions to a rehabilitation unit, 53 patients (36.3%) manifested agitation, and the brain imaging of those patients revealed either intracranial hemorrhage or diffuse axonal injury as pathology, whereas those with contusions were less likely to exhibit agitation (Singh, Venkateshwara, Nair, Khan, & Saad, 2014). Predictors of agitated behavior in an inpatient TBI rehabilitation unit included decreased cognitive function and infection in an updated prospective study of 3 years' duration. Of note in that study, there was also an association with higher levels of agitation in patients treated with medications in certain classes including sodium channel antagonist anticonvulsants, gamma-aminobutyric acid antianxiety agents, and second-generation antipsychotics. The possibility that these medications suppress cognitive functioning may explain this phenomenon, while medications that enhance attention and speed of processing such as norepinephrine–dopamine–5-hydroxy tryptamine agonists such as methylphenidate and wakefulness-promoting agents such as modafanil were associated with decreased agitation (Bogner et al., 2015). Another recent study by Roy, Vaishnavi, Han, and Rao (2017) highlighted that in 103 patients with first-time TBI and depression at 3 months after injury could predict the development of aggression at 12 months post-injury. This emphasizes the need to address depressive symptoms early in the course of treatment post-TBI to try to mitigate against the potential of the development of aggressive behavior later on in the recovery period. Genetics may also play a role in the expression of aggression in conjunction with lesion location, as was reported in one study of patients with different genotypes of the dopamine receptor D1 (DRD1); those patients with transcriptionally active DRD1 alleles and medial prefrontal cortex lesions were more likely to demonstrate aggression as measured by the NPI subscale compared to those with the same allele and lateral prefrontal cortex lesions. Further genetic studies of this type may help to inform more precise treatment approaches in the future (Pardini et al., 2014). Another tool that may improve the identification of patients likely to become agitated post-TBI is that of neuropsychological testing; in a study of 348 patients who had a brief neuropsychological assessment within the first month after trauma, the odds of being agitated, labile, irritable, and disinhibited were almost six times higher if the time of post-traumatic amnesia (PTA) lasted greater than 7 days as compared to those with less than 24 h of PTA (De Guise, LeBlanc, Feyz, Lamoureux, & Greffou, 2017).

Anatomically, agitation or aggression may be explained by damage to a number of different brain areas such as the hypothalamus, amygdala, medial temporal lobe, or orbitofrontal cortex, as these regions and their connections are involved in the regulation of emotion (Arciniegas & Beresford, 2001). Several neurotransmitter systems are also involved in the expression of agitation and aggression, but the literature differs in whether the neurotransmitters (norepinephrine, dopamine, serotonin, acetylcholine) are consistently decreased in the various studies reported (Silver et al., 2005).

Assessment

In acute agitation, the neuropsychiatrist must first assess if there are other underlying medical conditions (in addition to TBI) such as infections, metabolic imbalances, hypoxia, anemia, or medications such as narcotics, anticholinergic agents, or steroids which can be contributing to the patient's delirium. Additionally pain, constipation, urinary retention, or disturbance of the sleep–wake cycle may be involved as the causes of agitation and need to be identified (De Guzman & Ament, 2017; Luaute et al., 2016).

The neuropsychiatrist who is asked to evaluate a patient with the chronic form of agitation in his/her office will need to take a thorough history and perform a complete neuropsychiatric examination as well as any necessary laboratory studies to be able to rule out other medical problems which may be the underlying etiology for the agitation. Included in the possible diagnoses would be a new episode of delirium, being postictal, pain syndromes, and the use of alcohol or illicit drugs. Psychiatric diagnoses in which aggression can be seen include major depression, bipolar disorder, anxiety disorders such as PTSD and GAD, and personality disorders such as antisocial type (Silver et al., 2005). The relationship of mild TBI (with or without loss of consciousness) and the psychiatric diagnosis of intermittent explosive disorder (IED), which is marked by impulsive aggressive behavior, has recently been shown to be significantly correlated in 695 patients with IED compared to 453 healthy controls and 486 patients with other psychiatric illnesses (Mosti & Coccaro, 2018).

Treatment

To treat the symptoms of acute agitation, neuroleptics such as haloperidol are used. Short-term use in delirium to improve confusion and psychosis, with appropriate tapering and discontinuation of the neuroleptic when the delirium clears, is acceptable. Droperidol (Inapsine), an antipsychotic agent similar to halperidol, was reported to be effective in treating acute agitation in 27 patients with TBI (Stanislav & Childs, 2000), but this agent is not commonly used. Other medications sometimes used in the acute setting include BZDs such as lorazepam (Mysiw & Sandel, 1997). In one of 11 TBI patients who were between 4 and 23 days post injury, the treatment for acute agitation was the combined use of amantadine, methylphenidate, and trazodone. All the patients were noted to have resolution of their agitation as well as improvement in their cognitive function (Rosati, 2002). Additional randomized, controlled prospective studies are needed to determine the efficacy of this treatment approach. The environment must also be addressed in the treatment protocol for delirium to try to reduce agitation. It is recommended that unnecessary catheters, intravenous lines, and nasogastric tubes which can be a source of frustration and confusion for the patient be eliminated. The environment needs to be safe

to prevent falls and calm to try to foster proper rest and re-entrain the patient's circadian rhythm (Luaute et al., 2016).

There are few pharmacological agents with prospective studies of a randomized, placebo-controlled design which can definitively guide the treatment of agitation in TBI patients, but the beta-blocker propranolol and the stimulant methylphenidate have been exceptions in this regard. In separate studies of propranolol, it has been shown that there is either a statistically significant reduction in the maximum intensity of the episodes of agitation (Brooke, Patterson, et al., 1992; Brooke, Questad, et al., 1992) or in the actual number of aggressive episodes which occur (Greendyke, Kanter, Schuster, Verstreate, & Wootton, 1986). The Cochrane review in 2006 also noted that of six studies for aggression in TBI that were randomized-controlled, only the beta-blockers propranolol and pindolol showed any efficacy (Fleminger, Greenwood, & Oliver, 2006). Stimulants such as methylphenidate have been used successfully to treat temper outbursts marked by belligerence and hostility in 38 male patients who had sustained TBI 2 years prior to the study (Mooney & Haas, 1993). Amantadine, a dopaminergic agent, has been demonstrated to be of benefit in the treatment of aggressive behavior in TBI patients as noted in case reports (Chandler, Barnhill, & Gualtieri, 1988) as well as in a retrospective case analysis (Nickels et al., 1994). In an updated study using amantadine, 100 mg twice per day in a parallel-group, randomized double-blind placebo-controlled cohort of patients with chronic TBI and aggression, there was some modest reported improvement in anger and irritability on rating scales from the perspective of the participants in how they judged themselves on these parameters that was statistically significant compared to the placebo group (Hammond et al., 2017). The anticonvulsants including carbamazepine (Azouvi et al., 1999; Chatham-Showalter, 1996; Kennedy et al., 2001), valproic acid (Wroblewski, Joseph, Kupfer, & Kalliel, 1997), gabapentin (Rybach & Rybach, 1995), and lamotrigine (Pachet, Friesen, Wenkelaar, & Gray, 2003; Whiting, Sullivan, & Stewart, 2016) provide another option in the treatment of TBI patients with agitation, as has also been reported in case reports and open-label trials. However, since the evidence for efficacy of these agents is limited, they might be best used in those patients also suffering from post-traumatic epilepsy or pain syndromes, so that one drug can be used to address several issues (Kalra & Watanabe, 2017). Antidepressants, such as sertraline in the SSRI class (Kant, Smith-Seemiller, & Zeiler, 1998), amitriptyline in the TCA group (Mysiw, Jackson, & Corrigan, 1988), trazodone (Rowland, Mysiw, & Bogner, 1992), and bupropion (Teng et al., 2001) have been noted to be useful in treating agitation and aggression in this population as well. Buspirone, in the anxiolytic class, has been observed to be effective in the treatment of angry outbursts and behavioral problems in TBI patients (Gualtieri, 1991a, 1991b; Holzer, 1998). The side effects of all these medications have been previously reviewed. Although other agents such as the mood stabilizer lithium (Glenn et al., 1989) or the benzodiazepines (Freinhar & Alvarez, 1986) have been used in the management of agitation, these medications are probably best avoided in the TBI population due to the potential neurotoxic effects

(tremor, delirium, and seizures) of the former agent and possible cognitive disturbances (attention, alertness, and memory) of the latter (Perna, 2004). As with acute agitation and aggression, there are some reports of using the atypical antipsychotics such as quetiapine and ziprasidone to deal with patients who have chronic aggressive behavior secondary to TBI (Kim & Bijlani, 2006; Noe, Ferri, Trenor, & Chirivella, 2007). Preliminary work using dextromethorphan/quinidine in patients with other neurological disorders and concomitant aggression raise the possibility of its future use in TBI patients (Chen, Calcagno, & Shad, 2018). The use of ECT as an alternative treatment to medication was found to help one patient with severe TBI and behavioral disturbance when he proved unresponsive to a variety of psychopharmacological agents (Kant et al., 1995).

Finally, the neuropsychiatrist should also work in conjunction with the therapists on the rehabilitation team to be aware of the behavioral approaches which are being utilized to help the patient deal with agitation and aggression (Rothwell, LaVigna, & Willis, 1999). These can include altering the environment to decrease provocative stressors, coping skills training, and behavior modification involving reinforcements for appropriate behavior (Watson, Rutterford, Shortland, Williamson, & Alderman, 2001). Another approach with preliminary evidence documenting significant reduction in agitation in 14 patients is the utilization of the patient's preferred music choice as compared to classical relaxation music (Park, Williams, & Lee, 2016). The family members should also be encouraged to seek supportive psychotherapy to help them cope with the injured loved one's behavioral disturbance and personality changes.

Vignette

A 70-year-old woman sustained head trauma when her car was broadsided by a truck. She sustained a left hemispheric subarachnoid hemorrhage with extension into the bilateral sylvian fissures as well as a left parietal/occipital subdural hematoma. Several months later, when the patient was at the subacute rehabilitation facility, she became very angry when she felt that the staff did not appreciate that her abilities to perform tasks were much better than the rest of the patients there. She believed that she did not belong in the facility and was often packing her bags and threatening to leave the building. On one occasion, she ran out of the therapist's office into the parking lot with the staff in pursuit, and in another incident, while on a weekend pass to visit family, she refused to get in her daughter's car to be driven back to the rehabilitation center. She was physically aggressive toward family members, including biting, kicking, and hitting them.

After evaluation with the neuropsychiatrist, valproic acid was used to treat the patient, and she demonstrated a dramatic improvement in behavioral dyscontrol.

Arousal and Attention

When a patient sustains TBI, the physiological state known as arousal, which is defined by the level of wakefulness and the intensity of stimulation needed to elicit a meaningful response by the individual, can be altered by varying degrees. Although in normal consciousness, the person is fully awake and able to respond cognitively and emotionally to both internal needs as well as to external stimuli, the drowsy patient sustains wakefulness only with the application of some form of external stimuli. These patients are often inattentive and confused. At the level of stupor, a patient can only be roused by vigorously repeated and often noxious stimuli; once the stimulus ceases, the patient lapses back into unresponsiveness. The comatose patient appears to be asleep and incapable of being aroused by either external stimuli or their own internal needs, while the patient in a vegetative state undergoes alternate sleep–wake cycles, but does not regain awareness or purposeful behavior. When this condition extends beyond 1 month, the term "persistent vegetative state" is applied (Adams, Victor, & Ropper, 1997; Mesulam, 2000).

Overlapping with this concept is that of attention, since the ability to attend or concentrate on stimuli is predicated on one's degree of arousal. Impaired attention is a problem frequently observed in patients who have suffered TBI and its impact upon rehabilitation efforts is profound, since other cognitive processes such as encoding and storing items in memory, problem-solving, and language skills are dependent upon one's ability to focus on various stimuli (Stierwalt & Murray, 2002). The construct of attention is further divided into: (1) basic attention or the capacity to orient to simple stimuli; (2) selective attention, or the ability to prioritize some stimuli over others; (3) sustained attention, or vigilance, which represents the capacity to maintain attentional focus over time; and (4) divided attention, in which one must respond to or process multiple stimuli simultaneously (Niemann, Ruff, & Kramer, 1996). Often after TBI, the basic attention abilities recover, but psychometric testing in a few recent studies reveal that TBI patients, several years post injury, still struggle with cognitively challenging tasks when impairments in divided and sustained attention persisted (Dockree et al., 2004; Mangels, Craik, Levine, Schwartz, & Sluss, 2002; Stierwalt & Murray, 2002; Vanderploeg, Curtiss, & Belanger, 2005).

Anatomically, the arousal and attentional systems are complex and widely distributed through the brain and involve the ascending reticular formation of the brain stem, which extends from the medulla to the midbrain: the hypothalamus, thalamus, basal forebrain, limbic system, anterior cingulate, and parietal, temporal, and prefrontal cortical areas. Damage to any of these regions via mechanical injury or diffuse axonal impairment can disrupt the various neurotransmitter pathways (noradrenergic, dopaminergic, and cholinergic) which play key roles in the modulation of arousal and attention (Mesulam, 2000). Evidence for the latter neurotransmitter's role in this cognitive domain was highlighted in a recent study of TBI patients whose neuropsychological profile demonstrated decreases in sustained attention and reaction times, while the morphometric analysis of their brain imaging revealed reduced gray matter density in the regions of all the major cholinergic pathways (Salmond, Chatfield, Manon, Pickard, & Sahakian, 2005).

Assessment

The neuropsychiatrist who evaluates the patient with arousal and attention deficits needs to conduct a thorough history with regard to factors which can induce a decreased level of awareness, such as infections, metabolic abnormalities, seizures, strokes, drug intoxication, and medications (Adams et al., 1997). A careful neurological examination will include testing cranial nerves for pupillary reactivity, ocular motor movements, and oculovestibular reflexes to gauge brain stem function. Additionally, the level of arousal is assessed via the patient's ability to respond verbally, motorically, or via eye opening to various stimuli. Then, depending upon the patient's degree of alertness and ability to participate, a bedside cognitive evaluation which highlights tests of attention should be performed. Some examples of these tests include the digit-span (repetition of a list of numbers in which 7 ± 2 digits forward and 5 ± 1 digit in reverse is normal); a continuous performance test (the patient lifts his/her arm whenever the letter "A" is read aloud among a group of letters); trail-making tests (the patient connects in proper sequence an array of numbers or alternating numbers and letters which are arranged haphazardly on a paper); and an alternating sequences task (the patient must imitate a series of three hand gestures—palm, fist, and edge of hand—repetitively without error). These tests help to determine if there are attentional deficits as manifested by distractibility, perseveration, or response inhibition (Mesulam, 2000). For more extensive cognitive evaluation, a neuropsychological battery should be ordered which can further assess the subsets of attention with more sophisticated measures, sometimes using computerized auditory or visual stimuli (Cicerone, 2002; Stierwalt & Murray, 2002). An alternative approach to assessing attention that can be helpful in an acute rehabilitation setting is the use of a rating scale based on the staff's observations of patients in everyday activities. As has been discussed, a patient may have deficits in various subtypes of attention, and thus performance on different tasks may help to categorize what these impairments might be. Examples of these include the neurobehavioral rating scale (NRS) (Levin, High, Goethe, & Sisson, 1987), which documents alertness, attention, and fatigability, while the Moss Attention Rating Scale includes items for arousal, alertness, sustained attention, distractibility, and divided attention (Whyte, Hart, Bode, & Malec, 2003). Laboratory workup to elucidate the etiology of a diminished level of arousal should include routine blood tests, urinalysis, toxicology, brain imaging, lumbar puncture if warranted, and an EEG, as an alteration in brain waves occurs in virtually all disturbances of consciousness (Adams et al., 1997).

Treatment

Increasing a patient's level of arousal and attention after TBI has been attempted with medications as well as through nonpharmacological means. An example of the latter is the study by Wilson and colleagues (Wilson, Powell, Brock, & Thwaites, 1996),

who provided environmental sensory enhancement to 24 patients in a vegetative state. A more robust response, as measured by the frequency of eye-opening and body movements, was noted when each of the five senses was stimulated at each treatment session, as compared to when just a single sense was stimulated. In addition, an increased level of arousal was also observed when the individual was exposed to personal favorite stimuli such as foods, songs, or photos, as contrasted with the use of neutral stimuli. To improve attention, nonpharmacological approaches that have been utilized include teaching compensatory strategies, such as reducing distracting elements in the environment and taking breaks to maximize one's performance (Mateer, Kerns, & Eso, 1999), as well as learning to anticipate task demands, to repeat information, and to get clarification when having to manage tasks in the setting of time constraints (Cicerone, 2002).

In the pharmacological treatment of patients with deficits in arousal and attention, one approach that has been used is based on the idea of enhancing neurotransmitter systems which have been disrupted secondary to TBI. As discussed previously, psychostimulants such as methylphenidate and dextroamphetamine serve to augment the concentration of dopamine and norepinephrine by increasing their release and blocking their reuptake in the synapse. Controlled studies have been conducted with both of these agents (Evans, Gualtieri, & Patterson, 1987; Plenger et al., 1996), but the most frequently documented positive effect in neuropsychological tests of attention was in processing speed (Whyte et al., 1997), while the benefit of these medications to increase attention or reduce distractibility has been less certain (Whyte et al., 2004; Whyte, Vaccaro, Grieb-Neff, & Hart, 2002). A randomized placebo-controlled trial of 71 patients with TBI evaluated the use of methylphenidate with cognitive rehabilitation which used compensatory training strategies and indicated the combination of the two treatment approaches demonstrated a significant benefit on the measures of learning, working memory, and divided attention (McDonald et al., 2017). Case studies of TBI patients, including individuals in the persistent vegetative state or minimally conscious state, have indicated that amantadine, another dopaminergic agent, improves attention, concentration, and arousal (Kraus & Maki, 1997; Nickels et al., 1994; Zafonte, Watanabe, & Mann, 1998). Other dopaminergic agents which have been shown to be useful in enhancing alertness include levodopa and bromocriptine (Lal et al., 1988; Powell et al., 1996). A recent report which utilized bromocriptine in 36 minimally conscious patients indicated improvement in arousal, attention, and cognition from 4 to 40 days of its administration (Munakomi, Bhattarai, & Kumar, 2017). Modafinil, which appears to activate limbic areas and is approved for narcoplepsy, is an obvious potential choice for treating underarousal in TBI (Elovic, 2000; Teitelman, 2001). Antidepressants with noradrenergic effects such as amitriptyline and desipramine have also been demonstrated to improve arousal and responsiveness in three patients with severe TBI (Reinhard, Whyte, & Sandel, 1996). The side effects of these medications have been reviewed in previous sections.

Since TBI often results in the dysfunction of the cholinergic system in the hippocampus and frontal cortical areas, the regions which play a pivotal role in the cognitive function of attention (Arciniegas et al., 1999; Salmond et al., 2005),

the use of AchE-Is may also be useful pharmacological agents in treating these deficits, as several studies have indicated (Griffin et al., 2003; Kaye, 2003; Zhang, Plotkin, Wang, Sandel, & Lee, 2004). Side effects of these medications have already been reviewed. It is noteworthy that there is an overlap in both the neuroanatomic structures and the neurotransmitter systems which play key roles in the biology of arousal and attention, as well as motivation, since the latter provides an individual with the drive to respond to stimuli once he is alert and able to concentrate. Therefore, the use of similar pharmacological agents to treat disorders of these functions appears to be a sound clinical approach.

Vignette

A 34-year-old male with a history of cardiomyopathy suffered a cardiac arrest with a prolonged period of unresponsiveness of unknown duration. He was resuscitated and placed on life support and subsequently underwent successful cardiac transplantation. After recovery from his surgery, he was noted to be fatigued and sleepy a lot of the time. He would close his eyes during the rehabilitation sessions and say, "I want to sleep," in a monotone voice. Left to his own devices, he would immediately return to his room to sleep. He needed a great deal of repeated external stimulation by his therapists to enable him to remain alert and concentrate on a task for even brief periods. In addition, due to the anoxic encephalopathy which he suffered as a result of the cardiac arrest, his short-term memory was poor, and he lacked drive to do things spontaneously.

The patient was treated with a variety of stimulants, including methylphenidate, with only a slight improvement. Subsequently, the patient was placed on high-dose venlafaxine, as his clinicians thought his symptoms were consistent with depression; and on this medication, he did show some improvement. Later, in his course of treatment, donepezil and modafinil were added sequentially to help increase his level of attention. Therapists in the rehabilitation center who work with him have noted an improved level of alertness and ability to concentrate as well as increased spontaneity in answering questions with this combination of pharmacological medications.

Neurocognitive Disorders

The cardinal feature of dementia, which is now classified as the major neurocognitive disorder as defined by DSM-5, is a significant decline in cognition from a previous level of performance, as noted by the individual or knowledgeable informant. In addition, there must be a decline in at least one or more cognitive spheres such as complex attention, executive function, learning and memory, language, perceptual-motor, or social cognition. Furthermore, these deficits interfere with the individual's

ability to maintain independence in social and/or occupational functioning. In the acute period just after TBI, cognitive deficits can be present secondary to delirium or post-traumatic amnesia. Major neurocognitive disorder due to TBI, however, is a more insidious process and refers to residual deficits which persist for months or years post injury. Patients with TBI can also suffer with modest cognitive decline which is designated as mild neurocognitive disorder in DSM-5 in which the individual will have deficits in the above noted cognitive categories as in major neurocognitive disorder and which represents a decline from the usual baseline, but the deficits do not interfere with independence in function (American Psychiatric Association, 2013). An important diagnostic issue is that at times with the cognitive decline, patients can suffer behavioral disturbances such as psychosis or mood disturbances such that the cognitive problem then would subsume the symptoms of the other conditions. Depending on the predominance of the symptoms, and if the symptoms are deemed a direct pathophysiological sequelae of TBI, it may obviate the need for an additional diagnostic designation such as depression due to TBI (Wortzel & Arciniegas, 2014).

The underlying pathophysiological relationship between TBI and dementia is unclear: is TBI an inciting nidus for neurodegeneration or does it accelerate a neurodegenerative process that is already at work? The literature is fraught with complexity again due to how data is collected in studies and what definitions are used to define TBI including whether loss of consciousness plays a role or not (LoBue, et al., 2018).

The prevalence of dementia after TBI is not precisely known but has been reported to occur at a rate of between 5% and 17.5% (Gualtieri & Cox, 1991; Koponen et al., 2002), whereas the prevalence of memory disturbances alone, the most common cognitive problem after TBI, ranges from 23% to 79% (Levin, 1990). Dementia in TBI patients may be due to damage of the frontal anterior- and medial-temporal cortices as well as the underlying white matter which connects cortical to subcortical areas (Arciniegas & Beresford, 2001). In addition, since acetylcholine-rich hippocampal regions which are responsible for short-term memory function are frequently damaged in TBI, cholinergic dysfunction is believed to be etiologically related to the memory impairment seen in these patients (Arciniegas et al., 1999). One possible mechanism to explain the neuropathological overlap in these two entities suggests that the presence of the apo-lipoprotein E (epsilon) 4 allele, which retards neural repair after trauma, serves in turn as a risk factor for the deposition of beta-amyloid protein and the subsequent formation of neurodegenerative plaques in AD (Jellinger, 2004; Koponen et al., 2004; Luukinen et al., 2005).

Whether TBI is a definite risk factor for Alzheimer's disease (AD) remains controversial, as some research has shown an increased risk for AD in patients with head injury and other studies have not (Mehta et al., 1999; Plassman et al., 2000; Williams, Annegers, Kokmen, O'Brien, & Kurland, 1991). Some recent meta-analyses of the literature have also been contradictory with regard to whether the history of TBI is associated with subsequent neurodegerative diseases, which include not only AD but other dementias such as frontal temporal and Parkinson's disease (Huang, Lin, et al., 2018; Julien et al., 2017; Perry et al., 2016). Updated reports have tried to eliminate bias in prior studies where determination of TBI was

by self-report of the injured individual or by using billing codes for dementia. In one study of 7130 subjects of whom 1589 underwent autopsy at the time of death, the analysis indicated that instead of Alzheimer pathology, the patients were more likely to evidence the neuropathology of Parkinsonism including Lewy body disease and Parkinson's disease (Crane et al., 2016). A recent study by Weiner et al. (2017) which reviewed the records of TBI patients from the Veterans Administration revealed no correlation with prior TBI and the later development of AD as validated by the lack of structural abnormalities on MRI scans and no evidence of amyloid on PET scans.

In an epidemiological study of TBI patients, it was found that the observed time from the brain injury incident to the development of AD was less than expected (Nemetz et al., 1999), implying that TBI may hasten the yet undetermined cascade of events necessary to precipitate AD in those patients who are ultimately predisposed to its development. The recent report by Fann et al. (2018) of a population-based observational cohort study evaluated younger adults with TBI and spanned a period of 36 years. They reported that the younger the patient was at the time of injury, the higher the risk of dementia, and five or more TBIs increased the risk; the risk was higher in the first 6 months post TBI. These studies give support to the Satz model of cognitive reserve, which hypothesizes that the brain capacity which is available to carry on the basic ability to function as a human being differs for each person. Therefore, dementia would occur at the point where there is a critical reduction in those neurons necessary to carry on these basic functions; this decrease in neurons could be due to normal aging, disease, or external factors such as toxins or TBI (Satz, 1993). Additional hypotheses suggest that trauma may predispose the brain to varying types of neurodegeneration through mechanisms such as oxidative stress, neuronal cell death, white matter damage, and neuroinflammation, which in concert with the aging process can cause patients to reach the threshold for a dementing illness sooner than those without TBI (Liu et al., 2017; LoBue, et al., 2018). In light of this hypothesis, a recent study indicated a high risk level of 3.64 for a subsequent diagnosis of dementia in patients with type I diabetes who sustained a TBI usually as a result of falls. The possibility of reduced brain reserve in type I diabetics was considered to be a plausible etiological explanation for this phenomenon (Gilsanz, Albers, & Schnaider Beeri, 2018). In light of the above discussion further studies of the neurodegenerative disease known as chronic traumatic encephalopathy (CTE) which is caused by repetitive head impacts, is characterized clinically by cognitive and mood symptoms and on pathologic analysis of the brain evidences an abnormal accumulation of tau protein, may help to clarify some of the questions associated with TBI and dementia (Mahar, Alosco, & McKee, 2017).

Assessment

The evaluation of a patient with cognitive decline begins with a thorough history about the current TBI incident and its subsequent treatment as well as a review of medical and surgical problems including whether there have been prior TBIs, falls,

seizures, or strokes. Additionally, the patient's psychiatric history, family history of neurological, and psychiatric problems, medications, and social history, including the level of education, alcohol and drug use, and driving issues should also be assessed. Questions about the impact of the cognitive deficits on the patient's ability to function safely and independently at home, socially, or in the workplace if applicable are key points to address. A comprehensive physical and neurological examination, as well as a cognitive assessment which tests for attention, memory, and frontal lobe functions, is vital, as is a thorough mental status exam which assesses for psychiatric symptoms. Laboratory tests include a CBC, electrolytes, liver function tests, B12, and folate, as well as brain imaging such as an MRI (magnetic resonance imaging). Neuropsychological testing can help to establish the patient's baseline in terms of current cognitive strengths and weaknesses. A recent overview of neuropsychological deficits observed in patients with TBI include: slowed information processing, impaired long-term memory, attention, working memory, executive function, social cognition (ability to understand the behavior of others and to react appropriately in social settings), and self-awareness (Azouvi, Arnould, Dromer, & Vallat-Azouvi, 2017). The neuropsychiatrist must perform a thorough evaluation to rule out other possible etiologies in the differential diagnosis of cognitive decline, such as hydrocephalus, strokes, neoplasm, subdural hematoma, vitamin deficiencies, delirium, depression, and endocrine abnormalities such as hypothyroidism (Frederiks, Hofmann, & Reichard, 2002; Small et al., 1997) as well as hypopituitarism, which has been reported in TBI patients with cognitive impairment (Popovic et al., 2005; Springer & Chollet, 2001).

To be able to generate better hypotheses about the predictability of dementia in TBI patients, prospective studies using biomarkers would be of significant benefit. An exciting recent report utilizing single-chain variable fragments which bind disease-associated protein variants such as amyloid beta, alpha-synuclein, tau, and TDP-43, all of which are related to neurodegenerative dementias, and all of which can be seen accumulating early on after TBI, used this technology to analyze blood from patients with various types of TBI and was able to distinguish TBI cases from controls even years after the injury. Researchers were also able to subtype the samples, as not all samples had the same aggregates of these abnormal proteins, which prospectively would be an important component in monitoring damage over time and in trying to design studies of therapeutic modalities based on the subtypes (Williams, Peltz, Yaffe, Schulz, & Sierks, 2018).

Treatment

An important issue to address in treating TBI patients who have cognitive deficits is whether a concomitant diagnosis of depression is present. In a recent study, 28.4% of 74 patients with mild or moderate TBI who also suffered from major depression were found to have significantly lowered scores on measures of working and verbal memory, processing speed, and executive function as compared to patients without

this diagnosis (Rapoport, McCullagh, Shamni, & Feinstein, 2005). However there is ambiguity about whether cognition specifically improves in TBI patients with the use of serotoninergic agents. In one study of 15 patients with mild TBI and depression, neuropsychological tests were noted to improve when patients' mood symptoms had been successfully treated with the antidepressant sertraline (Fann, Uomoto, & Katon, 2001). However in a randomized controlled trial of 11 patients with severe TBI, the administration of sertraline 2 weeks after injury did not indicate significant improvements on attention and orientation (Meythaler et al., 2001); this was also the case in a more recent study of a double-blind randomized control design of 49 patients who received sertraline for 3 months after TBI and who showed no differences in cognitive testing compared to placebo even 12 months later (Banos et al., 2010). In a recent meta-analysis of this topic, postacute recovery actually seemed to be worsened by the administration of sertraline on tests of memory and reaction time (Wheaton, Mathias, & Vink, 2011). Failla, Juengst, Graham, Arenth, and Wagner (2016) examined 154 severe TBI patients who were taking antidepressants and found that the use of medication impaired cognition (processing speed, visual and verbal memory, attention, language fluency, and executive function) in these patients, even those without post-traumatic depression as some of the patients may have been on these medications for other indications rather than mood. These studies raise a conundrum as treatment with the SSRIs in TBI patients with cognitive issues who may also have concomitant depression may benefit from some amelioration of their depressive symptoms but could potentially suffer a worsening of cognitive ability. Further research on the prediction of which patients would improve from SSRI treatment in this scenario is needed (Yue et al., 2017).

Since TBI may produce cognitive impairment neurochemically via the disruption of cholinergic function, it has been suggested that using cholinergic-enhancing medications such as choline precursors or AchE-Is may be an appropriate pharmacological approach. Cytidine 5′diphosphocholi is a choline precursor which has been reported to be effective in improving cognition after TBI in both case studies (Leon-Carrion, Dominguez-Roldan, Murill-Cabezas, Dominguez-Morales, & Munoz-Sanchez, 2000; Spiers & Hochanadel, 1999) as well as in a randomized double-blind placebo-controlled study of 14 patients (Levin, 1991). Although physostigmine, an AchE-I, has not been shown to be consistently effective in the treatment of memory deficits of TBI patients in several different studies (Goldberg et al., 1982; Levin et al., 1986), donepezil, another member of the AchE-I class, has been reported to improve memory in TBI patients in open-label and case study reports (Masanic, Bayley, van Reekum, & Simard, 2001; Morey, Cilo, Berry, & Cusick, 2003; Taverni, Seliger, & Lichtman, 1998; Whelan, Walker, & Schultz, 2000; Whitlock, 1999). More recently in a 24-week randomized, placebo-controlled, double-blind, crossover trial with 18 patients who had sustained TBI less than 6 months prior to the study, Zhang and colleagues documented that donepezil significantly increased neuropsychological testing scores in short-term memory and sustained attention (Zhang et al., 2004). Furthermore, AchE-Is have also been shown to benefit mood, affect, and social interaction in brain-injured patients (Kaye, 2003; Whelan et al., 2000). In a nonrandomized, open-label study of 111 outpatients

with TBI who either received donepezil or one of the two newer AchE-Is, namely, rivastigmine (Exelon) or galantamine (Razadyne), the areas of vigilance, concentration, initiation, and general function were noted to be subjectively markedly improved in 61% and modestly improved in 39% of this population (Tenovuo, 2005). However a subsequent study in 2009 of a randomized, placebo-controlled crossover trial of 102 patients with mild-to-severe TBI did not show significant differences in cognition in the patients receiving rivastigmine (Tenovuo et al., 2009). Similarly, Silver et al. (2009) also failed to show benefit of improved cognition of this same agent in a double-blind study of mild-to-severe TBI patients. The use of memantine, an NMDA receptor antagonist, which targets the glutamate system has not been validated with any large-scale TBI studies, but one preliminary study demonstrated that improved cognitive scores after treatment with memantine in 17 TBI patients was correlated with an increased cerebral glucose metabolism on PET scans in prefrontal and parietal brain areas (Kim, Shin, & An, 2010). Large-scale randomized, double-blind placebo-controlled studies are needed to clarify the benefits of these agents in TBI patients. In addition to psychopharmacological strategies, the possibility of physical exercise serving a role in cognitive recovery after TBI bears further exploration (Morris, Gomes Osman, Tormos Munoz, Costa Miserachs, & Pascual Leone, 2016).

Vignette

A 50-year-old male sustained a left-frontal–temporal brain injury 3 years ago. Despite cognitive remediation and the use of compensatory strategies such as a memory book in which he writes his daily activities, his memory is still poor. He has trouble organizing what tasks he must complete and requires a lot of supervision from his wife. Due to his cognitive deficits, he was unable to return to his occupation as a clerk in an insurance company. After neuropsychiatric assessment, the patient decided to be started on donepezil with modest improvement in his memory.

Sexual Dysfunction

After TBI, a patient's sexuality can be altered due to physiological or performance issues such as libido and the physical sexual act, or because of a perceived change in his or her own subjective experience of sexual pleasure or ability to satisfy one's partner. Older literature reported the rate of sexual dysfunction to be in the range of 4–71% (Sandel, Williams, Dellapietra, & Derogatis, 1996), and this wide disparity is likely due to differences in the methodological design of studies. In more recent research, a prevalence rate of one-third to 50% of 208 TBI patients reported sexual dysfunction as measured by a sexuality questionnaire (Ponsford, Downing, & Stolwyk, 2013). In an attempt to refine the data with regard to time post injury,

the sexual functioning of 223 TBI patients (165 males/58 females) was evaluated with the Derogatis Interview for Sexual Function 1 year after injury (Derogatis, 1987, 1997). This self-report evaluates sexual cognition/fantasy, arousal, sexual behavior/experience, orgasm, and sexual drive/relationship, and the study concluded that men scored significantly below the normative sample scales on all except arousal, while women reported greater dysfunction in sexual cognition/fantasy as well as arousal (Sander et al., 2012). More recently, 986 moderate-to-severe TBI patients completed a brain injury questionnaire of sexuality at intervals of 1–20 years post injury, and factors such as older age, depression, shorter time post injury, and decreased independence in ADLs were associated with poorer sexual functioning (Ponsford et al., 2013). Sexual changes have been attributed to fatigue, low self-esteem, pain, decreased mobility (Downing, Stolwyk, & Ponsford, 2013), and unemployment and lower annual income (Bellamkonda & Zollman, 2014). These and other studies (Bivona et al., 2016; Moreno & McKerral, 2017) have helped to highlight an awareness of the biopsychosocial model of TBI and sexuality and emphasize not only the difficulties with the sexual act as a result of the injury, but also the roles that emotional features (such as mood and irritability), cognitive deficits (such as apathy, lack of insight, and empathy), and age and gender issues play in this complex human experience.

Another important aspect of TBI sexual dysfunction which has gained more attention in the literature is the impact of the injury on the sexual dysfunction of spouses and partners of these patients. There is caregiver stress since the partners are taking on roles previously fulfilled by the patient and there is also increased dependency on the partner by the TBI patient which can compromise intimacy and the emotional bond between them. In a recent multicenter study of 70 patients with mild-to-severe TBI, it was found that 20% of spouses reported sexual dysfunction and 44% reported dissatisfaction with sexual functioning. Older age of the partners and sexual functioning of the TBI patients were significant predictors of the well spouse/partner's sexual function (Sander et al., 2016). In contrast, factors cited as helpful to promote intimacy and to maintain strong relationships despite the impact of TBI include a strong pre-injury relationship, family bonds, spirituality, social support, and bonding through surviving the injury together (Gill, Sander, Robins, Mazzei, & Struchen, 2011). Attention to the well-being of spouses/partners as well as TBI patients is essential especially in light of the high rate of separation and divorce in this patient population. The importance of educating the rehabilitation team in feeling comfortable about addressing these issues must be underscored.

Patients with TBI can have a variety of physiological sexual dysfunctions including decreased drive, erectile dysfunction, and difficulties with orgasm. The clinical categorization of these various subtypes of sexual dysfunction as per DSM-5 (American Psychiatric Association, 2013) nosology is beyond the scope of this chapter, but it is of note that there is no longer any category of sexual dysfunction due to medical condition as in the previous versions of this manual. While most often patients suffer from hyposexuality as a result of the brain injury, hypersexuality can also occur, as was noted in 14% of subjects in one study (Kreutzer & Zasler, 1989). Of note, in a small minority of men with TBI, sexually aberrant behaviors

such as inappropriate touching, exhibitionism, or overt sexual aggression have also been reported (Simpson, Blaszczynski, & Hodgkinson, 1999). In an updated study, the point prevalence rate of this behavior was 8.9% (45/507 patients) with severe TBI. The manifestation of the inappropriate sexual behavior was mostly inappropriate sexual talk (57.9%), genital and nongenital touching (29.8%), and exhibitionism (10.5%) (Simpson, Sabaz, & Daher, 2013). Neuroanatomically, hyposexuality has been related to lesions of the medial orbital gyrus of the frontal lobe, hippocampus, anterior thalamus, and hypothalamus (Elliott & Biever, 1996), while hypersexuality can occur with damage to the frontal lobe and bilateral temporal lobes (Wesolowski, Zencius, & Burke, 1993; Zencius, Wesolowski, Burke, & Hough, 1990).

Vignette

A 40-year-old male suffered traumatic brain injury after a motor vehicle accident which caused injury to the right frontal–temporal brain regions. Subsequent to this, he was observed exposing himself and making inappropriate sexual overtures to female staff as well as family members. He began to masturbate in public places while using pornographic materials. His behavior is disruptive to his rehabilitation efforts and he is referred to the neuropsychiatrist for evaluation.

Assessment

As with other neuropsychiatric conditions, obtaining a history to try to narrow down the diagnostic possibilities is key, as sexual dysfunction after neurological insults can be due to either genital and/or nongenital causes. The patient and his/her sexual partner should be asked about the patient's premorbid and post-TBI sexual history, including marital status, sexual preference, sexual activities, sexual abuse, quality of relationships, libido, arousal, and physiological function (erection, ejaculation, vaginal lubrication, and orgasm). Any sexually intrusive behaviors—which can range from inappropriate remarks to aggressive behavior, including rape—should also be explored in the TBI patient (Bezeau, Bogod, & Mateer, 2004). Since many medications have sexual side effects, and because diseases such as diabetes or sexually transmitted diseases can cause sexual dysfunction, inquiry into these topics is pertinent. Endocrinological function is also particularly relevant since brain injuries which affect the pituitary gland, and hence hormonal levels, could be responsible for a patient's sexual problems. Decreased sensation or hypersensitivity, decreased mobility secondary to paralysis or orthopedic injuries, as well as tremor or balance problems, are all obvious impediments to sexually pleasurable activity and must be addressed as well. Prior psychiatric illness is relevant, as decreased or increased libido can be observed within the constellation of mood and anxiety disorders (Zasler & Martelli, 2005). A history of seizures is important to inquire about as

epilepsy is a common sequelae of TBI (about 12% in severe TBI) (Annegers, Grabow, Groover, & Laws, 1980), and those with temporal lobe epilepsy often suffer with hyposexuality. Since cognitive–behavioral and emotional problems can limit one's ability to effectively maintain an intimate relationship, it is important in the examination of the TBI patient to explore the impact of relevant issues such as poor concentration, memory deficits, motivation, lack of confidence, excessive dependency and loss of equality in the relationship, disinhibition, and insensitivity to a partner's needs. As part of the neuropsychiatrist's role, he/she can order lab tests such as hormone levels (follicle-stimulating hormone [FSH], luteinizing hormone [LH], estrogen, and testosterone) and then target appropriate ancillary consultations to the physiatrist, endocrinologist, gynecologist, or urologist to address those sexual issues which do not appear to be under his/her purview (Oddy, 2001; Zasler & Martelli, 2005).

Treatment

If the nature of the problem is ultimately determined to lie in the neuropsychiatric domain, the professional in this field can utilize different approaches to help the patient and his/her partner. In the above clinical case where hypersexuality is the clinical problem, one form of treatment would be to take advantage of the sexual side effects of antidepressants such as the SSRIs, which are known to decrease libido and cause problems in achieving ejaculation, orgasm, and erections (Krueger & Kaplan, 2002). Sometimes, mood stabilizers, especially anticonvulsants, are used to treat hypersexuality if this symptom is viewed as part of a manic or hypomanic state. If these fail, then sexual desire can be diminished with anti-androgens such as medroxyprogesterone (Britton, 1998) or depot-leuprolide acetate (Lupron) (Krueger & Kaplan, 2002) as was successfully done in the above-described clinical scenario. These medications are gonadotropin-releasing hormone analogs which cause a reduction in the pituitary production of LH and FSH, which in turn leads to a decrease in testosterone. Prior to starting this treatment, the patient requires baseline hormonal levels and a bone density evaluation, as bone loss can be a side effect of these agents (Krueger & Kaplan, 2002). A behavioral plan focused on the modification of these inappropriate actions should also be undertaken as part of the treatment.

Hyposexuality is a more common sexual dysfunction problem. Antidepressants, antipsychotics, anticonvulsants, and also antihypertensives, stimulant medications, and anticholinergics can be the source of decreased libido; therefore, dosage modification or elimination of the medication entirely may help to improve a patient's sexual interest and performance (Aloni & Katz, 1999). Conversely, the neuropsychiatrist must also assess whether depression is the underlying cause of the sexual dysfunction, in which case appropriate treatment may improve the patient's desire. Utilizing medications which do not have sexual side effects, such as bupropion, can be beneficial (Hirschfeld, 1999). If a depressed TBI patient has low testosterone

levels and sexual dysfunction, testosterone replacement may ameliorate both of these results (Zarrouf, Artz, Griffith, Sirbu, & Kommor, 2009). In patients with erectile dysfunction after TBI, the phosphodiesterase type 5 inhibitors may have a role (Simpson, McCann, & Lowy, 2016). Headaches and flushing as well as dyspepsia are common side effects (Lombardi, Nelli, Celso, Mencarini, & Del Popolo, 2012).

Psychotherapy, which can include both individual and couple's counseling to help the patient and his partner deal with the practical issues of sexual relations as well as emotional issues, should be part of the treatment paradigm. Since the reported rates of marital breakup after TBI are high, the role of psychotherapy in this area must be underscored. With a TBI group therapy format, patients can practice social skills with peers and have the opportunity to discuss common sexual problems and ways to cope with them (Katz & Aloni, 1999), while the availability of sexuality handbooks which address these topics can also be valuable resources for TBI patients who have sufficient cognitive abilities to benefit from this approach (Simpson & Long, 2004). Finally, in those cases of TBI patients with sexually intrusive behaviors, behavioral programs which focus on establishing clear boundaries in relationships, encourage adaptive and appropriate behaviors, and provide a relapse prevention plan have been demonstrated to be successful (Bezeau et al., 2004).

Conclusions

As has been observed from the patient vignettes in this chapter, the neuropsychiatric complications of TBI are numerous and complex. In reviewing our progress along the neuropsychiatric historical timeline, it is observed that we have advanced from the point where there was merely a glimmer of understanding about the possible existence of a relationship between brain and behavior to our more sophisticated, modern ideas about the brain and its definitive roles in emotion and cognition. Yet, despite learning about brain–behavior connections through the deficits suffered by TBI patients, much research still needs to be done to understand the intricate nature of these neuronal ties, as well as to improve the outcomes of our patients who have suffered these injuries. There is debate in the literature about whether cognitive and psychiatric sequelae of TBI are the result of specific brain lesions, psychological reactions to trauma, premorbid psychiatric illness [as was proposed in a study which noted an increased relative risk of 1.6 for subsequent TBI in patients who had had any indicator of psychiatric illness in the year prior to TBI (Fann et al., 2002)], or a combination thereof. For this reason, future studies of patients should be prospective in design using standardized diagnostic criteria which will more accurately categorize both the degrees of TBI (mild, moderate, severe) as well as the specific psychiatric syndrome. This will help to better predict outcomes of psychiatric comorbidity, cognitive impairment, and functional status, as well as to allocate resources appropriately to assist these patients in repairing their lives. In addition, randomized, blinded, placebo-controlled studies of psychopharmacological agents are crucial to providing a rational, consistent approach to treating the various

neuropsychiatric consequences of TBI. With these improvements as a start, patients who have had the misfortune of sustaining TBI can have the hope of enjoying an improved quality of life.

References

Achte, K. A., Hillbom, E., & Aalberg, V. (1969). Psychoses following war injuries. *Acta Psychiatrica Scandinavica, 45*, 1–18.

Adams, R. D., Victor, M., & Ropper, A. M. (1997). *Principles of neurology* (6th ed., pp. 344–366). New York, NY: McGraw-Hill Company. Chapter 17.

Al-Adawi, D. A., Burke, D. T., Huynh, C. C., Jacob, L., Knight, R., Shah, M. K., & Al-Hussaini, A. (2004). Apathy and depression in cross-cultural survivors of traumatic brain injury. *Journal of Neuropsychiatry and Clinical Neurosciences, 16*(4), 435–442.

Albicini, M., & McKinlay, A. (2018). Anxiety disorders in adults with childhood traumatic brain injury: Evidence of difficulties more than 10 years postinjury. *The Journal of Head Trauma Rehabilitation, 33*(3), 191–199.

Aloni, R., & Katz, S. (1999). A review of the effect of traumatic brain injury on the human sexual response. *Brain Injury, 13*, 269–280.

Alway, Y., Gould, K. R., Johnston, L., McKenzie, D., & Ponsford, J. (2016). A prospective examination of Axis I psychiatric disorders in the first 5 years following moderate to severe traumatic brain injury. *Psychological Medicine, 46*, 1331–1341.

Alway, Y., Gould, K. R., McKay, A., Johnston, L., & Ponsford, J. (2016). The evolution of post-traumatic stress disorder following moderate-to-severe traumatic brain injury. *Journal of Neurotrauma, 33*, 825–831.

American Psychiatric Association. (2013). *Diagnostic and statistical manual of mental disorders: DSM-5*. Washington, DC: American Psychiatric Press.

Andersson, S., & Bergedalen, A. M. (2002). Cognitive correlates of apathy in traumatic brain injury. *Neuropsychiatry, Neuropsychology, and Behavioral Neurology, 15*, 184–191.

Andersson, S., Gundersen, P. M., & Finset, A. (1999). Emotional activation during therapeutic interaction in traumatic brain injury: Effect of apathy, self-awareness and implications for rehabilitation. *Brain Injury, 13*, 393–404.

Andersson, S., Krogstad, J. M., & Finset, A. (1999). Apathy and depressed mood in acquired brain damage: Relationship to lesion localization and psychophysiological reactivity. *Psychological Medicine, 29*, 447–456.

Annegers, J. F., Grabow, J. D., Groover, R. V., & Laws, E. R. (1980). Seizures after head trauma: A population study. *Neurology, 30*, 683–689.

Arauco, A. R., Grados, M. A., & Vizcarra, D. (2008). A diffuse brain injury leading to a complex neurobehavioral syndrome. *Journal of Neuropsychiatry and Clinical Neurosciences, 20*(2), 240–241.

Arch, J. J., & Craske, M. G. (2009). First-line treatment: A critical appraisal of cognitive behavioral therapy developments and alternatives. *Psychiatric Clinics of North America, 32*(3), 525–547.

Arciniegas, D., Adler, L., Topkoff, J., Cawthra, E., Filley, C. M., & Reite, M. (1999). Attention and memory dysfunction after traumatic brain injury: Cholinergic mechanisms, sensory gating and a hypothesis for further investigation. *Brain Injury, 13*, 1–13.

Arciniegas, D. B., & Beresford, T. P. (2001). *Neuropsychiatry–An introductory approach* (p. 376). New York, NY: Cambridge University Press.

Arciniegas, D. B., Harris, S. N., & Brousseau, K. M. (2003). Psychosis following traumatic brain injury. *International Review of Psychiatry, 15*, 328–340.

Arco, L. (2008). Neurobehavioural treatment for obsessive-compulsive disorder in an adult with traumatic brain injury. *Neuropsychological Rehabilitation, 18*(1), 109–124.

Ardila, A. (2018). Psychiatric disorders associated with acquired brain pathology. *Applied Neuropsychology. Adult*, 1–7.

Armento, M. D., & Hopko, D. R. (2007). The environmental reward observation scale (EROS): Development, validity, and reliability. *Behavior Therapy, 38*(2), 107–119.

Arnould, A., Rochat, L., Azouvi, P., & Van der Linden, M. (2013). A multidimensional approach to apathy after traumatic brain injury. *Neuropsychology Review, 23*(3), 210–233.

Arnould, A., Rochat, L., Azouvi, P., & Van der Linden, M. (2018). Longitudinal course and predictors of apathetic symptoms after severe traumatic brain injury. *Archives of Clinical Neuropsychology, 33*, 808–820.

Arnould, A., Rochat, L., Dromer, E., Azouvi, P., & Van der Linden, M. (2018). Does multitasking mediate the relationships between episodic memory, attention, executive functions and apathetic manifestations in traumatic brain injury? *Journal of Neuropsychology, 12*, 101–119.

Ashman, T., Cantor, J. B., Tsaousides, T., Spielman, L., & Gordon, W. (2014). Comparison of cognitive behavioral therapy and supportive psychotherapy for the treatment of depression following traumatic brain injury: A randomized controlled trial. *The Journal of Head Trauma Rehabilitation, 29*(6), 467–478.

Ashman, T. A., Cantor, J. B., Gordon, W. A., Spielman, L., Flanagan, S., Ginsberg, A., … Greenwald, B. (2009). A randomized controlled trial of sertraline for the treatment of depression in persons with traumatic brain injury. *Archives of Physical Medicine and Rehabilitation, 90*(5), 733–740.

Ashman, T. A., Spielman, L. A., Hibbard, M. R., Silver, J. M., Chandna, T., & Gordon, W. A. (2004). Psychiatric challenges in the first six years after traumatic brain injury: Cross sequential analyses of axis I disorders. *Archives of Physical Medicine and Rehabilitation, 85*, S36–S42.

Azouvi, P., Arnould, A., Dromer, E., & Vallat-Azouvi, C. (2017). Neuropsychology of traumatic brain injury: An expert overview. *Revue Neurologique, 173*, 461–472.

Azouvi, P., Jokic, C., Attal, N., Denys, P., Markabi, S., & Bussel, B. (1999). Carbamazepine in agitation and aggressive behavior following severe closed-head injury: Results of an open trial. *Brain Injury, 13*, 797–804.

Babin, P. R. (2003). Diagnosing depression in persons with brain injuries: A look at theories, the DSM-IV and depression measures. *Brain Injury, 17*, 889–900.

Bacciardi, S., Maremmani, A. G., Nikoo, N., Cambiolo, L., Schutz, C., Jang, K., & Krausz, M. (2017). Is bipolar disorder associated with traumatic brain injury in the homeless? *Rivista di Psichiatria, 52*(1), 40–46.

Bahraini, N. H., Breshears, R. E., Hernandez, T. D., Schneider, A. L., Forster, J. E., & Brenner, L. A. (2014). Traumatic brain injury and posttraumatic stress disorder. *Psychiatric Clinics of North America, 37*(1), 55–74.

Bakchine, S., Lacomblez, L., Benoit, N., Parisot, D., Chain, F., & Lhermitte, F. (1989). Manic-like state after bilateral orbitofrontal and right temporoparietal injury: Efficacy of clonidine. *Neurology, 39*, 777–781.

Banos, J. H., Novack, T. A., Brunner, R., Renfroe, S., Lin, H. Y., & Meythaler, J. (2010). Impact of early administration of sertraline on cognitive and behavioral recovery in the first year after moderate to severe traumatic brain injury. *The Journal of Head Trauma Rehabilitation, 25*(5), 357–361.

Barker, F. G. (1995). Phineas among the phrenologists: The American crowbar case and nineteenth century theories of cerebral localization. *Journal of Neurosurgery, 82*, 672–682.

Barman, R., Kumar, S., Pagadala, B., & Detweiler, M. B. (2017). Cocaine abuse, traumatic brain injury, and preexisting brain lesions as risk factors for bupropion-associated psychosis. *Journal of Clinical Psychopharmacology, 37*(4), 459–463.

Batty, R. A., Francis, A., Thomas, N., Hopwood, M., Ponsford, J., & Rossell, S. L. (2016). A brief neurocognitive assessment of patients with psychosis following traumatic brain injury (PFTBI): Use of the Repeatable battery for the Assessment of Neuropsychological Status (RBANS). *Psychiatry Research, 237*, 27–36.

Baxter, L. R., Schwartz, J. M., Bergman, K. S., Szuba, M. P., Guze, B. H., Mazziotta, J. C., … Phelps, M. E. (1992). Caudate glucose metabolic rate changes with both drug and behavior therapy for obsessive-compulsive disorder. *Archives of General Psychiatry, 49*, 681–689.

Bellamkonda, E., & Zollman, F. (2014). Relationship between employment status and sexual functioning after traumatic brain injury. *Brain Injury, 28*(8), 1063–1069.

Bennouna-Greene, M., Frank, J., Kremer, S., Bursztejn, C., & Foucher, J. (2010). Manic psychosis and auditory hallucinations following traumatic brain injury in a 13-year-old boy. *The Journal of Clinical Psychiatry, 71*(4), 506–507.

Berthier, M. L., Kulisevsky, J., Gironell, A., & Lopez, O. L. (2001). Obsessive-compulsive disorder and traumatic brain injury: Behavioral, cognitive and neuroimaging findings. *Neuropsychiatry, Neuropsychology, and Behavioral Neurology, 14*, 23–31.

Bezeau, S. C., Bogod, N. M., & Mateer, C. A. (2004). Sexually intrusive behavior following brain injury: Approaches to assessment and rehabilitation. *Brain Injury, 18*, 299–313.

Bhalerao, S. U., Geurtjens, C., Thomas, G. R., Kitamura, C. R., Zhou, C., & Marlborough, M. (2013). Understanding the neuropsychiatric consequences associated with significant traumatic brain injury. *Brain Injury, 27*(7-8), 767–774.

Bivona, U., Antonucci, G., Contrada, M., Rizza, F., Leoni, F., Zasler, N. D., & Formisano, R. (2016). A biopsychosocial analysis of sexuality in adult males and their partners after severe traumatic brain injury. *Brain Injury, 30*(9), 1082–1095.

Bodnar, C.N., Morganti, J.M., & Bachstetter, A.D. (2018). Depression following a tramatic brain injury: Uncovering cytokine dysregulation as a pathogenic mechanism. *Neural Regeneration Research, 13*(10),1693–1704.

Bogner, J., Barrett, R. S., Hammond, F. M., Horn, S. D., Corrigan, J. D., Rosenthal, J., … Garmoe, W. (2015). Predictors of agitated behavior during inpatient rehabilitation for traumatic brain injury. *Archives of Physical Medicine and Rehabilitation, 96*(8 Suppl 3), S274–S281.

Bogner, J. A., Corrigan, J. D., Fugate, L., Mysiw, W. J., & Clinchot, D. (2001). Role of agitation in prediction of outcomes after traumatic brain injury. *American Journal of Physical Medicine & Rehabilitation, 80*, 636–644.

Bombardier, C. H., Fann, J. R., Ludman, E. J., Vannoy, S. D., Dyer, J. R., Barber, J. K., & Temkin, N. R. (2017). The relations of cognitive, behavioral, and physical activity variables to depression severity in traumatic brain injury: Reanalysis of data from a randomized controlled trial. *The Journal of Head Trauma Rehabilitation, 32*(5), 343–353.

Bombardier, C. H., Fann, J. R., Temkin, N. R., Esselman, P. C., Barber, J., & Dikmen, S. S. (2010). Rates of major depressive disorder and clinical outcomes following traumatic brain injury. *Journal of the American Medical Association, 303*(19), 1938–1945.

Bombardier, C. H., Hoekstra, T., Dikmen, S., & Fann, J. R. (2016). Depression trajectories during the first year after traumatic brain injury. *Journal of Neurotrauma, 33*, 2115–2124.

Bontke, C. F., Rattok, J., & Boake, C. (1996). Do patients with mild brain injuries have posttraumatic stress disorder too? *The Journal of Head Trauma Rehabilitation, 11*, 95–102.

Branca, B., & Lake, A. E. (2004). Psychological and neuropsychological integration in multidisciplinary pain management after TBI. *The Journal of Head Trauma Rehabilitation, 19*, 40–57.

Brenner, L. A., Bahraini, N., Homaifar, B. Y., Monteith, L. L., Nagamoto, H., Dorsey-Holliman, B., & Forster, J. E. (2015). Executive functioning and suicidal behavior among veterans with and without a history of traumatic brain injury. *Archives of Physical Medicine and Rehabilitation, 96*, 1411–1418.

Britton, K. R. (1998). Medroxyprogesterone in the treatment of aggressive hypersexual behavior in traumatic brain injury. *Brain Injury, 12*, 703–707.

Brooke, M. M., Patterson, D. R., Questad, K. A., Cardenas, D., & Farrel-Roberts, L. (1992). The treatment of agitation during initial hospitalization after traumatic brain injury. *Archives of Physical Medicine and Rehabilitation, 73*, 917–921.

Brooke, M. M., Questad, K. A., Patterson, D. R., & Bashak, K. J. (1992). Agitation and restlessness after closed head injury: A prospective study of 100 consecutive admissions. *Archives of Physical Medicine and Rehabilitation, 73*, 320–323.

Brundin, L., Bryleva, E. Y., & Rajamani, K. T. (2017). Role of inflammation in suicide: From mechanisms to treatment. *Neuropsychopharmacology, 42*, 271–283.

Bryant, R. A. (2001). Post-traumatic stress disorder and mild brain injury: Controversies, causes and consequences. *Journal of Clinical and Experimental Neuropsychology, 23*, 718–728.

Bryant, R. A. (2011). Acute stress disorder as a predictor of posttraumatic stress disorder. *Journal of Clinical Psychiatry, 72*, 233–239.

Bryant, R. A., Nickerson, A., Creamer, M., O'Donnell, M., Forbes, D., Galatzer-Levy, I., … Silove, D. (2015). Trajectory of post-traumatic stress following traumatic injury: 6-year follow-up. *The British Journal of Psychiatry, 206*, 417–423.

Bryant, R. A., O'Donnell, M. L., Creamer, M., McFarlane, A. C., Clark, C. R., & Silove, D. (2010). The psychiatric sequelae of traumatic injury. *The American Journal of Psychiatry, 167*, 312–320.

Butler, P. V. (2000). Diurnal variation in Cotard's syndrome (copresent with Capgras delusion) following traumatic brain injury. *The Australian and New Zealand Journal of Psychiatry, 34*, 684.

Buysse, D. J., Reynolds, C. F., III, Monk, T. H., Berman, S. R., & Kupfer, D. J. (1989). The Pittsburgh Sleep Quality Index: A new instrument for psychiatric practice and research. *Psychiatry Research, 28*(2), 193–213.

Calabrese, J. R., Bowden, C. L., McElroy, S. L., & Cookson, J. (1999). Spectrum of activity of lamotrigine in treatment-refractory bipolar disorder. *American Journal of Psychiatry, 156*, 1019–1023.

Cassidy, J. W. (1989). Fluoxetine: A new serotonergically active antidepressant. *The Journal of Head Trauma Rehabilitation, 4*, 67–69.

Cavinato, M., Iaia, V., & Piccione, F. (2012). Repeated sessions of sub-threshold 20-Hz rTMS. Potential cumulative effects in a brain-injured patient. *Clinical Neurophysiology, 123*, 1893–1895.

Chandler, M. C., Barnhill, J. L., & Gualtieri, C. T. (1988). Amantadine for the agitated head-injury patient. *Brain Injury, 2*, 309–311.

Chatham-Showalter, P. E. (1996). Carbamazepine for combativeness in acute traumatic brain injury. *Journal of Neuropsychiatry & Clinical Neurosciences, 8*, 96–99.

Chaves, C., Trzesniak, C., Derenusson, G. N., Araujo, D., Marchado-De-Sousa, L. W. A. J. P., Carlotti, C. G., Jr., … Hallak, J. E. C. (2012). Late-onset social anxiety disorder following traumatic brain injury. *Brain Injury, 26*(6), 882–886.

Chen, Q. L., Calcagno, H. E., & Shad, M. (2018). Efficacy of dextromethorphan/quinidine for patients with psychosis-related aggression: A retrospective case series. *The Primary Care Companion for CNS Disorders, 20*(3), 18m02284.

Chiu, H. Y., Lin, E. Y., Chiu, H. T., & Chen, P. Y. (2017). A feasibility randomized controlled crossover trial of home-based warm footbath to improve sleep in the chronic phase of traumatic brain injury. *Journal of Neuroscience Nursing, 49*(6), 380–385.

Cicerone, K. D. (2002). Remediation of 'working attention' in mild traumatic brain injury. *Brain Injury, 16*, 185–195.

Cittolin-Santos, G. F., Fredeen, J. C., & Cotes, R. O. (2017). A case report of mania and psychosis five months after traumatic brain injury successfully treated using olanzapine. *Hindawi Case Reports in Psychiatry, 2017*, 7541307. 3 pages.

Ciurli, P., Formisano, R., Bivona, U., Cantagallo, A., & Angelelli, P. (2011). Neuropsychiatric disorders in persons with severe traumatic brain injury: Prevalence, phenomenology, and relationship with demographic, clinical, and functional features. *The Journal of Head Trauma Rehabilitation, 26*(2), 116–126.

Clark, A. F., & Davison, K. (1987). Mania following head injury. A report of two cases and a review of the literature. *British Journal of Psychiatry, 150*, 841–844.

Cnossen, M. C., Scholten, A. C., Lingsma, H. F., Synnot, A., Haagsma, J., Steyerberg, E. W., & Polinder, S. (2017). Predictors of major depression and posttraumatic stress disorder following traumatic brain injury: A systematic review and meta-analysis. *The Journal of Neuropsychiatry and Clinical Neurosciences, 29*(3), 206–224.

Coetzer, R. (2011). Does memory impairment exclude a diagnosis of OCD after traumatic brain injury? *Journal of Neuropsychiatry and Clinical Neurosciences, 22*(3), E12.

Collicutt McGrath, J. (2008). Fear of falling after brain injury. *Clinical Rehabilitation, 22*, 635–645.

Corrigan, J. D. (1989). Development of a scale for assessment of agitation following traumatic brain injury. *Journal of Clinical and Experimental Neuropsychology, 11*, 261–277.

Crane, P. K., Gibbons, L. E., Dams-O'Connor, K., Trittschuh, E., Leverenz, J. B., Keene, C. D., … Larson, E. B. (2016). Association of traumatic brain injury with late-life neurodegenerative conditions and neuropathologic findings. *Journal of the American Medical Association Neurology, 73*(9), 1062–1069.

Crow, S., Meller, W., Christenson, G., & Mackenzie, T. (1996). Use of ECT after brain injury. *Convulsive Therapy, 12*, 113–116.

Cummings, J. L. (2000). Cholinesterase inhibitors: A new class of psychotropic compounds. *American Journal of Psychiatry, 157*, 4–15.

Cummings, J. L., & Back, C. (1998). The cholinergic hypothesis of neuropsychiatric symptoms in Alzheimer's disease. *The American Journal of Geriatric Psychiatry, 6*, S64–S78.

Cummings, J. L., Mega, M., Gray, K., Rosemberg-Thompson, S., & Gornbein, J. (1994). The Neuropsychiatric Inventory: Comprehensive assessment of psychopathology in dementia. *Neurology, 44*, 2308–2314.

Cusimano, M. D., Holmes, S. A., Sawicki, C., & Topolovec-Vranic, J. (2014). Assessing aggression following traumatic brain injury: A systematic review of validated aggression scales. *The Journal of Head Trauma Rehabilitation, 29*(2), 172–184.

Daniels, J. P., & Felde, A. (2008). Quetiapine treatment for mania secondary to brain injury in 2 patients. *Journal of Clinical Psychiatry, 69*(3), 497–498.

David, A.S. & Prince, M. (2005). Psychosis following head injury: A critical review. The *Journal of Neurology, Neurosurgery and Psychiatry, 76*(Suppl 1):53–60.

Davison, K., & Bagley, C. R. (1969). Schizophrenia-like psychoses associated with organic disorders of the central nervous system: A review of the literature. In R. N. Herrington (Ed.), *Current problems in neuropsychiatry. Schizophrenia, epilepsy, the temporal lobe* (pp. 113–184). London: Headley.

De Guise, E., LeBlanc, J., Feyz, M., Lamoureux, J., & Greffou, S. (2017). Prediction of behavioural and cognitive deficits in patients with traumatic brain injury at an acute rehabilitation setting. *Brain Injury, 31*(8), 1061–1068.

De Guzman, E., & Ament, A. (2017). Neurobehavioral management of traumatic brain injury in the critical care setting: An update. *Critical Care Clinics, 33*(3), 423–440.

Deb, S., Lyons, S. I., Koutzoukis, C., Ali, I., & McCarthy, G. (1999). Rate of psychiatric illness one year after traumatic brain injury. *American Journal of Psychiatry, 156*, 374–378.

DeBoussard, C. N., Lannsjo, M., Stenberg, M., Stalnacke, B. M., & Godbolt, A. K. (2017). Behavioural problems in the first year after severe traumatic brain injury: A prospective multicentre study. *Clinical Rehabilitation, 31*(4), 555–566.

Derivan, A., Haskins, T., Rudolph, R., Pallay, A., & Aguiar, L. (June, 1998). *Double-blind placebo-controlled study of once daily venlafaxine XR in outpatients with generalized anxiety disorder*. Abstract presented at the American Psychiatric Association Annual Meeting, Toronto, Canada.

Derogatis, L. P. (1987). *Derogatis interview for sexual functioning-SR*. Baltimore, MD: Clinical Psychometric Research.

Derogatis, L. P. (1997). The Derogatis interview for sexual functioning (DISF/DISF-SR): An inventory report. *The Journal of Sex Education and Therapy, 23*, 291–304.

Dikmen, S. S., Bombardier, C. H., Machamer, J. E., Fann, J. R., & Temkin, N. R. (2004). Natural history of depression in traumatic brain injury. *Archives of Physical Medicine and Rehabilitation, 85*, 1457–1464.

Dockree, P. M., Kelly, S. P., Roche, R. A. P., Hogan, M. J., Reilly, R. B., & Robertson, I. H. (2004). Behavioral and physiological impairments of sustained attention after traumatic brain injury. *Cognitive Brain Research, 20*, 403–414.

Douglass, A. R., & Smyth, U. (2018). A case report of guardian-consent forced paliperidone palmitate for behavioral disturbance due to traumatic brain injury. *Mental Health Clinician, 8*(3), 155–158.

Downing, M. G., Stolwyk, R., & Ponsford, J. L. (2013). Sexual changes in individuals with traumatic brain injury: A control comparison. *The Journal of Head Trauma Rehabilitation, 28*(3), 171–178.

Drange, O. K., Vaaler, A. E., Morken, G., Andreassen, O. A., Malt, U. F., & Finseth, P. I. (2018). Clinical characteristics of patients with bipolar disorder and premorbid traumatic brain injury: A cross-sectional study. *International Journal of Bipolar Disorders, 6*, 19.

Dreer, L. E., Tang, X., Nakase-Richardson, R., Pugh, M. J., Cox, M. K., Bailey, E. K., … Brenner, L. A. (2018). Suicide and traumatic brain injury: A review by clinical researchers from the National Institute for Disability and Independent Living Rehabilitation Research (NIDILRR) and Veterans Health Administration traumatic brain injury model systems. *Current Opinion in Psychology, 22*, 73–78.

Dretsch, M. N., Williams, K., Emmerich, T., Crynen, G., Ait-Ghezala, G., Chaytow, H., … Iverson, G. L. (2016). Brain-derived neurotropic factor polymorphisms, traumatic stress, mild traumatic brain injury, and combat exposure contribute to postdeployment traumatic stress. *Brain and Behavior: A Cognitive Neuroscience Perspective, 6*(1), e00392.

Duclos, C., Beauregard, M. P., Bottari, C., Ouellet, M. C., & Gosselin, N. (2015). The impact of poor sleep on cognition and activities of daily living after traumatic brain injury: A review. *Australian Occupational Therapy Journal, 62*(1), 2–12.

Duclos, C., Dumont, M., Blais, H., Paquet, J., Laflamme, E., de Beaumont, L., … Gosselin, N. (2014). Rest-activity cycle disturbances in the acute phase of moderate to severe traumatic brain injury. *Neurorehabilitation and Neural Repair, 28*(5), 472–482.

Ekinci, O., Direk, M. C., Ekinci, N., & Okuyaz, C. (2016). Manic symptoms due to methylphenidate use in an adolescent with traumatic brain injury. *Clinical Psychopharmacology and Neuroscience, 14*(3), 314–317.

Elliott, M. L., & Biever, L. S. (1996). Head injury and sexual dysfunction. *Brain Injury, 10*, 703–717.

Elovic, E. (2000). Use of provigil for underarousal following TBI. *The Journal of Head Trauma Rehabilitation, 15*, 1068–1071.

Elovic, E. P., Lansang, R., Li, Y., & Ricker, J. H. (2003). The use of atypical antipsychotics in traumatic brain injury. *The Journal of Head Trauma Rehabilitation, 18*, 177–195.

Emilien, G., & Maloteaux, J. M. (1998). Current therapeutic uses and potential of beta-adrenoceptor agonists and antagonists. *European Journal of Clinical Pharmacology, 53*, 389–404.

Evans, R. W., Gualtieri, C. T., & Patterson, D. (1987). Treatment of chronic closed head injury with psychostimulant drugs: A controlled case study and an appropriate evaluation procedure. *The Journal of Nervous and Mental Disease, 175*, 106–110.

Failla, M. D., Juengst, S. B., Graham, K. M., Arenth, P. M., & Wagner, A. K. (2016). Effects of depression and antidepressant use on cognitive deficits and functional cognition following severe traumatic brain injury. *The Journal of Head Trauma Rehabilitation, 31*(6), E62–E73.

Fann, J. R., Bombardier, C. H., Temkin, N., Esselman, P., Warms, C., Barber, J., & Dikmen, S. (2017). Sertraline for major depression during the year following traumatic brain injury: A randomized controlled study. *The Journal of Head Trauma Rehabilitation, 32*(5), 332–342.

Fann, J. R., Burington, B., Leonetti, A., Jaffe, K., Katon, W. J., & Thompson, R. S. (2004). Psychiatric illness following traumatic brain injury in an adult health maintenance organization population. *Archives of General Psychiatry, 61*, 53–61.

Fann, J. R., Katon, W. J., Uomoto, J. M., & Esselman, P. C. (1995). Psychiatric disorders and functional disability in outpatients with traumatic brain injuries. *American Journal of Psychiatry, 152*, 1493–1499.

Fann, J. R., Leonetti, A., Jaffe, K., Katon, W. J., Cummings, P., & Thompson, R. S. (2002). Psychiatric illness and subsequent traumatic brain injury: A case control study. *Journal of Neurology, Neurosurgery, and Psychiatry, 72*, 615–620.

Fann, J. R., Ribe, A. R., Pederson, H. S., Fenger-Gron, M., Christensen, J., Benros, M. E., & Vestergaard, M. (2018). Long-term risk of dementia among people with traumatic brain injury in Denmark: A population-based observational cohort study. *Lancet Psychiatry, 5*, 424–431.

Fann, J. R., Uomoto, J. M., & Katon, W. J. (2000). Sertraline in the treatment of major depression following mild traumatic brain injury. *Journal of Neuropsychiatry & Clinical Neurosciences, 12*, 226–232.

Fann, J. R., Uomoto, J. M., & Katon, W. J. (2001). Cognitive improvement with treatment of depression following mild traumatic brain injury. *Psychosomatics, 42*, 48–54.

Fazel, S., Wolf, A., Pillas, D., Lichtenstein, P., & Langstrom, N. (2014). Suicide, fatal injuries, and other causes of premature mortality in patients with traumatic brain injury: A 41-year Swedish population study. *The Journal of the American Medical Association Psychiatry, 71*(3), 326–333.

Federoff, J. P., Starkstein, S. E., Forrester, A. W., Geisler, F. H., Jorge, R. E., Arndt, S. V., & Robinson, R. G. (1992). Depression in patients with acute traumatic brain injury. *American Journal of Psychiatry, 149*, 918–923.

Ferguson, P.L., Smith, G.M., Wannamaker, B.B., Thurman, D.J., Pickelsimer, E.E., & Selassie, A.W. (2010). A population-based study of risk of epilepsy after hospitalization for traumatic brain injury. *Epilepsia, 51*(5), 891–898.

Finger, S. (2000). *Minds behind the brain—A history of the pioneers and their discoveries* (Vol. 9, pp. 13–15). New York, NY: Oxford University Press.

Fisher, L. B., Pedrelli, P., Iverson, G. L., Bergquist, T. F., Bombardier, C. H., Hammond, F. M., … Zafonte, R. (2016). Prevalence of suicidal behaviour following traumatic brain injury: Longitudinal follow-up data from the NIDRR traumatic brain injury model systems. *Brain Injury, 30*(11), 1311–1318.

Fitzgerald, P. B., Brown, T. L., Marston, N. A., Daskalakis, Z. J., DeCastella, A., & Kulkarni, J. (2003). Transcranial magnetic stimulation in the treatment of depression: A double-blind, placebo-controlled trial. *Archives of General Psychiatry, 60*, 1002–1008.

Fitzgerald, P. B., Hoy, K. E., Maller, J. J., Herring, S., Segrave, R., McQueen, S., … Daskalakis, Z. J. (2011). Transcranial magnetic stimulation for depression after a traumatic brain injury: A case study. *The Journal of ECT, 27*(1), 38–40.

Fleminger, S., Greenwood, R. R. J., & Oliver, D. L. (2006). Pharmacological management for agitation and aggression in people with acquired brain injury. *Cochrane Database of Systematic Reviews*, (4), CD003299.

Fralick, M., Sy, E., Hassan, A., Burke, M. J., Mostofsky, E., & Karsies, T. (2018). Associate of concussion with the risk of suicide: A systematic review and meta-analysis. *The Journal of the American Medical Association Neurology*, E1–E9.

Frederiks, C., Hofmann, M. T., & Reichard, R. (2002). Advances in diagnosis and treatment of Alzheimer's disease. *Hospital Physician, 38*, 47–54.

Freinhar, J. P., & Alvarez, W. A. (1986). Clonazepam treatment of organic brain syndromes in three elderly patients. *Journal of Clinical Psychiatry, 47*, 525–526.

Fujii, D., & Ahmed, I. (2002). Characteristics of psychotic disorder due to traumatic brain injury: An analysis of case studies in the literature. *Journal of Neuropsychiatry & Clinical Neurosciences, 14*, 130–140.

Fujii, D., Ahmed, I., & Hishinuma, E. (2004). A neuropsychological comparison of psychotic disorder following traumatic brain injury, traumatic brain injury without psychotic disorder and schizophrenia. *Journal of Neuropsychiatry & Clinical Neurosciences, 16*, 306–314.

Fujii, D., & Fujii, D. C. (2012). Psychotic disorder due to traumatic brain injury: Analysis of case studies in the literature. *The Journal of Neuropsychiatry and Clinical Neurosciences, 24*(3), 278–289.

Fujii, D. E., & Ahmed, I. (2001). Risk factors in psychosis secondary to traumatic brain injury. *Journal of Neuropsychiatry & Clinical Neurosciences, 13*, 61–69.

Gill, C. J., Sander, A. M., Robins, N., Mazzei, D., & Struchen, M. A. (2011). Exploring experiences of intimacy from the viewpoint of individuals with traumatic brain injury and their partners. *The Journal of Head Trauma Rehabilitation, 26*(1), 56–68.

Gilsanz, P., Albers, K., & Schnaider Beeri, M. (2018). Traumatic brain injury associated with dementia risk among people with type 1 diabetes. *Neurology, 91*, e1611–e1618.

Glenn, M. B., Wroblewski, B., Parziale, J., Levine, L., Whyte, J., & Rosenthal, M. (1989). Lithium carbonate for aggressive behavior or affective instability in ten brain-injured patients. *American Journal of Physical Medicine and Rehabilitation, 68*, 221–226.

Goldberg, E., Gertsman, L. J., Mattis, S., Hughes, J. E., Sirio, C. A., & Bilder, R. M. (1982). Selective effects of cholinergic treatment on verbal memory in post-traumatic amnesia. *Journal of Clinical Neuropsychology, 4*, 219–234.

Gomez, R., Skilbeck, C., Thomas, M., & Slatyer, M. (2017). Growth mixture modeling of depression symptoms following traumatic brain injury. *Frontiers in Psychology, 8*, 1320.

Gomez-Hernandez, R., Max, J. E., Kosier, T., Paradiso, S., & Robinson, R. G. (1997). Social impairment and depression after traumatic brain injury. *Archives of Physical Medicine and Rehabilitation, 78*, 1321–1326.

Gould, K. R., Ponsford, J. L., & Spitz, G. (2014). Association between cognitive impairments and anxiety disorders following traumatic brain injury. *Journal of Clinical and Experimental Neuropsychology, 36*(1), 1–14.

Grados, M. A. (2003). Obsessive-compulsive disorder after traumatic brain injury. *International Review of Psychiatry, 15*, 350–358.

Greendyke, R. M., Kanter, D. R., Schuster, D. B., Verstreate, S., & Wootton, J. (1986). Propranolol treatment of assaultive patients with organic brain disease. A double-blind crossover, placebo-controlled study. *The Journal of Nervous and Mental Disease, 174*, 290–294.

Griffin, S. L., van Reekum, R., & Masanic, C. (2003). A review of cholinergic agents in the treatment of neurobehavioral deficits following traumatic brain injury. *Journal of Neuropsychiatry & Clinical Neurosciences, 15*, 17–26.

Grima, N. A., Ponsford, J. L., & Pase, M. P. (2017). Sleep complications following traumatic brain injury. *Current Opinion in Pulmonary Medicine, 23*, 493–499.

Gualtieri, C. T. (1991a). Buspirone for the behavior problems of patients with organic brain disorders. *Journal of Clinical Psychopharmacology, 11*, 280–281.

Gualtieri, C. T. (1991b). Buspirone: Neuropsychiatric effects. *The Journal of Head Trauma Rehabilitation, 6*, 90–92.

Gualtieri, T., & Cox, D. R. (1991). The delayed neurobehavioral sequelae of traumatic brain injury. *Brain Injury, 5*, 219–232.

Gualtieri, T. C., & Evans, R. W. (1988). Stimulant treatment for the neurobehavioral sequelae of traumatic brain injury. *Brain Injury, 2*, 273–290.

Guerreiro, D. F., Navarro, R., Silva, M., Carvalho, M., & Gois, C. (2009). Psychosis secondary to traumatic brain injury. *Brain Injury, 23*(4), 358–361.

Hale, M. S., & Donaldson, J. O. (1982). Lithium carbonate in the treatment of organic brain syndrome. *The Journal of Nervous and Mental Disease, 170*, 362–365.

Hammond, F. M., Malec, J. F., Zafonte, R. D., Sherer, M., Bogner, J., Dikmen, S., … Moser, E. A. (2017). Potential impact of amantadine on aggression in chronic traumatic brain injury. *The Journal of Head Trauma Rehabilitation, 32*(5), 308–318.

Hammond, F. M., Sauve, W., Ledon, F., David, C., & Formella, A. E. (2018). Safety, tolerability, and effectiveness of dextromethorphan/quinidine for pseudobulbar affect among study participants with traumatic brain injury: Results from the PRISM-II open label study. *Physical Medicine and Rehabilitation, 10*(10), 993–1003.

Hamner, M. B., Brodrick, P. S., & Labbate, L. A. (2001). Gabapentin in PTSD: A retrospective, clinical series of adjunctive therapy. *Annals of Clinical Psychiatry, 13*, 141–146.

Handel, S. F., Ovitt, L., Spiro, J. R., & Rao, V. (2007). Affective disorder and personality change in a patient with traumatic brain injury. *Psychosomatics, 48*(1), 67–70.

Hart, T., Fann, J. R., Chervoneva, I., Juengst, S. B., Rosenthal, J. A., Krellman, J. W., … Kroenke, K. (2016). Prevalence, risk factors, and correlates of anxiety at 1 year after moderate to severe traumatic brain injury. *Archives of Physical Medicine and Rehabilitation, 97*, 701–707.

Heinrich, T. W., & Junig, J. T. (2004). Recurrent mania associated with repeated brain injury. *General Hospital Psychiatry, 26*, 490–492.

Hendrickson, R. C., & Raskind, M. A. (2016). Noradrenergic dysregulation in the pathophysiology of PTSD. *Experimental Neurology, 284*, 181–195.

Hendrickson, R. C., Schindler, A. G., & Pagulayan, K. F. (2018). Untangling PTSD and TBI: Challenges and strategies in clinical care. *Current Neurology and Neuroscience Reports, 18*, 106.

Herrmann, N., & Lanctot, K. L. (2005). Do atypical antipsychotics cause stroke? *CNS Drugs, 19*, 91–103.

Hertzberg, M. A., Butterfield, M. I., Feldman, M. E., Beckham, J. C., Sutherland, S. M., Connor, K. M., & Davidson, R. T. (1999). A preliminary study of lamotrigine for the treatment of post-traumatic stress disorder. *Biological Psychiatry, 45*, 1226–1229.

Hesdorffer, D. C., Rauch, S. L., & Tamminga, C. A. (2009). Long-term psychiatric outcomes following traumatic brain injury: A review of the literature. *The Journal of Head Trauma Rehabilitation, 24*(6), 452–459.

Hibbard, M. R., Ashman, T. A., Spielman, L. A., Chun, D., Charatz, H. J., & Melvin, S. (2004). Relationship between depression and psychosocial functioning after traumatic brain injury. *Archives of Physical Medicine and Rehabilitation, 85*, S43–S53.

Hibbard, M. R., Uysal, S., Kepler, K., Bogdany, J., & Silver, J. (1998). Axis I Psychopathology in individuals with traumatic brain injury. *The Journal of Head Trauma Rehabilitation, 13*, 24–39.

Hiott, D. W., & Labbate, L. (2002). Anxiety disorders associated with traumatic brain injuries. *NeuroRehabilitation, 17*, 345–355.

Hippocrates. (n.d.). *On injuries of the head*. 400 BCE, parts 13 and 19.

Hirschfeld, R. M. (1999). Care of the sexually active depressed patient. *Journal of Clinical Psychiatry, 60*(Suppl 17), 32–35.

Ho, C. H., Hsieh, K. Y., Liang, F. W., Li, C. J., Wang, J. J., Chio, C. C., … Kuo, J. R. (2014). Pre-existing hyperlipidaemia increased the risk of new-onset anxiety disorders after traumatic brain injury: A 14-year population-based study. *British Medical Journal Open, 4*, e005269.

Hofer, H., Frigerio, S., Frischknecht, E., Gassmann, D., Gutbrod, K., & Muri, R. M. (2013). Diagnosis and treatment of an obsessive-compulsive disorder following traumatic brain injury: A single case and review of the literature. *Neurocase, 19*(4), 390–400.

Hoge, E. A., Brandstetter, K., Moshier, S., Pollack, M. H., Wong, K. K., & Simon, N. M. (2009). Broad spectrum of cytokine abnormalities in panic disorder and posttraumatic stress disorder. *Depression and Anxiety, 26*, 447–455.

Holland, D., Witty, T., Lawler, J., & Lanzisera, D. (1999). Biofeedback-assisted relaxation training with brain-injured patients in acute stages of recovery. *Brain Injury, 13*, 53–57.

Hollander, E., Braun, A., & Simeon, D. (2008). Should OCD leave the anxiety disorders in DSM-V? The case for obsessive compulsive-related disorders. *Depression and Anxiety, 25*(4), 317–329.

Holsinger, T., Steffens, D. C., Phillips, C., Helms, M. J., Havlik, R. J., Breitner, J. C. S., … Plassman, B. L. (2002). Head injury in early adulthood and the lifetime risk of depression. *Archives of General Psychiatry, 59*, 17–22.

Holzer, J. C. (1998). Buspirone and brain injury. *Journal of Neuropsychiatry & Clinical Neurosciences, 10*, 113.

Hoofien, D., Gilboa, A., Vakil, E., & Donovick, P. J. (2001). Traumatic brain injury (TBI) 10-20 years later: A comprehensive outcome study of psychiatric symptomatology, cognitive abilities and psychosocial functioning. *Brain Injury, 15*(3), 189–209.

Horinkova, J., Bartecek, R., & Fedorova, S. (2017). Electroconvulsive therapy in the treatment of patient with depressive disorder after traumatic brain injury, intracranial bleeding, and poly-trauma. *The Journal of ECT, 33*(3), e22–e23.

Hornstein, A., Lennihan, L., Seliger, G., Lichtman, S., & Schroeder, K. (1996). Amphetamine in recovery from brain injury. *Brain Injury, 10*, 145–148.

Horsfield, S. A., Rosse, R. B., Tomasino, V., Schwartz, B. L., Mastropaolo, J., & Deutsch, S. I. (2002). Fluoxetine's effects on cognitive performance in patients with traumatic brain injury. *International Journal of Psychiatry in Medicine, 32*, 337–344.

Hoskins, M., Pearce, J., Bethell, A., Dankova, L., Barbui, C., Tol, W. A., … Bisson, J. I. (2015). Pharmacotherapy for post-traumatic stress disorder: Systematic review and meta-analysis. *British Journal of Psychiatry, 206*(2), 93–100.

Huang, C. H., Lin, C. W., Lee, Y. C., Huang, C. Y., Huang, R. Y., Tai, Y. C., … Wang, H. K. (2018). Is traumatic brain injury a risk factor for neurodegeneration? A meta-analysis of population-based studies. *BMC Neurology, 18*(1), 184.

Huang, M. F., Su, C. H., Tu, H. P., Liu, T. L., Lin, C. L., Chen, C. S., & Yeh, Y. C. (2018). Association between bipolar disorder and subsequent traumatic brain injury in patients who received inpatient treatment. *Psychiatry Research, 261*, 517–521.

Hughes, C. G., Patel, M. B., & Pandharipande, P. P. (2012). Pathophysiology of acute brain dysfunction: What's the cause of all this confusion? *Current Opinion of Critical Care, 18*, 518–526.

Iliceto, A., Seiler, R. L., & Sarkar, K. (2018). Repetitive transcranial magnetic stimulation for treatment of depression in a patient with severe traumatic brain injury. *The Ochsner Journal, 18*, 264–267.

Isaacs, K. H., & Geracioti, T. D. (2015). Post-TBI central hypogonadism and PTSD. *The American Journal of Psychiatry, 172*(11), 1160.

Jang, S. H., & Kwon, H. G. (2017). Apathy due to injury of the prefrontocaudate tract following mild traumatic brain injury. *American Journal of Physical Medicine & Rehabilitation, 96*(7), e130–e133.

Janicak, P. G., Davis, J. M., Preskorn, S. H., & Ayd, F. J. (1993). *Principles and practice of psychopharmacotherapy*. Baltimore, MD: Williams and Wilkins.

Jellinger, K. A. (2004). Head injury and dementia. *Current Opinion in Neurology, 17*, 719–723.

Johansson, S. H., Jamora, C. W., Ruff, R. M., & Pack, N. M. (2008). A biopsychosocial perspective of aggression in the context of traumatic brain injury. *Brain Injury, 22*, 999–1006.

Johns, M. W. (1991). A new method for measuring daytime sleepiness: The Epworth sleepiness scale. *Sleep, 14*(6), 540–545.

Johnson, J. C. S., & Ward, D. A. (2017). Electroconvulsive therapy for psychosis in a patient with a traumatic brain injury. *The Australian and New Zealand Journal of Psychiatry, 51*(9), 1210.

Jorge, R. E., Acion, L., Burin, D., & Robinson, R. G. (2016). Sertraline for preventing mood disorders following traumatic brain injury. A randomized clinical trial. *Journal of the American Medical Association Psychiatry, 73*(10), 1041–1047.

Jorge, R. E., & Robinson, R. G. (2003). Mood disorders following traumatic brain injury. *International Review of Psychiatry, 15*, 317–327.

Jorge, R. E., Robinson, R. G., Moser, D., Tateno, A., Crespo-Facorro, B., & Arndt, S. (2004). Major depression following traumatic brain injury. *Archives of General Psychiatry, 61*, 42–50.

Jorge, R. E., Robinson, R. G., Starkstein, S. E., Arndt, S. V., Forrester, A. W., & Geisler, F. H. (1993). Secondary mania following traumatic brain injury. *American Journal of Psychiatry, 150*, 916–921.

Jorge, R. E., Starkstein, S. E., Arndt, S., Moser, D., Crespo-Facorro, B., & Robinson, R. G. (2005). Alcohol misuse and mood disorders following traumatic brain injury. *Archives of General Psychiatry, 62*, 742–749.

Jorge, R.E., Acion, L., Starkstein, S.E., & Magnotta, V. (2007). Hippocampal volume and mood disorders after traumatic brain injury. *Biological Psychiatry*, 62:332–338.

Juengst, S. B., Kumar, R. G., Arenth, P. M., & Wagner, A. K. (2014). Exploratory associations with tumor necrosis factor-alpha, disinhibition and suicidal endorsement after traumatic brain injury. *Brain, Behavior, and Immunity, 41*, 134–143.

Julien, J., Joubert, S., Ferland, M. C., Frenette, L. C., Boudreau-Duhaime, M. M., Malo-Veronneau, L., & De Guise, E. (2017). Association of traumatic brain injury and Alzheimer disease onset: A systematic review. *Annals of Physical and Rehabilitation Medicine, 60*, 347–356.

Kadyan, V., Colachis, S. C., Depalma, M. J., Sanderson, J. D., & Mysiw, W. J. (2003). Early recognition of neuroleptic malignant syndrome during traumatic brain rehabilitation. *Brain Injury, 17*, 631–637.

Kadyan, V., Mysiw, W. J., Bogner, J. A., Corrigan, J. D., Fugate, L. P., & Clinchot, D. M. (2004). Gender differences in agitation after traumatic brain injury. *American Journal of Physical Medicine & Rehabilitation, 83*, 747–752.

Kaiser, P. R., Valko, P. O., Werth, E., Thomann, J., Meier, J., Stocker, R., … Baumann, C. R. (2010). Modafinil ameliorates excessive daytime sleepiness after traumatic brain injury. *Neurology, 75*(20), 1780–1785.

Kalra, I. D., & Watanabe, T. K. (2017). Mood stabilizers for traumatic brain injury-related agitation. *The Journal of Head Trauma Rehabilitation, 32*(6), E61–E64.

Kant, R., Bogyi, A. M., Carosella, N. W., Fishman, E., Kane, V., & Coffey, C. E. (1995). ECT as a therapeutic option in severe brain injury. *Convulsive Therapy, 11*, 45–50.

Kant, R., Duffy, J. D., & Pivovarnik, A. (1998). Prevalence of apathy following head injury. *Brain Injury, 12*, 87–92.

Kant, R., Smith-Seemiller, L., & Zeiler, D. (1998). Treatment of aggression and irritability after head injury. *Brain Injury, 12*, 661–666.

Katz, S., & Aloni, R. (1999). Sexual dysfunction of persons after traumatic brain injury: Perceptions of professionals. *International Journal of Rehabilitation Research, 22*, 45–53.

Kaye, N. S. (2003). An open-label trial of donepezil (Aricept) in the treatment of persons with mild traumatic brain injury. *Journal of Neuropsychiatry & Clinical Neurosciences, 15*, 383–384.

Kelly, G., Todd, J., Simpson, G., Kremer, P., & Martin, C. (2006). The Overt Behaviour Scale (OBS): A tool for measuring challenging behaviours following ABI in community settings. *Brain Injury, 20*(3), 307–319.

Kemp, S., Biswas, R., Neumann, V., & Coughlan, A. (2004). The value of melatonin for sleep disorders occurring post-head injury: A pilot RCT. *Brain Injury, 18*(9), 911–919.

Kennedy, R., Burnett, D. M., & Greenwald, B. D. (2001). Use of antiepileptics in traumatic brain injury: A review for psychiatrists. *Annals of Clinical Psychiatry, 13*, 163–171.

Khouzam, H. R., & Donnelly, N. J. (1998). Remission of traumatic brain injury-induced compulsions during venlafaxine treatment. *General Hospital Psychiatry, 20*, 62–63.

Kim, E., & Bijlani, M. (2006). A pilot study of quetiapine treatment of aggression due to traumatic brain injury. *Journal of Neuropsychiatry and Clinical Neurosciences, 18*(4), 547–549.

Kim, E., & Humaran, T. J. (2002). Divalproex in the management of neuropsychiatric complications of remote acquired brain injury. *Journal of Neuropsychiatry & Clinical Neurosciences, 14*, 202–205.

Kim, E. (2008). Does traumatic brain injury predispose individuals to develop schizophrenia? Current Opinion in Psychiatry, 21, 286–289.

Kim, Y. W., Shin, J. C., & An, Y. (2010). Changes in cerebral glucose metabolism in patients with posttraumatic cognitive impairment after memantine therapy: A preliminary study. *Annals of Nuclear Medicine, 24*, 363–369.

Knutson, K. M., Dal Monte, O., Raymont, V., Wassermann, E. M., Krueger, F., & Grafman, J. (2014). Neural correlates of apathy revealed by lesion mapping in participants with traumatic brain injuries. *Human Brain Mapping, 35*, 943–953.

Kontos, A. P., Van Cott, A. C., Roberts, J., Pan, J. W., Kelly, M. B., McAllister-Deitrick, J., & Hetherington, H. P. (2017). Clinical and magnetic resonance spectroscopic imaging findings in veterans with blast mild traumatic brain injury and post-traumatic stress disorder. *Military Medicine, 182*(3/4), 99–104.

Koopowitz, L. F., & Berk, M. (1997). Response of obsessive compulsive disorder to carbamazepine in two patients with comorbid epilepsy. *Annals of Clinical Psychiatry, 9*, 171–173.

Koponen, S., Taiminen, T., Hiekkanen, H., & Tenovuo, O. (2011). Axis I and II psychiatric disorders in patients with traumatic brain injury: A 12-month follow-up study. *Brain Injury, 25*(11), 1029–1034.

Koponen, S., Taiminen, T., Kairisto, V., Portin, R., Isoniemi, H., Hinkka, S., & Tenovuo, O. (2004). APOE-e4 predicts dementia but not other psychiatric disorders after traumatic brain injury. *Neurology, 63*, 749–750.

Koponen, S., Taiminen, T., Portin, R., Himanen, L., Isoniemi, H., Heinonen, H., … Tenovuo, O. (2002). Axis I and II psychiatric disorders after traumatic brain injury: A 30 year follow-up study. *American Journal of Psychiatry, 159*, 1315–1321.

Kraus, M. F., & Maki, P. M. (1997). Effect of amantadine hydrochloride on symptoms of frontal lobe dysfunction in brain injury: Case studies and review. *Journal of Neuropsychiatry & Clinical Neurosciences, 9*, 222–230.

Krauthammer, C., & Klerman, G. L. (1978). Secondary mania: Manic syndromes associated with antecedent physical illnesses or drugs. *Archives of General Psychiatry, 35*, 1333–1339.

Kreitzer, N., Ancona, R., McCullumsmith, C., Kurowski, B. G., Foreman, B., Ngwenya, L. B., & Adeoye, O. (2019). The effect of antidepressants on depression after traumatic brain injury: A meta-analysis. *The Journal of Head Trauma Rehabilitation, 34*, E47–E54.

Kreutzer, J., & Zasler, N. (1989). Psychosexual consequences of traumatic brain injury: Methodology and preliminary findings. *Brain Injury, 3*, 177–186.

Krueger, R. B., & Kaplan, M. S. (2002). Behavioral and psychopharmacological treatment of the paraphilic and hypersexual disorders. *Journal of Psychiatric Practice, 8*, 21–32.

Labbate, L. A., & Warden, D. L. (2000). Common psychiatric syndromes and pharmacologic treatments of traumatic brain injury. *Current Psychiatry Reports, 2*, 268–273.

Lal, S., Merbtiz, C. P., & Grip, J. C. (1988). Modification of function in head-injured patients with sinemet. *Brain Injury, 2*, 225–233.

Lee, H., Kim, S. W., Kim, J. M., Shin, I. S., Yang, S. J., & Yoon, J. S. (2005). Comparing effects of methylphenidate, sertraline and placebo on neuropsychiatric sequelae in patients with traumatic brain injury. *Human Psychopharmacology: Clinical and Experimental, 20*, 97–104.

Leon-Carrion, J., Dominguez-Roldan, J. M., Murill-Cabezas, F., Dominguez-Morales, M. R., & Munoz-Sanchez, M. A. (2000). The role of citicholine in neuropsychological training after traumatic brain injury. *NeuroRehabilitation, 14*, 33–40.

Lequerica, A., Jasey, N., Portelli Tremont, J. N., & Chiaravalloti, N. D. (2015). Pilot study on the effect of ramelteon on sleep disturbance after traumatic brain injury: Preliminary evidence from a clinical trial. *Archives of Physical Medicine and Rehabilitation, 96*(10), 1802–1809.

Levin, H. S. (1990). Memory deficit after closed head injury. *Journal of Clinical and Experimental Neuropsychiatry, 12*, 129–153.

Levin, H. S. (1991). Treatment of postconcussional symptoms with CDP-choline. *Journal of Neurological Sciences, 103*, S39–S42.

Levin, H. S., & Grossman, R. G. (1978). Behavioral sequelae of closed head injury: A quantitative study. *Archives in Neurology, 35*, 720–727.

Levin, H. S., High, W., Goethe, K. E., & Sisson, R. A. (1987). The Neurobehavioral Rating Scale: Assessment of the behavioral sequelae of head injury by the clinician. *Journal of Neurology, Neurosurgery, and Psychiatry, 50*, 183–193.

Levin, H. S., Peters, B. H., Kalisky, Z., High, W. M., von Laufen, A., Eisenberg, H. M., … Gary, H. E. (1986). Effects of oral physostigmine and lecithin on memory and attention in the closed head injured patient. *Central Nervous System Trauma, 3*, 333–342.

Lindquist, L. K., Love, H. C., & Elbogen, E. B. (2017). Traumatic brain injury in Iraq and Afghanistan veterans: New results from a national random sample study. *The Journal of Neuropsychiatry and Clinical Neurosciences, 29*(3), 254–259.

Liu, H., Yang, Y., Xia, Y., Zhu, W., Leak, R. K., Wei, Z., … Hu, X. (2017). Aging of cerebral white matter. *Ageing Research Reviews, 34*, 64–76.

LoBue, C., Cullum, C. M., Didehbani, N., Yeatman, K., Jones, B., Kraut, M. A., & Hart, J. (2018). Neurodegenerative dementias after traumatic brain injury. *Journal of Neuropsychiatry and Clinical Neurosciences, 30*(1), 7–13.

Lombardi, G., Nelli, F., Celso, M., Mencarini, M., & Del Popolo, G. (2012). Treating erectile dysfunction and central neurological diseases with oral phosphodiesterase type 5 inhibitors. Review of the literature. *The Journal of Sexual Medicine, 9*, 970–985.

Lopez, K. C., Leary, J. B., Pham, D. L., Chou, Y. Y., Dsurney, J., & Chan, L. (2017). Brain volume, connectivity, and neuropsychological performance in mild traumatic brain injury: The impact of post-traumatic stress disorder symptoms. *Journal of Neurotrauma, 34*, 16–22.

Luaute, J., Plantier, D., Wiart, L., Tell, L., & the SOFMER group. (2016). Care management of the agitation or aggressiveness crisis in patients with TBI. Systematic review of the literature and practice recommendations. *Annuals of Physical and Rehabilitation Medicine, 59*, 58–67.

Luria, A. R. (1972). *The man with a shattered world. The history of a brain wound* (p. 38). New York, NY: Basic Books.

Luukinen, H., Viramo, P., Herala, M., Kervinen, K., Kesaniemi, Y. A., Savola, O., … Hillborn, M. (2005). Fall-related brain injuries and the risk of dementia in elderly people: A population based study. *European Journal of Neurology, 12*, 86–92.

Mackelprang, J. L., Bombardier, C. H., Fann, J. R., Temkin, N. R., Barber, J. K., & Dikmen, S. S. (2014). Rates and predictors of suicidal ideation during the first year after traumatic brain injury. *American Journal of Public Health, 104*(7), e100–e107.

Madsen, T., Erlangsen, A., Orlovska, S., Mofaddy, R., Nordentoft, M., & Benros, M. E. (2018). Association between traumatic brain injury and risk of suicide. *The Journal of the American Medical Association, 320*(6), 580–588.

Mahar, I., Alosco, M. L., & McKee, A. C. (2017). Psychiatric phenotypes in chronic traumatic encephalopathy. *Neuroscience and Biobehavioral Reviews, 83*, 622–630.

Maia, T. V., Cooney, R. E., & Peterson, B. S. (2008). The neural bases of obsessive-compulsive disorder in children and adults. *Development and Psychopathology, 20*, 1251–1283.

Malaspina, D., Goetz, R. R., Friedman, J. H., Kaufmann, C., Faraone, S. V., Tsuang, M., … Blehar, M. C. (2001). Traumatic brain injury and schizophrenia in members of schizophrenia and bipolar disorder pedigrees. *American Journal of Psychiatry, 158*, 440–446.

Mallya, S., Sutherland, J., Pongracic, S., Mainland, B., & Ornstein, T. J. (2015). The manifestation of anxiety disorders after traumatic brain injury: A review. *Journal of Neurotrauma, 32*, 411–421.

Mangels, J. A., Craik, F. I. M., Levine, B., Schwartz, M. L., & Sluss, D. T. (2002). Effects of divided attention on episodic memory in chronic traumatic brain injury: A function of severity and strategy. *Neuropsychologia, 40*, 2369–2385.

Marcotte, D. (1998). Use of topiramate, a new anti-epileptic as a mood stabilizer. *Journal of Affective Disorders, 50*, 245–251.

Marin, R. S., Biedrzycki, R. C., & Firinciogullaari, S. (1991). Reliability and validity of the apathy evaluation scale. *Psychiatry Research, 38*, 143–162.

Marin, R. S., & Wilkosz, P. A. (2005). Disorders of diminished motivation. *The Journal of Head Trauma Rehabilitation, 20*, 377–388.

Masanic, C. A., Bayley, M. T., van Reekum, R., & Simard, M. (2001). Open-label study of donepezil in traumatic brain injury. *Archives of Physical Medicine and Rehabilitation, 82*, 896–901.

Masterman, D. L., & Cummings, J. L. (1997). Frontal-subcortical circuits: The anatomic basis of executive, social and motivated behaviors. *Journal of Psychopharmacology, 11*(2), 107–114.

Mateer, C. A., Kerns, K. A., & Eso, K. L. (1999). Management of attention and memory disorders following traumatic brain injury. *Journal of Learning Disabilities, 29*, 618–632.

Mathias, J. L., & Alvaro, P. K. (2012). Prevalence of sleep disturbances, disorders, and problems following traumatic brain injury: A meta-analysis. *Sleep Medicine, 13*(7), 898–905.

Mayou, R. A., Black, J., & Bryant, B. (2000). Unconsciousness, amnesia and psychiatric symptoms following road traffic accident injury. *British Journal of Psychiatry, 177*, 540–545.

McAllister, T. W. (2011). Neurobiological consequences of traumatic brain injury. *Dialogues in Clinical Neuroscience, 13*(3), 287–300.

McDonald, B. C., Flashman, L. A., Arciniegas, D. B., Ferguson, R. J., Xing, L., Harezlak, J., … McAllister, T. W. (2017). Methylphenidate and memory and attention adaptation training for persistent cognitive symptoms after traumatic brain injury: A randomized, placebo-controlled trial. *Neuropsychopharmacology, 42*, 1766–1775.

McKinlay, W. W., Brooks, D. N., Bond, M. R., Martinage, D. P., & Marshall, M. M. (1981). The short-term outcome of severe blunt head injury as reported by the relatives of the injured person. *Journal of Neurology, Neurosurgery, and Psychiatry, 44*, 527–533.

McNeil, J. E., & Greenwood, R. (1996). Can PTSD occur with amnesia for the precipitating event? *Cognitive Neuropsychiatry, 1*, 239–246.

Mehta, K. M., Ott, A., Kalmijn, S., Slooter, A. J. C., van Duijn, C. M., Hofman, A., & Breteler, M. M. B. (1999). Head trauma and risk of dementia and Alzheimer's disease—The Rotterdam study. *Neurology, 53*, 1959–1962.

Merz, Z. C., Roskos, P. T., Gfeller, J. D., & Bucholz, R. D. (2017). Impact of psychiatric symptomatology on neuropsychological assessment performance in persons with TBI: A comparison of OEF/OIF veteran and civilian samples. *Brain Injury, 31*(11), 1422–1428.

Mesulam, M. M. (2000). *Principles of behavioral and cognitive neurology* (2nd ed., pp. 121–256). New York, NY: Oxford University Press. Chapters 2–3.

Meyer, A. (1904). The anatomical facts and clinical varieties of traumatic insanity. *American Journal of Insanity, 60*, 373–441.

Meythaler, J. M., Depalma, L., Devivo, M. J., Guin-Renfroe, S., & Novack, T. A. (2001). Sertraline to improve arousal and alertness in severe traumatic brain injury secondary to motor vehicle crashes. *Brain Injury, 15*(4):321–331.

Meyer, E. G., & Writer, B. W. (2015). In E. C. Ritchie (Ed.), *Posttraumatic stress disorder and related diseases* (pp. 245–255). Cham: Springer International Publishing. Chapter 18.

Michals, M. L., Crismon, M. L., Roberts, S., & Childs, A. (1993). Clozapine response and adverse effects in nine brain-injured patients. *Journal of Clinical Psychopharmacology, 13*, 198–203.

Moldover, J. E., Goldberg, K. B., & Prout, M. F. (2004). Depression after traumatic brain injury: A review of evidence for clinical heterogeneity. *Neuropsychology Review, 14*, 143–154.

Molloy, C., Conroy, R. M., Cotter, D. R., & Cannon, M. (2011). Is traumatic brain injury a risk factor for schizophrenia? A meta-analysis of case-controlled population-based studies. *Schizophrenia Bulletin, 37*(6), 1104–1110.

Monji, A., Yoshida, I., Koga, H., Tashiro, K., & Tashiro, N. (1999). Brain injury-induced rapid-cycling affective disorder successfully treated with valproate. *Psychosomatics, 40*, 448–449.

Mooney, G. F., & Haas, L. J. (1993). Effect of methylphenidate on brain injury-related anger. *Archives of Physical Medicine and Rehabilitation, 74*, 153–160.

Moreira, T., Khemiri, L., & Runeson, L. (2011). Impulse control loss rapidly reversed by aripiprazole in a patient with concomitant bipolar disease type I and posttraumatic frontal lobe lesions. *British Medical Journal Case Reports*.

Moreno, J. A., & McKerral, M. (2017). Towards a taxonomy of sexuality following traumatic brain injury: A pilot exploratory study using cluster analysis. *NeuroRehabilitation, 41*, 281–291.

Moreno-Lopez, L., Sahakian, B. J., Manktelow, A., Menon, D. K., & Stamatakis, E. A. (2016). Depression following traumatic brain injury: A functional connectivity perspective. *Brain Injury, 30*(11), 1319–1328.

Morey, C. E., Cilo, M., Berry, J., & Cusick, C. (2003). The effect of Aricept in persons with persistent memory disorder following traumatic brain injury: A pilot study. *Brain Injury, 17*, 809–815.

Morris, T., Gomes Osman, J., Tormos Munoz, J. M., Costa Miserachs, D., & Pascual Leone, A. (2016). The role of physical exercise in cognitive recovery after traumatic brain injury: A systematic review. *Restorative Neurology and Neuroscience, 34*(6), 977–988.

Mosti, C., & Coccaro, E. F. (2018). Mild traumatic brain injury and aggression, impulsivity, and history of other- and self-directed aggression. *Journal of Neuropsychiatry and Clinical Neurosciences, 30*(3), 220–227.

Moutaouakil, F., El Otmani, H., Fadel, H., & Slassi, I. (2009). Severe apathy following head injury: Improvement with selegiline treatment. *Neurochirurgie, 55*, 551–554.

Muller, U., Murai, T., Bauer-Wittmund, T., & Von Cramon, D. Y. (1999). Paroxetine versus citalopram treatment of pathological crying after brain injury. *Brain Injury, 13*, 805–811.

Muller, U., & Von Cramon, Y. (1994). The therapeutic potential of bromocriptine in neuropsychological rehabilitation of patients with acquired brain damage. *Progress in Neuro-Psychopharmacology & Biological Psychiatry, 18*, 1103–1120.

Munakomi, S., Bhattarai, B., & Kumar, B. M. (2017). Role of bromocriptine in multi-spectral manifestations of traumatic brain injury. *Chinese Journal of Traumatology, 20*, 84–86.

Murai, T., & Fujimoto, S. (2003). Rapid cycling bipolar disorder after left temporal polar damage. *Brain Injury, 17*, 355–358.

Mustafa, B., Evrim, O., & Sari, A. (2005). Secondary mania following traumatic brain injury. *Journal of Neuropsychiatry & Clinical Neurosciences, 17*, 122–124.

Mysiw, W. J., Jackson, R. D., & Corrigan, J. D. (1988). Amitryptyline for post-traumatic agitation. *American Journal of Physical Medicine & Rehabilitation, 67*, 29–33.

Mysiw, W. J., & Sandel, M. E. (1997). The agitated brain injured patient: Part 2: Pathophysiology and treatment. *Archives of Physical Medicine and Rehabilitation, 78*, 213–220.

Nathan, D. E., Frost Bellgowan, J. A., French, L. M., Wolf, J., Oakes, T. R., Mielke, J., … Riedy, G. (2017). Assessing the impact of post-traumatic stress symptoms on the resting-state default

mode network in a military chronic mild traumatic brain injury sample. *Brain Connectivity, 7*(4), 236–249.
Nemetz, P. N., Leibson, C., Naessens, J. M., Beard, M., Kokmen, E., Annegers, J. F., & Kurland, L. T. (1999). Traumatic brain injury and time to onset of Alzheimer's disease: A population-based study. *American Journal of Epidemiology, 149*, 32–40.
Newburn, G. (1998). Psychiatric disorders associated with traumatic brain injury: Optimal treatment. *CNS Drugs, 6*, 441–456.
Newburn, G., & Newburn, D. (2005). Selegiline in the management of apathy following traumatic brain injury. *Brain Injury, 19*, 149–154.
Nickels, J. L., Schneider, W. N., Dombovy, M. L., & Wong, T. M. (1994). Clinical use of amantadine in brain injury rehabilitation. *Brain Injury, 8*, 709–718.
Niemann, H., Ruff, R. M., & Kramer, J. H. (1996). An attempt towards differentiating attentional deficits in traumatic brain injury. *Neuropsychological Review, 1*, 11–46.
Noe, E., Ferri, J., Trenor, C., & Chirivella, J. (2007). Efficacy of ziprasidone in controlling agitation during post-traumatic amnesia. *Behavioral Neurology, 18*(1), 7–11.
O'Donnell, M. D., Alkemade, N., Creamer, M. C., McFarlane, A. C., Silove, D., Bryant, R. A., & Forbes, D. (2016). The long-term psychiatric sequelae of severe injury: A 6-year follow-up study. *The Journal of Clinical Psychiatry, 77*(4), e473–e479.
O'Donnell, M. L., Creamer, M., Bryant, R. A., Schnyder, U., & Shalev, A. (2003). Posttraumatic disorders following injury: An empirical and methodological review. *Clinical Psychology Review, 23*, 587–603.
Oddy, M. (2001). Sexual relationships following brain injury. *Sexual and Relationship Therapy, 16*, 247–259.
Oddy, M., Coughlan, T., Tyerman, A., & Jenkins, D. (1985). Social adjustment after closed head injury: A further follow-up seven years after injury. *Journal of Neurology, Neurosurgery, and Psychiatry, 48*, 564–568.
Oquendo, M. A., Friedman, J. H., Grunebaum, M. F., Burke, A., Silver, J. M., & Mann, J. J. (2004). Suicidal behavior and mild traumatic brain injury in major depression. *The Journal of Nervous and Mental Disease, 192*, 430–434.
Osborn, A. J., Mathias, J. L., & Fairweather-Schmidt, A. K. (2016). Prevalence of anxiety following adult traumatic brain injury: A meta-analysis comparing measures, samples and postinjury intervals. *Neuropsychology, 30*(2), 247–261.
Osborn, A. J., Mathias, J. L., Fairweather-Schmidt, A. K., & Anstey, K. J. (2018). Traumatic brain injury and depression in a community-based sample: A cohort study across the adult life span. *The Journal of Head Trauma Rehabilitation, 33*(1), 62–72.
Oster, T. J., Anderson, C. A., Filley, C. M., Wortzel, H. S., & Aciniegas, D. B. (2007). Quetiapine for mania due to traumatic brain injury. *CNS Spectrums, 12*(10), 764–769.
Ouellet, M. C., Beaulieu-Bonneau, S., & Morin, C. M. (2015). Sleep-wake disturbances after traumatic brain injury. *Lancet Neurology, 14*(7), 746–757.
Ouellet, M. C., Beaulieu-Bonneau, S., Sirois, M. J., Savard, J., Turgeon, A. F., Moore, L., … Laviolette, V. (2018). Depression in the first year after traumatic brain injury. *Journal of Neurotrauma, 35*, 1620–1629.
Ouellet, M. C., Savard, J., & Morin, C. M. (2004). Insomnia following traumatic brain injury: A review. *Neurorehabilitation and Neural Repair, 18*, 187–198.
Pachet, A., Friesen, S., Wenkelaar, D., & Gray, S. (2003). Beneficial behavioral effects of lamotrigine in traumatic brain injury. *Brain Injury, 17*, 715–722.
Paraschakis, A., & Katsanos, A. H. (2017). Antidepressants for depression associated with traumatic brain injury: A meta-analytical study of randomized controlled trials. *East Asian Archives of Psychiatry, 27*, 142–149.
Pardini, M., Krueger, F., Hodgkinson, C. A., Raymont, V., Strenziok, M., Amore, M., … Grafman, J. H. (2014). Aggression, DRD1 polymorphism, and lesion location in penetrating traumatic brain injury. *CNS Spectrums, 19*, 382–390.
Park, S., Williams, R. A., & Lee, D. (2016). Effect of preferred music on agitation after traumatic brain injury. *Western Journal of Nursing Research, 38*(4), 394–410.

Parvizi, J., Anderson, S. W., Martin, C. O., Damasio, H., & Damasio, A. R. (2001). Pathological laughter and crying: A link to the cerebellum. *Brain, 124*, 1708–1719.

Perino, C., Rago, R., Cicolin, A., Torta, R., & Monaco, F. (2001). Mood and behavioral disorders following traumatic brain injury: Clinical evaluation and pharmacological management. *Brain Injury, 15*, 139–148.

Perna, R. (2004). Benzodiazepines and antipsychotics: Cognitive side-effects. *The Journal of Head Trauma Rehabilitation, 19*, 516–518.

Perry, D. C., Sturm, V. E., Peterson, M. J., Pleper, C. F., Bullock, T., Boeve, B. F., … Welsh-Bohmer, K. A. (2016). Association of traumatic brain injury with subsequent neurological and psychiatric disease: A meta-analysis. *Journal of Neurosurgery, 124*, 511–526.

Perry, S. A., Coetzer, R., & Saville, C. W. N. (2018). The effectiveness of physical exercise as an intervention to reduce depressive symptoms following traumatic brain injury: A meta-analysis and systematic review. *Neuropsychological Rehabilitation, 13*, 1–15.

Plassman, B. L., Havlik, R. J., Steffens, D. C., Helms, M. J., Newman, T. N., Drosdick, D., … Breitner, J. C. S. (2000). Documented head injury in early adulthood and risk of Alzheimer's disease and other dementias. *Neurology, 55*, 1158–1166.

Plenger, P. M., Dixon, C. E., Castillo, R. M., Frankowski, R. F., Yablon, S. A., & Levin, H. S. (1996). Subacute methylphenidate treatment for moderate to moderately severe traumatic brain injury: A preliminary double-blind placebo-controlled study. *Archives of Physical Medicine and Rehabilitation, 77*, 536–540.

Ponsford, J., Alway, Y., & Gould, K. R. (2018). Epidemiology and natural history of psychiatric disorders after TBI. *Journal of Neuropsychiatry & Clinical Neurosciences, 30*(4), 262–270.

Ponsford, J. L., Downing, M. G., & Stolwyk, R. (2013). Factors associated with sexuality following traumatic brain injury. *The Journal of Head Trauma Rehabilitation, 28*(3), 195–201.

Pope, H. G., McElroy, S. L., Satlin, A., Hudson, J. I., Keck, P. E., & Kalish, R. (1988). Head injury, bipolar disorder and response to valproate. *Comprehensive Psychiatry, 29*, 34–38.

Popovic, V., Aimaretti, G., Casanueva, F. F., & Ghigo, E. (2005). Hypopituitarism following traumatic brain injury. *Growth Hormone & IGF Research, 15*, 177–184.

Powell, J. H., Al-Adawi, S., Morgan, J., & Greenwood, R. J. (1996). Motivational deficits after brain injury: Effects of bromocriptine in 11 patients. *Journal of Neurology, Neurosurgery, and Psychiatry, 60*, 416–421.

Prigatano, G. P. (1991). Disordered mind, wounded soul. The emerging role of psychotherapy in rehabilitation after brain injury. *The Journal of Head Trauma Rehabilitation, 64*, 1–10.

Rabner, J., Gottlieb, S., Lazdowsky, L., & LeBel, A. (2016). Case-series: Psychosis following traumatic brain injury and cannabis use in late adolescence. *The American Journal on Addictions, 25*, 91–93.

Randolph, C., Tierney, M. C., Mohr, E., & Chase, T. N. (1998). The Repeatable Battery for the Assessment of Neuropsychological Status (RBANS): Preliminary clinical validity. *Journal of Clinical and Experimental Neuropsychology, 20*, 310–319.

Rao, N., Jellinek, H. M., & Woolston, D. C. (1985). Agitation in closed head injury: Haloperidol effects on rehabilitation outcome. *Archives of Physical Medicine and Rehabilitation, 66*, 30–34.

Rao, V., & Lyketsos, C. G. (2002). Psychiatric aspects of traumatic brain injury. *Psychiatric Clinics of North America, 25*, 43–69.

Rao, V., McCann, U., Han, D., Bergey, A., & Smith, M. T. (2014). Does acute TBI-related sleep disturbance predict subsequent neuropsychiatric disturbances? *Brain Injury, 28*(1), 20–26.

Rao, V., Mielke, M., Xu, X., Smith, G. S., McCann, U. D., Bergey, A., … Mori, S. (2012). Diffusion tensor imaging atlas-based analyses in major depression after mild traumatic brain injury. *The Journal of Neuropsychiatry and Clinical Neurosciences, 24*, 309–315.

Rapoport, M. J., Chan, F., Lanctot, K., Herrmann, N., McCullagh, S., & Feinstein, A. (2008). An open-label study of citalopram for major depression following traumatic brain injury. *Journal of Psychopharmacology, 22*(8), 860–864.

Rapoport, M. J., McCullagh, S., Shamni, P., & Feinstein, A. (2005). Cognitive impairment associated with major depression following mild and moderate traumatic brain injury. *Journal of Neuropsychiatry & Clinical Neurosciences, 17*, 61–65.

Rapoport, M. J., McCullagh, S., Streiner, D., & Feinstein, A. (2003). The clinical significance of major depression following mild traumatic brain injury. *Psychosomatics, 44*, 31–37.

Rauch, S. L., Shin, L. M., & Phelps, E. A. (2006). Neurocircuitry models of posttraumatic stress disorder and extinction: Human neuroimaging research—Past, present, and future. *Biological Psychiatry, 60*(4), 376–382.

Reinhard, D. L., Whyte, J., & Sandel, M. E. (1996). Improved arousal and initiation following tricyclic antidepressant use in severe brain injury. *Archives of Physical Medicine and Rehabilitation, 77*, 80–83.

Reti, I. M., Schwarz, N., Bower, A., Tibbs, M., & Rao, V. (2015). Transcranial magnetic stimulation: A potential new treatment for depression associated with traumatic brain injury. *Brain Injury, 29*(7-8), 789–797.

Roane, D. M., Feinberg, T. E., Meckler, L., Miner, C. R., Scicutella, A., & Rosenthal, R. N. (2000). Treatment of dementia-associated agitation with gabapentin. *Journal of Neuropsychiatry & Clinical Neurosciences, 12*(1), 40–43.

Rocca, G., Caputo, F., Frigiolini, F. M. E., Verde, A., & Ventura, F. (2018). Delirium resulting from traumatic brain injury as an acute risk factor for suicide: A case report and review of the literature. *The Journal of Neuropsychiatry & Clinical Neurosciences in Advance, 31*, 86–88.

Rosati, D. L. (2002). Early polyneuropharmacologic intervention in brain injury agitation. *American Journal of Physical Medicine & Rehabilitation, 81*, 90–93.

Rosso, I. M., Crowley, D. J., Silveri, M. M., Rauch, S. L., & Jensen, J. E. (2017). Hippocampus glutamate and n-acetyl aspartate markers of excitotoxic neuronal compromise in posttraumatic stress disorder. *Neuropsychopharmacology, 42*(8), 1698–1705.

Rothwell, N. A., LaVigna, G. W., & Willis, T. J. (1999). A non-aversive rehabilitation approach for people with severe behavioral problems resulting from brain injury. *Brain Injury, 13*, 521–533.

Rowland, R. T., Mysiw, W. J., & Bogner, J. A. (1992). Trazodone for post-traumatic agitation [abstract]. *Archives of Physical Medicine and Rehabilitation, 73*, 963.

Roy, D., Koliatsos, V., Vaishnavi, S., Han, D., & Rao, V. (2018). Risk factors for new-onset depression after first-time traumatic brain injury. *Psychosomatics, 59*(1), 47–57.

Roy, D., Vaishnavi, S., Han, D., & Rao, V. (2017). Correlates and prevalence of aggression at six months and one year after first-time traumatic brain injury. *Journal of Neuropsychiatry and Clinical Neurosciences, 29*(4), 334–342.

Ruedrich, S. L., Chu, C. C., & Moore, S. L. (1983). ECT for major depression in a patient with acute brain trauma. *American Journal of Psychiatry, 140*, 928–929.

Ruff, R. L., Ruff, S. S., & Wang, X. F. (2009). Improving sleep: Initial headache treatment in OIF/OEF veterans with blast-induced mild traumatic brain injury. *Journal of Rehabilitation Research and Development, 46*(9), 1071–1084.

Ruff, R. M., & Hibbard, K. M. (2003). *Ruff neurobehavioral inventory: Professional manual*. Lutz, FL: Psychological Assessment Resources, Inc..

Rybach, R., & Rybach, L. (1995). Gabapentin for behavioral dyscontrol. *American Journal of Psychiatry, 152*, 1399–1401.

Rydon-Grange, M., & Coetzer, R. (2015). What do we know about obsessive-compulsive disorder following traumatic brain injury? *CNS Spectrums, 20*, 463–465.

Sabaz, M., Simpson, G. K., Walker, A. J., Rogers, J. M., Gillis, I., & Strettles, B. (2014). Prevalence, comorbidities, and correlates of challenging behavior among community-dwelling adults with severe traumatic brain injury: A multicenter study. *The Journal of Head Trauma Rehabilitation, 29*(2), E19–E30.

Sachdev, P., Smith, J. S., & Cathcart, S. (2001). Schizophrenia-like psychosis following traumatic brain injury: A chart-based descriptive and case–control study. *Psychological Medicine, 31*, 231–239.

Sadock, B. J., Sadock, V. A., & Ruiz, P. (Eds.). (2015). *Kaplan & Sadock's synopsis of psychiatry* (11th ed.). Philadelphia, PA: Wolters Kluwer.

Salmond, C. H., Chatfield, D. A., Manon, D. K., Pickard, J. D., & Sahakian, B. J. (2005). Cognitive sequelae of head injury: Involvement of basal forebrain and associated structures. *Brain, 128*, 189–200.

Salter, K. L., McClure, A., Foley, N. C., Sequeira, K., & Teasell, R. W. (2016). Pharmacotherapy for depression posttraumatic brain injury: A meta-analysis. *The Journal of Head Trauma Rehabilitation, 31*(4), E21–E32.

Sami, M., Piggott, K., Coysh, C., & Fialho, A. (2015). Psychosis, psychedelic substance misuse and head injury: A case report and 23 year follow-up. *Brain Injury, 29*(11), 1383–1386.

Sandel, M. E., & Mysiw, W. J. (1996). The agitated brain injured patient. Part I. Definitions, differential diagnosis and assessment. *Archives of Physical Medicine and Rehabilitation, 77*, 617–623.

Sandel, M. E., Williams, K. S., Dellapietra, L., & Derogatis, L. R. (1996). Sexual functioning following traumatic brain injury. *Brain Injury, 10*, 719–728.

Sander, A. M., Maestas, K. L., Pappadis, M. R., Hammond, F. M., Hanks, R. A., & the NIDILRR Traumatic Brain Injury Model Systems Module Project on Sexuality After TBI. (2016). Multicenter study of sexual functioning in spouses/partners of persons with traumatic brain injury. *Archives of Physical Medicine and Rehabilitation, 97*, 753–759.

Sander, A. M., Maestas, K. L., Pappadis, M. R., Sherer, M., Hammond, F. M., Hanks, R., & the NIDRR Traumatic Brain Injury Model Systems Module Project on Sexuality after TBI. (2012). Sexual functioning 1 year after traumatic brain injury: Findings from a prospective traumatic brain injury model systems collaborative study. *Archives of Physical Medicine and Rehabilitation, 93*, 1331–1337.

Saran, A. S. (1985). Depression after minor closed head injury: Role of dexamethasone suppression test and antidepressants. *Journal of Clinical Psychiatry, 46*, 335–338.

Satz, P. (1993). Brain reserve capacity on symptom onset after brain injury: A formulation and review of evidence for threshold theory. *Neuropsychology, 7*, 273–295.

Sayal, K., Ford, T., & Pipe, R. (2000). Case study: Bipolar disorder after head injury. *Journal of the American Academy of Child and Adolescent Psychiatry, 39*, 525–528.

Scheutzow, M. H., & Wiercisiewski, D. R. (1999). Panic disorder in a patient with traumatic brain injury: A case report and discussion. *Brain Injury, 13*, 705–714.

Schneier, F. R., Campeas, R., Carcamo, J., Glass, A., Lewis-Fernandez, R., Neria, Y., … Wall, M. M. (2015). Combined mirtazapine and SSRI treatment of PTSD: A placebo-controlled trial. *Depression and Anxiety, 32*(8), 570–579.

Scholten, A. C., Haagsma, J. A., Cnossen, M. C., Olff, M., van Beeck, E. F., & Polinder, S. (2016). Prevalence of and risk factors for anxiety and depressive disorders after traumatic brain injury: A systematic review. *Journal of Neurotrauma, 33*, 1969–1994.

Schreiber, S., Klag, E., Gross, Y., Segman, R. H., & Pick, C. G. (1998). Beneficial effect of risperidone on sleep disturbance and psychosis following traumatic brain injury. *International Clinical Psychopharmacology, 13*, 273–275.

Scicutella, A. (2001). Anxiety disorders in epilepsy. In A. B. Ettinger & A. M. Kanner (Eds.), *Psychiatric issues in epilepsy a practical guide to diagnosis and treatment* (pp. 95–109). Philadelphia, PA: Lippincott Williams and Wilkins.

Scofield, D. E., Proctor, S. P., Kardouni, J. R., Hill, O. T., & McKinnon, C. J. (2017). Risk factors for mild traumatic brain injury and subsequent post-traumatic stress disorder and mental health disorders among United States army soldiers. *Journal of Neurotrauma, 34*, 3249–3255.

Seel, R. T., Kreutzer, J. S., Rosenthal, M., Hammond, F. M., Corrigan, J. D., & Black, K. (2003). Depression after traumatic brain injury: A National Institute on Disability and Rehabilitation research model systems multicenter investigation. *Archives of Physical Medicine and Rehabilitation, 84*(2), 177–184.

Seel, R. T., Macciocchi, S., & Kreutzer, J. S. (2010). Clinical considerations for the diagnosis of major depression after moderate to severe TBI. *The Journal of Head Trauma Rehabilitation, 25*(2), 99–112.

Shaughnessy, R. (1995). Psychopharmacotherapy of neuropsychiatric disorders. *Psychiatric Annals, 25*, 634–640.

Sheehan, D. V., Ballenger, J., & Jacobsen, G. (1980). Treatment of endogenous anxiety with phobic, hysterical, and hypochondriacal symptoms. *Archives of General Psychiatry, 37*, 51–59.

Sherer, M., Yablon, S. A., & Nick, T. G. (2014). Psychotic symptoms as manifestations of the posttraumatic confusional state: Prevalence, risk factors, and association with outcome. *The Journal of Head Trauma Rehabilitation, 29*(2), E11–E18.

Silver, J. M., Koumaras, B., Meng, X., Potkin, S. G., Reyes, P. F., Harvey, P. D., … Arciniegas, D. B. (2009). Long-term effects of rivastigmine capsules in patients with traumatic brain injury. *Brain Injury, 23*(2), 123–132.

Silver, J. M., Kramer, R., Greenwald, S., & Weissman, M. (2001). The association between head injuries and psychiatric disorders: Findings from the New Haven NIMH epidemiologic catchment area study. *Brain Injury, 15*, 935–945.

Silver, J. M., Yudofsky, S. C., & Anderson, K. E. (2005). Aggressive disorders. In J. M. Silver, T. W. McAllister, & S. C. Yudofsky (Eds.), *Textbook of traumatic brain injury* (pp. 259–277). Washington, DC: American Psychiatric Press.

Simpson, G., Blaszczynski, A., & Hodgkinson, A. (1999). Sex offending as a psychosocial sequelae of traumatic brain injury. *The Journal of Head Trauma Rehabilitation, 14*(6), 567–580.

Simpson, G., & Long, E. (2004). An evaluation of sex education and information resources and their provision to adults with traumatic brain injury. *The Journal of Head Trauma Rehabilitation, 19*, 413–428.

Simpson, G., & Tate, R. (2002). Suicidality after traumatic brain injury: Demographic, injury and clinical correlates. *Psychological Medicine, 32*, 687–697.

Simpson, G. K., McCann, B., & Lowy, M. (2016). Treating male sexual dysfunction after traumatic brain injury: Two case reports. *NeuroRehabilitation, 38*, 281–289.

Simpson, G. K., Sabaz, M., & Daher, M. (2013). Prevalence, clinical features, and correlates of inappropriate sexual behavior after traumatic brain injury: A multicenter study. *The Journal of Head Trauma Rehabilitation, 28*(3), 202–210.

Simpson, G. K., Tate, R. L., Whiting, D. L., & Cotter, R. E. (2011). Suicide prevention after traumatic brain injury: A randomized controlled trial of a program for the psychological treatment of hopelessness. *The Journal of Head Trauma Rehabilitation, 26*(4), 290–300.

Sinclair, K. L., Ponsford, J. L., Taffe, J., Lockley, S. W., & Rajaratnam, S. M. (2014). Randomized controlled trial of light therapy for fatigue following traumatic brain injury. *Neurorehabilitation and Neural Repair, 28*(4), 303–313.

Singh, R., Mason, S., Lecky, F., & Dawson, J. (2018). Prevalence of depression after TBI in a prospective cohort: The SHEFBIT study. *Brain Injury, 32*(1), 84–90.

Singh, R., Venkateshwara, G., Nair, K. P. S., Khan, M., & Saad, R. (2014). Agitation after traumatic brain injury and predictors of outcome. *Brain Injury, 28*(3), 336–340.

Small, G. W., Rabins, P. V., Barry, P. P., Burkholtz, N. S., DeKosky, S. T., Ferris, S. H., … Tune, L. E. (1997). Diagnosis and treatment of Alzheimer's disease and related disorders: Consensus statement of the American Association for Geriatric Psychiatry, The Alzheimer's Association and the American Geriatrics Society. *Journal of the American Medical Association, 278*, 1363–1371.

Spier, S. A., Tesar, G. E., Rosenbaum, J. F., & Woods, S. W. (1986). Treatment of panic disorder and agoraphobia with clonazepam. *Journal of Clinical Psychiatry, 47*, 238–242.

Spiers, P. A., & Hochanadel, G. (1999). Citicholine for traumatic brain injury: Report of two cases, including my own. *Journal of International Neuropsychological Society, 5*, 260–264.

Spinella, M., & Eaton, L. A. (2002). Hypomania induced by herbal and pharmaceutical psychotropic medicines following mild traumatic brain injury. *Brain Injury, 16*, 359–367.

Spitz, G., Alway, Y., Gould, K. R., & Ponsford, J. L. (2017). Disrupted white matter microstructure and mood disorders after traumatic brain injury. *Journal of Neurotrauma, 34*, 807–815.

Springer, J., & Chollet, A. (2001). A traumatic car crash. *Lancet, 357*, 1848.

Srienc, A., Narang, P., Sarai, S., Xiong, Y., & Lippman, S. (2018). Is electroconvulsive therapy a treatment for depression following traumatic brain injury? *Innovations in Clinical Neuroscience, 15*(3-4), 43–46.

Stanislav, S. W. (1997). Cognitive effects of antipsychotic agents in persons with traumatic brain injury. *Brain Injury, 11*, 335–341.

Stanislav, S. W., & Childs, A. (2000). Evaluating the usage of droperidol in acutely agitated persons with brain injury. *Brain Injury, 14*, 261–265.

Starkstein, S., & Leentjens, A. F. (2008). The nosologic position of apathy in clinical practice. *Journal of Neurology, Neurosurgery and Psychiatry, 79*, 1088–1092.

Starkstein, S. E., Boston, J. D., & Robinson, R. G. (1988). Mechanisms of mania after brain injury: 12 case reports and review of the literature. *The Journal of Nervous and Mental Disease, 176*, 87–100.

Stengler-Wenzke, K., & Muller, U. (2002). Fluoxetine for OCD after brain injury. *American Journal of Psychiatry, 159*, 872.

Stewart, J. T., & Hemsath, R. H. (1988). Bipolar illness following traumatic brain injury: Treatment with lithium and carbamazepine. *Journal of Clinical Psychiatry, 49*, 74–75.

Stierwalt, J. A. G., & Murray, L. L. (2002). Attention impairment following traumatic brain injury. *Seminars in Speech and Language, 23*, 129–138.

Tan, C. L., Alavi, S. A., Baldeweg, S. E., Belli, A., Carson, A., Feeney, C., … Hutchinson, P. J. (2017). The screening and management of pituitary dysfunction following traumatic brain injury in adults: British Neurotrauma Group guidance. *Journal of Neurology, Neurosurgery, and Psychiatry, 88*, 971–981.

Tateno, A., Jorge, R. E., & Robinson, R. G. (2003). Clinical correlates of aggressive behavior after traumatic brain injury. *Journal of Neuropsychiatry & Clinical Neurosciences, 15*, 155–160.

Tateno, A., Jorge, R. E., & Robinson, R. G. (2004). Pathological laughing and crying following traumatic brain injury. *The Journal of Neuropsychiatry & Clinical Neurosciences, 16*, 426–434.

Taverni, J. P., Seliger, G., & Lichtman, S. W. (1998). Donepezil-mediated memory improvement in traumatic brain injury during post acute rehabilitation. *Brain Injury, 12*, 77–80.

Taylor, C. A., Bell, J. M., Breiding, M. J., & Xu, L. (2017). Traumatic brain injury-related emergency department visits, hospitalizations, and deaths-United States, 2007 and 2013. *MMWR Surveillance Summaries, 66*(9), 1–16.

Teasdale, T. W., & Engberg, A. W. (2001). Suicide after traumatic brain injury: A population study. *Journal of Neurology, Neurosurgery and Psychiatry, 71*, 436–440.

Teitelman, E. (2001). Off-label uses of modafinil. *The American Journal of Psychiatry, 158*, 1341.

Teng, C. J., Bhalerao, S., Lee, Z., Farber, H. M., Foran, T., & Tucker, W. (2001). The use of bupropion in the treatment of restlessness after a traumatic brain injury. *Brain Injury, 15*, 463–467.

Tenovuo, O. (2005). Central acetylcholinesterase inhibitors in the treatment of chronic traumatic brain injury—Clinical experience in 111 patients. *Progress in Neuro-Psychopharmacology & Biological Psychiatry, 29*, 61–67.

Tenovuo, O., Alin, J., & Helenius, H. (2009). A randomized controlled trial of rivastigmine for chronic sequels of traumatic brain injury-what it showed and taught? *Brain Injury, 23*(6):548–558.

Theadom, A., Cropley, M., Parmar, P., Barker-Collo, S., Starkey, N., Jones, K., … on behalf of the BIONIC Group. (2015). Sleep difficulties one year following mild traumatic brain injury in a population-based study. *Sleep Medicine, 16*, 926–932.

Thomsen, I. V. (1984). Late outcome of very severe blunt head trauma: A 10–15 year second-follow-up. *Journal of Neurology, Neurosurgery, and Psychiatry, 47*, 260–268.

Tiersky, L. A., Anselmi, V., Johnston, M. V., Kurtyka, J., Roosen, E., Schwartz, T., & DeLuca, J. (2005). A trial of neuropsychologic rehabilitation in mild-spectrum traumatic brain injury. *Archives in Physical Medicine & Rehabilitation, 86*, 1565–1574.

Tremeau, F., Mauro, C. J., Shope, C., Riber, L. M., Dhami, S., & Citrome, L. (2011). High dose quetiapine in the treatment of psychosis due to traumatic brain injury. A case report. *Progress in Neuro-Psychopharmacology & Biological Psychiatry, 35*, 280–281.

Trimble, M. R. (1991). Interictal psychoses of epilepsy. *Advances in Neurology, 55*, 143–152.

Tsaousides, T., Cantor, J. B., & Gordon, W. A. (2011). Suicidal ideation following traumatic brain injury: Prevalence rates and correlates in adults living in the community. *The Journal of Health Trauma Rehabilitation, 26*(4), 265–275.

Turkalj, I., Stojanovic, S., Petrovic, K., Njagulj, V., Mikov, P., & Spanovic, M. (2012). Psychosis following stab brain injury by a billiard stick. *Hippokratia, 16*(3), 275–277.

Turner-Stokes, L., Nibras, H., Pierce, K., & Clegg, F. (2002). Managing depression in brain injury rehabilitation: The use of an integrated care pathway and preliminary report of response to sertraline. *Clinical Rehabilitation, 16*, 261–268.

Ummar, S., Kumar, N., & Ramanathan, S. A. (2016). Organic bipolar disorder: An unusual neuropsychiatric sequelae following right frontotemporal injury. *Indian Journal of Psychological Medicine, 38*(3), 257–259.

Underhill, A. T., Lobello, S. G., Stroud, T. P., Terry, K. S., Devivos, M. J., & Fine, P. R. (2003). Depression and life satisfaction in patients with traumatic brain injury: A longitudinal study. *Brain Injury, 17*, 973–982.

Vaishnavi, S., Rao, V., & Fann, J. R. (2009). Neuropsychiatric problems after traumatic brain injury: Unraveling the silent epidemic. *Psychosomatics, 50*, 198–205.

Van Reekum, R., Bayley, M., Garner, S., Burke, I. M., Fawcett, S., Hart, A., & Thompson, W. (1995). N of 1 study: Amantadine for the amotivational syndrome in a patient with traumatic brain injury. *Brain Injury, 9*, 49–53.

Van Reekum, R., Bolago, I., Finlayson, M. A. J., Garner, S., & Links, P. S. (1996). Psychiatric disorders after traumatic brain injury. *Brain Injury, 10*, 319–327.

Vanderploeg, R. D., Curtiss, G., & Belanger, H. G. (2005). Long-term neuropsychological outcomes following mild traumatic brain injury. *Journal of International Neuropsychological Society, 11*, 228–236.

Vasterling, J. J., Brailey, K., Proctor, S. P., Kane, R., & Heeren, T. (2012). Neuropsychological outcomes of mild traumatic brain injury, post-traumatic stress disorder and depression in Iraq-deployed US Army soldiers. *The British Journal of Psychiatry, 201*, 186–192.

Vasterling, J. J., Jacob, S. N., & Rasmusson, A. (2018). Traumatic brain injury and posttraumatic stress disorder: Conceptual, diagnostic, and therapeutic considerations in the context of co-occurrence. *Journal of Neuropsychiatry and Clinical Neurosciences, 30*(2), 91–100.

Viana, B. D. M., Prais, H. A. C., Nicolato, R., & Caramelli, P. (2010). Posttraumatic brain injury psychosis successfully treated with olanzapine. *Progress in Neuro-Psychopharmacology & Biological Psychiatry, 34*, 233–235.

Viola-Saltzman, M., & Musleh, C. (2016). Traumatic brain injury-induced sleep disorders. *Neuropsychiatric Disease and Treatment, 12*, 339–348.

Waldron, B., Casserly, L. M., & O'Sullivan, C. (2013). Cognitive behavioural therapy for depression and anxiety in adults with acquired brain injury. What works for whom? *Neuropsychological Rehabilitation, 23*(1), 64–101.

Warden, D. L., Labbate, L. A., Salazar, A. M., Nelson, R., Sheley, E., Staudenmeier, J., & Martin, E. (1997). Posttraumatic stress disorder in patients with traumatic brain injury and amnesia for the event? *Journal of Neuropsychiatry & Clinical Neurosciences, 9*, 18–22.

Watson, C., Rutterford, N. A., Shortland, D., Williamson, N., & Alderman, N. (2001). Reduction of chronic aggressive behavior 10 years after brain injury. *Brain Injury, 15*, 1003–1005.

Weiner, M. W., Harvey, D., Hayes, J., Landau, S. M., Aisen, P. S., Petersen, R. C., … Department of Defense Alzheimer's Disease Neuroimaging Initiative. (2017). Effects of traumatic brain injury and posttraumatic stress disorder on development of Alzheimer's disease in Vietnam veterans using the Alzheimer's disease neuroimaging initiative: Preliminary report. *Alzheimer's Dement (NY), 3*(2), 177–188.

Wesolowski, M. D., Zencius, A., & Burke, W. H. (1993). Effects of feedback and behavior contracting on head trauma person's inappropriate sexual behavior. *Behavourial Residential Treatment, 8*, 89–96.

Wheaton, P., Mathias, J. L., & Vink, R. (2011). Impact of pharmacological treatments on cognitive and behavioral outcome in the postacute stages of adult traumatic brain injury: A meta-analysis. *Journal of Clinical Psychopharmacology, 31*(6), 745–757.

Whelan, F. J., Walker, M. S., & Schultz, S. K. (2000). Donepezil in the treatment of cognitive dysfunction associated with traumatic brain injury. *Annals in Clinical Psychiatry, 12*, 131–135.

Whelan-Goodinson, R., Ponsford, J., Johnston, L., & Grant, F. (2009). Psychiatric disorders following traumatic brain injury: Their nature and frequency. *The Journal of Head Trauma Rehabilitation, 24*(5), 324–332.

Whiting, W. L., Sullivan, G. A., & Stewart, J. T. (2016). Lamotrigine treatment for agitation following traumatic brain injury. *Psychosomatics, 57*(3), 330–333.

Whitlock, J. A. (1999). Brain injury, cognitive impairment and donepezil. *The Journal of Head Trauma Rehabilitation, 14*, 424–427.

Whyte, J., Hart, T., Bode, R. K., & Malec, J. F. (2003). The Moss attention rating scale for traumatic brain injury: Initial psychometric assessment. *Archives in Physical Medicine & Rehabilitation, 84*, 268–276.

Whyte, J., Hart, T., Schuster, K., Fleming, M., Polansky, M., & Coslett, H. B. (1997). Effects of methylphenidate on attentional function after traumatic brain injury: A randomized, placebo-controlled trial. *American Journal of Physical Medicine & Rehabilitation, 76*, 440–450.

Whyte, J., Hart, T., Vaccaro, M., Grieb-Neff, P., Risser, A., Polansky, M., & Coslett, H. B. (2004). Effects of methylphenidate on attention deficits after traumatic brain injury. A multidimensional, randomized, controlled trial. *American Journal of Physical Medicine & Rehabilitation, 83*, 401–420.

Whyte, J. W., Vaccaro, M., Grieb-Neff, P., & Hart, T. (2002). Psychostimulant use in the rehabilitation of individuals with traumatic brain injury. *The Journal of Head Trauma Rehabilitation, 17*, 284–299.

Wickwire, E. M., Schnyer, D. M., Germain, A., Williams, S. G., Lettieri, C. J., McKeon, A. B., … Manley, G. T. (2018). Sleep, sleep disorder, and circadian health following mild traumatic brain injury in adults: Review and research agenda. *Journal of Neurotrauma, 35*, 2615–2631.

Williams, D. B., Annegers, J. F., Kokmen, E., O'Brien, P. C., & Kurland, L. T. (1991). Brain injury and neurologic sequelae: A cohort study of dementia, parkinsonism and amyotrophic lateral sclerosis. *Neurology, 41*, 1554–1557.

Williams, S. M., Peltz, C., Yaffe, K., Schulz, P., & Sierks, M. R. (2018). CNS disease-related protein variants as blood-based biomarkers in traumatic brain injury. *Neurology, 91*, 702–709.

Williams, W. H., Evans, J. J., & Fleminger, S. (2003). Neurorehabilitation and cognitive-behaviour therapy of anxiety disorders after brain injury: An overview and a case illustration of obsessive-compulsive disorder. *Neuropsychological Rehabilitation, 13*, 133–148.

Wilson, S. A. K. (1924). Some problems in neurology. II: Pathological laughter and crying. *The Journal of Neurology and Psychopathology, 4*, 299–333.

Wilson, S. L., Powell, G. E., Brock, D., & Thwaites, H. (1996). Vegetative state and responses to sensory stimulation: An analysis of 24 cases. *Brain Injury, 10*, 807–818.

Woodman, C. L., & Noyes, R. (1994). Panic disorder: Treatment with valproate. *Journal of the Clinical Psychiatry, 55*, 134–136.

Work, S. S., Colamonico, J. A., Bradley, W. G., & Kaye, R. E. (2011). Pseudobulbar affect: An under-recognized and under-treated neurological disorder. *Advances in Therapy, 28*, 586–601.

Wortzel, H. S., & Arciniegas, D. B. (2013). A forensic neuropsychiatric approach to traumatic brain injury, aggression, and suicide. *The Journal of the American Academy of Psychiatry and the Law, 41*(2), 274–286.

Wortzel, H. S., & Arciniegas, D. B. (2014). The DSM-5 approach to the evaluation of traumatic brain injury and its neuropsychiatric sequelae. *NeuroRehabilitation, 34*, 613–623.

Wroblewski, B. A., Joseph, A. B., & Cornblatt, R. R. (1996). Antidepressant pharmacotherapy and the treatment of depression in patients with severe traumatic brain injury: A controlled, prospective study. *Journal of Clinical Psychiatry, 57*, 582–587.

Wroblewski, B. A., Joseph, A. B., Kupfer, J., & Kalliel, K. (1997). Effectiveness of valproic acid on destructive and aggressive behaviors in patients with acquired brain injury. *Brain Injury, 11*, 37–47.

Yue, J. K., Burke, J. F., Upadhyayula, P. S., Winkler, E. A., Deng, H., Robinson, C. K., … Tarapore, P. E. (2017). Selective serotonin reuptake inhibitors for treating neurocognitive and neuropsychiatric disorders following traumatic brain injury: An evaluation of current evidence. *Brain Sciences, 7*(8), 93.

Yurgelun-Todd, D. A., Bleuler, C. E., McGlade, E. C., Churchwell, J. C., Brenner, L. A., & Lopez-Larson, M. P. (2011). Neuroimaging correlates of traumatic brain injury and suicidal behavior. *The Journal of Head Trauma Rehabilitation, 26*(4), 276–289.

Zafonte, R. D., Watanabe, T., & Mann, N. R. (1998). Amantadine: A potential treatment for the minimally conscious state. *Brain Injury, 12*, 617–621.

Zarrouf, F. A., Artz, S., Griffith, J., Sirbu, C., & Kommor, M. (2009). Testosterone and depression: Systematic review and meta-analysis. *Journal of Psychiatric Practice, 15*, 289–305.

Zasler, N. D., & Martelli, M. F. (2005). Sexual dysfunction. In J. M. Silver, T. W. McAllister, & S. C. Yudofsky (Eds.), *Textbook of traumatic brain injury* (pp. 437–450). Washington, DC: American Psychiatric Press Inc.

Zeilig, G., Drubach, D. A., Katz-Zeilig, M., & Karatinos, J. (1996). Pathological laughter and crying in patients with closed traumatic brain injury. *Brain Injury, 10*, 591–597.

Zencius, A., Wesolowski, M., Burke, W., & Hough, S. (1990). Managing hypersexual disorders in brain-injured clients. *Brain Injury, 4*, 175–181.

Zhang, L., Plotkin, R. C., Wang, G., Sandel, M. E., & Lee, S. (2004). Cholinergic augmentation with donepezil enhances recovery in short-term memory and sustained attention after traumatic brain injury. *Archives in Physical Medicine & Rehabilitation, 85*, 1050–1055.

Zincir, S. B., Izci, F., & Acar, G. (2014). Mania secondary to traumatic brain injury: A case report. *Journal of Neuropsychiatry and Clinical Neurosciences, 26*(2), E31.

Chapter 11
Neuropsychology in the Outpatient Rehabilitation Setting

Rosanne Pachilakis and Kathryn Mirra

What Is Neuropsychology?

Clinical neuropsychology is a subspecialty within the field of psychology emphasizing the applied science of brain–behavior relationships. It is a recognized specialty by the American Psychological Association (APA) and Canadian Psychological Association (CPA). A clinical neuropsychologist is a professional with advanced clinical training who may engage in assessment, diagnosis, treatment, and/or rehabilitation of individuals across the lifespan with various conditions and presentations, most typically stemming from neurological, medical, developmental, and/or psychiatric causes (National Academy of Neuropsychology (NAN), 2001). Clinical neuropsychologists objectively evaluate the presenting cognitive, behavioral, and emotional sequelae stemming from various etiologies, to inform diagnostic impression, and provide treatment recommendations to further assist the medical and rehabilitation teams, as well as the individual and family members.

Rehabilitation psychology in general emphasizes teamwork in an effort to aim for optimal rehabilitation gains after injury or illness and employs a holistic, interdisciplinary approach to care, including medical, psychological, social, cultural, and environmental considerations. The rehabilitation-oriented neuropsychologist can provide a unique contribution to the treatment team through relating their understanding of brain–behavior basis of injury and illness to inform clinical care efforts, monitor progress throughout the recovery process, and provide added insight into the potential long-term impact on daily functioning, safety, and inform appropriate goal-setting and planning.

According to the guidelines initially purported in the Houston Conference on Specialty Education and Training in Clinical Neuropsychology (1998) and more recently revisited in an interorganizational effort, a clinical neuropsychologist

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specializes in the application of assessment and intervention principles based on the scientific study of brain–behavior as related to normal versus abnormal functioning of the central nervous system. A clinical neuropsychologist has obtained a doctoral degree in psychology (Ph.D. or Psy.D.) from an accredited university. Course of study should provide education in generic psychology and a clinical core, in addition to the foundations of the brain–behavior relationship and clinical neuropsychology practice. As a component of graduate-level education, clinical work in the form of practicum/externships in various community settings supports the student in beginning supervised exposure to clinical practice. The American Academy of Clinical Neuropsychology (AACN) has published guidelines for practicum training based on a workgroup of multiple senior-level neuropsychologists (Nelson et al., 2015). The current guidelines indicate that a neuropsychologist must complete a predoctoral 1-year equivalent internship in a clinically relevant area and the equivalence of 2 years of supervised postdoctoral level specialty training in the extended study and professional practice of clinical neuropsychology. A neuropsychologist must hold the license for independent practice of psychology in his/her state or province having completed all necessary requirements, which vary by location but may include components of a credential review, written and/or oral examinations, and/or focused demonstration of ethics procedures and knowledge base through varied means. Depending on the practice location, requirements for maintaining a psychology license (e.g., continuing education credits) need be continually met.

Though not a requirement, board certification through an established credentialing board is encouraged to further evidence to the public one's competence to practice in the profession, as one has demonstrated skill through completion of a rigorous peer-review process, including formal credential verification and various examination procedures (e.g., written and oral examinations). Board certification is one mechanism aimed to define and measure an individual provider's competence and serves as a means to consumer/patient protection and has also been shown to have benefits to the individual psychologist (Cox, 2010). The American Board of Professional Psychology (ABPP) established in 1947 through sponsorship of the APA is one credentialing body of specialists in multiple concentrations within the field of psychology (15 specialty boards and one subspecialty at the time of this writing), including clinical neuropsychology and rehabilitation psychology. There has been a published book series on special competencies in professional psychology, prepared with the intention to describe functional and foundational competencies in the specialty areas within psychology, including clinical neuropsychology (Lamberty & Nelson, 2012) and rehabilitation psychology (Cox, Cox, & Caplan, 2013).

Role of the Neuropsychologist in Brain Injury Rehabilitation

Neuropsychologists can serve in various roles depending on the setting where they practice. Based on recommendations of national organizations (e.g., American Congress of Rehabilitation Medicine, ACRM) and accrediting bodies (e.g.,

Commission on Accreditation of Rehabilitation Facilities, CARF), the incorporation and inclusion of psychologists is considered best practice in brain injury rehabilitation at the inpatient and outpatient levels of care. The interdisciplinary rehabilitation team is made up of providers from multiple disciplines with special interest, training, experience, and expertise in head injury rehabilitation, and serves an individual in collaborative manner to assess and coordinate clinical care efforts. At the inpatient level, there are state mandates which identify the areas of specialty needed to serve as part of the treatment team, at minimum those most often include physicians, nursing staff, physical therapists, occupational therapists, speech-language pathologists, social workers, nutrition/dietician, recreation therapists, and clinical psychologists with training and experience in neuropsychology (e.g., NYS 405 regulations).

Within outpatient settings, the disciplines needed for acute medical management (e.g., nursing) are no longer warranted and often play limited roles, with involvement more periodically and in consultation with specialists, as needed on an individual basis. In outpatient brain injury rehabilitation, in addition to the clinical role as a member of the interdisciplinary team, neuropsychologists often serve in leadership positions and may engage in administrative duties (Karol, 2014). The neuropsychology service may be designed as a consultation model, such that services are requested on an individual basis to further evaluate cognitive, behavioral, and emotional functioning to provide insight into level of functioning and inform care plans and treatment recommendations. An evaluation can serve to raise insight into potential barriers or inform appropriateness of treatment efforts and refine recommendations.

In addition, many neuropsychologists serve in supervisory roles by teaching coursework, training, and providing mentorship to graduate students pursuing degrees within the field. Neuropsychologists are trained in psychometrics, test construction, research methodology and design, and statistical analysis. This translates to promoting evidence-based practice and often engagement in ongoing research efforts/involvement within the interdisciplinary rehabilitation setting and/or larger medical institution to improve outcomes and quality of care.

Clinical Responsibilities of Neuropsychologists in Outpatient Settings

Assessment

The neuropsychological evaluation serves to provide an objective assessment of an individual's cognitive and emotional functioning, daily activities, and behavioral presentation. A comprehensive assessment will serve to assist the treatment team, family, and the individual to foster greater understanding into the pattern of cognitive strengths and weaknesses of the individual, the nature of presenting symptoms,

further inform understanding based on clinical features and injury/illness characteristics, and can promote individualized, case-specific evidence-based practice recommendations, and guide efforts to develop appropriate care plans. Counter to other settings where the goal of a neuropsychological evaluation may often be to inform diagnostic impression, often injury/illness characteristics are known to the neuropsychologist in rehabilitation and the role involves characterization of skill set and integration to bolster treatment and management efforts.

Treatment

A neuropsychologist is a trained clinical psychologist and therefore has experience and has shown competence in providing psychotherapy services. In outpatient brain injury rehabilitation, neuropsychologists often provide psychotherapy/counseling services to varying degrees. The typical aim of treatment is often to address maladjustment to the presenting injury or illness, assess coping, and provide added support and education for the individual and family, with the application and utilization of various appropriate therapeutic techniques. Cognitive rehabilitation efforts may often be intertwined into psychotherapy sessions depending on the make-up, qualifications, and responsibilities of specific treatment team members in the setting. As described elsewhere in this book, additional team members, such as speech-language pathologists and occupational therapists may also address cognition (see Chaps. 7 and 9) neuropsychologists may serve to support efforts to integrate compensatory strategy use. Behavioral management may also be a component of psychotherapy services depending on the presenting features and individual needs. Sometimes the neuropsychologist may take on case management duties in an effort to coordinate care across disciplines, such as through relaying observations of the rehabilitation team to the treating physician, or may provide guidance in the exploration and/or application process for appropriate community support programs.

The Neuropsychological Evaluation

Common Referral Questions

Following an acquired brain injury, individuals may be referred for neuropsychological evaluation at various time points in their recovery and depending on timing the referral question will vary. The evaluation serves to provide an objective measure regarding the presence of cognitive impairment. Early in the recovery process, whether when receiving inpatient services or initiating outpatient services, an evaluation can assist the team to target present difficulties and further promote holistic

understanding of the person's abilities. Further along in recovery, an evaluation can document skills to inform appropriateness of community reintegration efforts, such as when considering a return to school/work and other aspects of daily functioning (e.g., driving). There are no established guidelines for when neuropsychology should be consulted and in regard to the timing of neuropsychological testing, rather this is individualized based on the needs of the individual and referral question being asked.

Outpatient Evaluation Process

The evaluation process often begins with a review of available medical records and thorough clinical interview. Depending on practice setting, the amount of available documentation prior to initial patient interaction will vary. The clinical interview often involves the patient and family/caregivers to provide collateral report. This is important given the potential for reduced insight and/or cognitive challenges that could present as barriers for the patient to relay accurate details of their history, as well as their current level of functioning or activity. A thorough interview would involve discussion of the nature of the injury/illness, their presenting cognitive, emotional, and physical symptoms, degree of independence with activities of daily living (e.g., self-care) and instrumental activities of daily living (e.g., financial management, medication management, driving), as well as a comprehensive review of background information, including medical and psychiatric history, substance use history, social history (e.g., education, employment), and review of family medical history, when appropriate. The daily routine and presence or absence of support services may also be highlighted, as such a discussion of daily/weekly activities may be useful to tailor specific recommendations. The presence of family members can also assist with beginning to identify additional community resources that may be useful for the family system given any perceived current challenges and needs. Behavior presentation and or personality changes should also be assessed on interview and possibly through self-report and/or informant questionnaires, if deemed appropriate, as such change after brain injury is often typical and can have significant impact on functioning.

The testing portion of an evaluation involves objective measurement of cognitive functioning using standardized neuropsychological and psychological measures to assess cognition, presenting symptoms (mood, anxiety), and/or personality. Most of the common measures used are paper-based, with some computer applications available. A comprehensive neuropsychological battery will involve evaluation across multiple cognitive domains, including attention and processing speed, executive functioning, language, learning and memory, visuospatial skills, sensorimotor skills, and psychological functioning. Indicators of premorbid functioning (e.g., single word reading) are used, in conjunction with demographic information gath-

ered on interview, to provide an estimate of the individual's ability level prior to the injury/illness. Various indicators of symptom and performance validity are included to assess engagement and used to inform interpretation and quality of data.

Test administration should be done in a quiet 1:1 environment according to standards of the test materials and clinical practice mandates. The chosen battery given should be selected according to the providers practice model, with most clinical neuropsychologists (82% of respondents) employing a flexible, hypothesis-testing battery selection process according to the 2015 TCN Professional Practice and Salary Survey (Sweet, Benson, Nelson, & Moberg, 2015), meaning standardized test measures are selected and a battery administered based on individual needs to address the presenting referral question with variations case-by-case. Typically, the data collection phase of an evaluation can take several hours, which varies based on selected battery and ability level of the individual. Based on various factors (e.g., fatigue, physical features) or logistics, the testing may occur in a single session or be broken-up into multiple sessions, depending on the provider's practice model.

The test performances are then scored based on standardized procedures and protocol and compared to appropriate normative samples according to the neuropsychologist's practice methods. Results and pattern of performance are further interpreted by the neuropsychologist to inform their clinical impressions and formulate treatment recommendations accordingly. Findings are relayed to the individual and family and treatment team in various forms, often in an in-person feedback session or through follow-up phone consultation, relayed in meetings with the interdisciplinary team, and as a written report generated to inform the referral source/medical team of results, impression, and recommendations. This document will also often summarize the background and presenting symptoms as discussed during the clinical interview, though the written product will vary by provider. Psychoeducation about the findings and ramifications is often emphasized in the feedback to further promote recovery efforts and consider appropriate care referrals. Discussion of the mechanism of injury to assist better understanding of the present complaints can help the individual and family members address challenges in novel ways.

Repeated neuropsychological evaluations can be useful to assess cognitive trajectory over time, inform diagnostic impression based on comparison of results, and to revisit treatment recommendations, including considering appropriateness of ongoing rehabilitation efforts. While some degree of improvement is often expected, difficulties that were not in the forefront early in the rehabilitation process can emerge, when the individual attempts reintegration and begins to increase responsibilities. Emotional needs and behavioral ramifications can vary throughout the recovery process and periodic evaluation can allow needs to be revisited. For instance, when an individual moves further along and gains insight into their degree of ongoing disability, emotionally salient features can emerge and consultation with neuropsychology at a later time point could assist to promote ways to optimally address new-onset symptoms.

Case Examples

Case Example 1

Presenting Features and Background Information

Case 1 is a middle-aged, right-handed female, found unresponsive at home. Her duration of unconsciousness was unknown. Subsequent medical workup revealed a posterior subarachnoid hemorrhage due to aneurysm rupture, with bihemisphere involvement. She underwent endovascular coiling procedure. Her recovery was complicated by hydrocephalus and a shunt was placed. After several weeks, she was transferred to an acute inpatient rehabilitation unit for 1 month to address changes in her cognitive skills and physical functioning (i.e., speech-language therapy (ST), occupational therapy (OT), and physical therapy (PT)). She was then discharged to her home and began outpatient rehabilitation services. After several months of treatment and recovery gains, a comprehensive neuropsychological evaluation was requested to assess her current cognitive functioning and help guide community and work reintegration recommendations. Prior to her hospitalization, she was working as an office manager. She and her family indicated they were pleased with her progress and were hoping for her to resume driving and working in the near future. The neuropsychological evaluation was performed 7 months after her acquired brain injury.

Results and Recommendations

Results revealed difficulties with attention and concentration, and she needed additional time to perform tasks due to significantly slowed processing speed. She tended to make errors when working quickly, but was much more precise when she slowed her pace. She did well on complex problem-solving tasks. Her ability to learn and remember new information was intact, as was her language and visual processing abilities. Her outlook for the future was positive, and she was not endorsing significant emotional symptoms.

The results were discussed with Case 1 and her family, her therapists, and her referring physician. Recommendations included various compensatory strategies and provided guidance for return to work given the challenge her weakness in attention and speed could pose in an office setting.

Discussion

Case 1 exhibits the role a neuropsychologist can play in community reintegration, as the client was eager to resume working quickly and her various improvements in rehabilitation session supported continuing to increase responsibilities. However, the findings of the neuropsychological evaluation indicated the presence of specific

weaknesses which could greatly interfere with a successful work re-entry to office work, given the high need for attention to detail and efficiency in her prior position. Thus, discussion of a more appropriate work re-entry timeline was necessary with the client, her family, her medical providers, and the rehabilitation team. The neuropsychologist was able to generate specific recommendations and suggested workplace accommodations, which could assist in optimizing her success. It was also advised that she continue with her current rehabilitation program, targeting her specific weaknesses identified in the neuropsychological profile to individualize her treatment plan, with the goal of mock vocational exercise tailored to her role and supporting her gradual reintegration. Her reported goal to resume driving was also addressed, as readiness seemed likely. She was recommended to the appropriate community resource for an on-road driving safety evaluation, following consultation with her medical provider.

Follow-Up: Three months after the neuropsychological evaluation, the client continued to receive outpatient ST and OT services. She obtained medical clearance and passed her on-road driving evaluation. She coordinated work re-entry with her employer, and her job duties were adjusted to accommodate her present need and various supports were afforded to her. She was successful in her return and was gradually increasing responsibilities.

Case Example 2

Presenting Features and Background Information

Case 2 is a right-handed male in his early 60s, who experienced a left hemisphere stroke 18 months prior to his neuropsychological evaluation. He was referred for comprehensive neuropsychological evaluation by his neurologist to assess current cognitive functioning and inform postrehabilitation recommendations, as he was about to be discharged from outpatient treatment. The treatment team had identified multiple areas of residual deficit, and he also continued to manifest a right-sided hemiparesis. He was retired and resided with his wife.

Results and Recommendations

Results of the neuropsychological evaluation were consistent with a residual aphasia involving his expressive and receptive language. Though able to engage in simple informal conversations, he had significant difficulty when trying to convey complex thoughts and feelings, and he was unable to read or write. He also demonstrated difficulty with attention skills, and he was slow to process information. Executive functioning/reasoning skills were weak. Visuospatial skills were variable, such that construction was intact. Learning and memory were intact and represented a relative strength. He endorsed severe symptoms of anxiety and depression. He

denied suicidal ideation. Recommendations included postrehabilitation programs, community support resources, need for ongoing assistance with various daily tasks, and compensatory strategies to help boost daily efficiency.

Discussion

Case 2 highlights the role a neuropsychologist can play in objectively evaluating the degree of deficits to inform ongoing management of individuals with complex cognitive and physical presentations. During the clinical interview, it was reported that the client in this case was driving immediately since leaving the hospital and continued to do so, unbeknownst to his rehabilitation team and medical providers. This was an obvious safety concern given his compromised cognition and called into question his judgment and safety awareness. These concerns were immediately discussed with him, family, and the rehabilitation and medical team. He was recommended to cease driving and reasons for this were thoroughly explained. He was advised to further discuss with his medical providers his stated desire to continue driving. Education and guidance were provided to his spouse regarding his need for ongoing assistance and supervision with daily tasks. Given his prominent language dysfunction, it was recommended that she, or a trusted family member, accompany him to medical appointments to ensure proper carryover of information to assist care. The neuropsychologist also emphasized the need to continue addressing general health conditions that could be further interfering with daily efficiency, and reinforced the importance of continued medical management.

Case Example 3

Presenting Features and Background Information

Case 3 is a college student in his early 20s, who sustained a sports-related concussion, without loss of consciousness, 18 months prior. He was referred for neuropsychological evaluation by neurology to further assess cognitive functioning. He noted vestibular imbalance initially, and subsequently experienced nausea and vomiting. Medical screening evaluation was unremarkable. He developed worsening back pain, fatigue, sleep problems, and nervousness, which continued. He had experienced headaches throughout his adolescence, but indicated increased frequency since his concussion. He denied significant cognitive difficulties, though noted some added challenge in his coursework, such that he had to employ added effort and organization strategies. He denied depression, but acknowledged increased anxiety and emotionality. Despite his reported symptoms, he continued to perform well academically and was involved in multiple clubs and activities on campus. He worked part-time and was beginning to consider career choices.

Results and Recommendations

Results of the neuropsychological evaluation revealed intact (average or better) functioning across all cognitive domains, including basic attention, executive functioning, learning and memory, language, and visuospatial skills. He exhibited elevated anxiety on a self-report measure, with heightened concern regarding his health noted on interview. Recommendations included further addressing heightened anxiety and stress through psychotherapy, sleep hygiene, continued optimal headache management, and involved discussion of general strategies, which may optimize organization and study habits.

Discussion

The role of the neuropsychologist in concussion management involves education, managing expectations, and comprehensive evaluation, when appropriate (Nelson et al., 2018). Though improvement is expected typically within 3 months of the event, many experience resolution of symptoms in just a few weeks (Vanderploeg, Belanger, & Kaufmann, 2014). Given that Case 3's concussion was 18 months prior, it was not expected that his current symptoms would be associated. The psychometric evaluation in this case served to confirm that cognitive functioning was intact across all domains, as expected. The clinical interview and his self-report inventories highlighted several modifiable factors that could likely interfere with his cognitive efficiency at times, namely, his ongoing physical symptoms (headache, pain) and untreated anxiety, which predated the concussion. In a feedback session, recommendations to address his anxiety were emphasized, with a short-course of community psychotherapy encouraged to assist him in integrating healthy stress management and relaxation strategies (e.g., deep breathing, meditation, and mindfulness) further into his routine and to promote proper sleep hygiene. The neuropsychologist served a unique role in providing necessary education and reassurance regarding his concussion and recovery, while being able to objectively evaluate his cognitive functioning given subjective experience of change. Recommendations were geared toward moving the client forward in his academic and vocational planning, as well as addressing longstanding emotional needs optimally.

Follow-Up: Three months after the evaluation, he had initiated psychotherapy within the community and was working on stress management strategies with personal report of improvement. He also successfully completed a summer internship. He reported that his various symptoms and complaints were improving, if not fully resolved. He was exploring options and looking forward to applying to graduate school.

Conclusion

The role of the neuropsychologist practicing in an outpatient brain injury rehabilitation setting can vary greatly from the responsibilities and approach in other treatment and practice settings. In rehabilitation, the neuropsychologist is often an interdisciplinary team member with a unique and highly specialized knowledge-base, extensive training, and expertise into the brain–behavior relationship. This allows the neuropsychologist to serve as an evaluator, therapist, consultant, teacher, mentor, and researcher. Often the neuropsychologist is involved in comprehensive cognitive assessment and psychotherapeutic and/or behavioral interventions to address the individualized needs of a client, family members/caregivers, and the medical and rehabilitation team. Neuropsychologists are viewed as experts on individualizing plan of care and practicing with a holistic approach.

References

Cox, D. R. (2010). Board certification in professional psychology: Promoting competency and consumer protection. *The Clinical Neuropsychologist, 24*, 493–505. https://doi.org/10.1080/13854040902802947

Cox, D. R., Cox, R. H., & Caplan, B. (2013). *Specialty competencies in rehabilitation psychology*. New York, NY: Oxford University Press.

Hannay, H. J., Bieliauskas, L. A., Crosson, B. A., Hammeke, T. A., Hamsher, K. d S., & Koffler, S. P. (1998). Proceedings of the Houston Conference on Specialty Education and Training in Clinical Neuropsychology. *Archives of Clinical Neuropsychology, 13*(2), 157–250.

Karol, R. L. (2014). Team models in neurorehabilitation: Structure, function, and culture change. *NeuroRehabilitation, 34*, 655–669. https://doi.org/10.3233/NRE-141080

Lamberty, G. J., & Nelson, N. W. (2012). *Specialty competencies in clinical neuropsychology*. New York, NY: Oxford University Press.

National Academy of Neuropsychology. (2001). Definition of a Clinical neuropsychologist. *Archives of Clinical Neuropsychology, 18*, 551–555.

Nelson, A. P., Roper, B. L., Slomine, B. S., Morrison, C., Greher, M. R., Janusz, J., … Wodushek, T. R. (2015). Official position of the American Academy of Clinical Neuropsychology (AACN): Guidelines for practicum, training in clinical neuropsychology. *The Clinical Neuropsychologist, 29*, 879–904. https://doi.org/10.1080/13854046.2015.1117658

Nelson, L. D., Furger, R. E., Ranson, J., Terima, S., Hammeke, T. A., Randolph, C., … McCrea, M. A. (2018). Acute clinical predictors of symptom recovery in emergency department patients with uncomplicated mild traumatic brain injury or non-traumatic brain injuries. *Journal of Neurotrauma, 35*, 249–259. https://doi.org/10.1089/neu.2017.4988

Sweet, J. J., Benson, L. M., Nelson, N. W., & Moberg, P. J. (2015). The American Academy of Clinical Neuropsychology, National Academy of Neuropsychology, and Society for Clinical Neuropsychology (APA Division 40) 2015 TCN professional practice and salary service: Professional practices, beliefs, and incomes of U.S. neuropsychologists. *The Clinical Neuropsychologist, 29*, 1069–1162. https://doi.org/10.1080/13854046.2016.1140228

Vanderploeg, R. D., Belanger, H. G., & Kaufmann, P. M. (2014). Nocebo effects and mild traumatic brain injury: Legal implications. *Psychological Injury and Law, 7*, 245–254. https://doi.org/10.1007/s12207-014-9201-3

Chapter 12
Counseling Individuals Post Acquired Brain Injury: Considerations and Objectives

Jean Elbaum

"I was just crossing the street, carrying bags full of presents to my friends for the holidays when I was hit by a car."

"I just got off the treadmill and I noticed that the right side of my body wasn't working right and I had trouble speaking."

"My partner and I fell 80 ft when our equipment broke. He died and I sustained a brain injury."

"I was on the way home from an Honor Society meeting in 12th grade and was hit by a car right near my school."

"I got into a car with a girl who had been drinking and taking pills and we drove right into a tree."

"I was working alone in my law office when someone came in and hit my head with a baseball bat several times."

"I lost oxygen to my brain after a heart attack I experienced when I was at the local library with my children."

"I lost oxygen to my brain due to a heroin and xanax overdose that made my heart stop."

"I started having headaches and experiencing these odd sensations where I would smell the scent of pine. I was diagnosed with a brain tumor."

"I was on vacation with my wife when our car was hit by a drunk driver, and as a result of the accident I totally lost my vision in addition to sustaining a brain injury."

"I told a lot of people that I fell down the stairs because I was carrying too much laundry, but my friends told me that I fell because I had been drinking too much."

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These types of introductions are familiar to members of the neurorehabilitation team. Although survivors generally don't remember their injury, they usually are able to describe what others told them occurred. Survivors of acquired brain injuries (ABIs) all need to cope with the suddenness of an unexpected, life changing injury. In counseling brain injury survivors, the clinician encounters individuals whose characteristics vary across a multitude of dimensions, not limited to age, gender, cultural background, severity of trauma, or time since the injury. The clinician meets survivors whose educational backgrounds range from limited to extensive, whose personality styles range from private to demonstrative, whose coping skills are fragile to admirable, and whose support systems are uninvolved to overly involved. Whether employed as a firefighter, janitor, professor, ironworker, physician, or fisherman at the moment of the injury, the individual abruptly becomes a brain injury survivor, trying to recapture as much of the positive aspects of his or her preinjury self as possible.

The exact nature of the injury will vary in terms of typicality, from the most common ABIs due to motor vehicle accidents, falls, or strokes to less common occurrences, such as unusual work accidents, random acts of violence, attempted suicides, and atypical encephalopathies, such as secondary to complications of anorexia or lariam-induced toxicity. Over the last decade, we have seen an increase in drug overdoses linked to heroin in combination with other drugs leading to cardiac arrest and loss of oxygen to the brain. The exact nature of the injury will also vary in terms of causal factors, number of others injured or killed, the survivor's relationship to the others involved, and how responsibility is assigned. Survivors will vary in terms of severity of challenges, from subtle to very pronounced. The framework within which the survivor views the injury will also affect his or her emotional status. All these factors must be considered in understanding the survivor and facilitating an effective postinjury counseling experience.

Counseling Considerations and Goals

Emotional and social challenges following a brain injury may be the result of organic damage, reaction to the injury, or a combination of both. These include loss of ability to show empathy, disinhibition, childish behavior, apathy, emotional lability, irritability, and suspiciousness. Typical emotional reactions to ABI seen clinically include sadness and frustration due to factors such as loss of identity, change in status, lengthy setbacks, diminished control, lack of home or work support, and loss of hope regarding the future.

Very common counseling goals include improving awareness, mood, frustration tolerance, attitude, stress management, self-esteem, and the reintegration to meaningful roles. In some situations, the counselor is able to facilitate post-ABI growth, where unexpected positive changes occur consequent to the injury.

It is critical that the entire team be aware of the impact of emotional and psychosocial changes on a survivor's recovery. Referral for counseling and/or neuropsychi-

atric consultation (see Chap. 10) allows the survivor the opportunity to address emotional and behavioral challenges that may be interfering with recovery.

The clinician must always be sensitive to the uniqueness of each individual, provide education and encouragement, boost motivation, be knowledgeable about cultural differences, help differentiate short- and long-term goals, and assist in creatively finding ways to eliminate barriers toward progress. The counselor should also reinforce that survivors keep notes of sessions to aid recall and actively attempt to carryover counseling goals to the home and community.

Survivors need to be able to express fears and concerns in an arena that offers trust and respect. Once trust is established and a therapeutic alliance has developed, an individual becomes more receptive to feedback. Timing and sensitivity are very important in providing challenging feedback.

The survivor often needs assistance in shifting from focus on postinjury changes to a more productive focus that involves reframing the injury in a manner that allows the acquisition of hope regarding the future. Whereas one-to-one counseling offers more individualized attention, group counseling helps alleviate feelings of isolation and difference. Peer support is also a powerful way for survivors to receive feedback about their behavior.

The Importance of Awareness

Awareness of deficit (AD) after ABI is a fascinating topic that has been researched extensively (Hart & Sherer, 2005; Prigatano & Schacter, 1991; Toglia & Kirk, 2000). Survivors that have good AD are often active partners in the recovery process. They are disturbed by their difficulties and eager to make progress. They have more favorable rehabilitation outcomes and do better in terms of psychological adjustment and social functioning (Leung & Liu, 2011). Individuals with poor insight have significant difficulty "seeing" post-brain-injury changes and how these difficulties affect daily living skills. Some survivors are totally unaware of very severe difficulties, and see themselves the way they were prior to the injury. These individuals can pose a very serious safety risk, as they may insist upon returning to activities prematurely; for example, return to work, school, or driving. Prigatano and Schacter (1991) describe the "catastrophic consequences" that lack of insight can have on employment choice and interpersonal relationships. Other survivors may have partial awareness of their difficulties, with underestimation of how their difficulties affect their performance. Reduced awareness is associated with more severe injuries and a greater number of brain lesions (Sherer, 2005).

Kortte, Wegener, and Chwalisz (2003) discuss the challenge of differentiating organic lack of awareness from psychological denial. They describe how those in denial of their deficits show resistance when shown their difficulties, whereas those with organic lack of awareness are surprised when their difficulties are pointed out to them. They found that individuals who show a higher level of denial tend to use a greater number of coping strategies aimed at avoidance. They conclude that those

who engage in avoidant coping strategies instead of actively processing the trauma are more at risk for depression.

Survivors who are unaware of their difficulties will vary in terms of receptivity to feedback. Some will be willing to follow clinical recommendations despite the fact they don't see their weaknesses. Crosson et al. (1989) provided a theoretical framework for self-awareness that consists of three levels: "intellectual awareness," "emergent awareness," and "anticipatory awareness." Intellectual awareness refers to the ability to recognize that particular difficulties exist secondary to an ABI. Emergent awareness refers to the ability to recognize the impact of these difficulties on everyday life. The model suggests that a person must possess some degree of both intellectual and emergent awareness before developing the third and highest level of anticipatory awareness. Anticipatory awareness refers to the ability to foresee difficulties in everyday life that could occur consequent to the injury and resultant impairments. The Dynamic Comprehensive Model of Awareness examines different aspects of metacognition including self-monitoring, error detection, and self-regulation (Toglia & Kirk, 2000). This model looks at the dynamic relationship between knowledge, belief, task demands, and context.

For many, awareness increases during the first few months postinjury, whereas for others it can take many years and repeated failure experiences to build awareness. Survivors ultimately need to establish a good level of insight so they can focus on ways to work around their deficits and become effective compensators.

LG was a 19-year-old college student status post a severe brain injury due to a pedestrian accident. Her insight into her difficulties was poor, and despite the fact that she had significant impairments in selective and divided attention, short-term memory, processing speed, visuoperceptual skills, fine motor coordination, and expressive and receptive language difficulties, she felt that she was "totally fine" and able to return to school. She was challenging in all her therapy sessions, stating that she was exactly as she had been prior to the accident.

Before the injury, LG was an extremely good student, with a 3.8 GPA. At 1 year following her injury, the neurorehabilitation team felt that LG was ready to take one course at a local college with reasonable accommodations, such as extended testing in a private room and use of a note taker. LG was totally resistant to the idea of working with the Office of Disabilities and insisted that she take a minimum of four courses. Despite individual and group counseling efforts supported by her family and friends, and a meeting with another college student post-TBI who had successfully reintegrated to school on a gradual basis with reasonable accommodations, LG was uncooperative with recommendations. She thought that returning to school would mean a return to her former self.

At this point, the team and family agreed that it would be helpful for LG to return to school full-time so that she could see her true status. This was a major turning point in LG's awareness level. Her insight finally improved as she saw for herself that she could not perform academically as in the past. She did very poorly in her classes. LG went through a period of reduced mood and increased anger about her injury and altered capacities, which was followed by a gradual shift toward acceptance. She did return to school about 1 year later on a part-time basis, with accom-

modations. Prior to relocating to Florida a few years later with her parents, LG was instrumental in assisting several other clients with reduced insight in benefiting from her experience.

As illustrated above, lack of awareness can negatively interfere with the recovery process. Not only can it waste a significant amount of clinical time, but it can also engender conflict between the survivor, family, and therapy team. The sooner the impaired awareness is addressed, the better the ultimate outcome, as a study by Evans, Sherer, Nick, Nakase-Richardson, and Yablon (2005) found. According to this study, impaired self-awareness has an early, negative effect on prognosis, warranting early intervention.

Awareness is a high-level integrative activity involving the integration of thoughts and feelings. When an individual has an impaired monitor, there is a defect in the feedback system preventing proper integration of information and monitoring of responses. Functional magnetic resonance and tensor diffusion images have shown the brain areas involved in awareness of deficit as well as the bundles of fibers that connect these regions. Activity within the frontoparietal control network seems to be altered in these clients, with decreased resting state connectivity found between the dorsal anterior cingulate cortex, a key component of the network mainly responsible for monitoring, and the rest of the frontoparietal control network (Ham et al., 2014). Studies have shown that decreased awareness is associated with lack of compliance with rehabilitation, greater caregiver distress, decreased functional status at discharge, and reduced employment outcome (Sherer, 2005). Realistic self-appraisal is critical for positive therapeutic outcomes.

One effective way to build insight is through bombardment of the individual with feedback from many different sources, inclusive of significant others, therapists, and peers. Feedback from other survivors who overcame insight challenges can be very powerful in leading unaware survivors to a breakthrough in insight. Allowing survivors to view videotaped segments of their behavior can sometimes help improve awareness. Of course, it is clinically important to balance confrontation with support (Cicerone, 1989).

Educating survivors regarding awareness difficulties can also be an effective insight-building tool. The survivor and primary team members can devise a list of current strengths and goal areas, to be reviewed and reinforced daily. Another way to improve insight is through performance feedback. Asking survivors to predict their performance on tasks and providing them with feedback regarding their actual performance can help improve awareness (Youngjohn & Altman, 1989). Villalobos, Bilbao, Espejo, and Garcia-Pacios (2018) provided evidence in favor of using a structured intervention program to build AD as part of the rehabilitation process, highlighting the importance of a biopsychosocial approach including psychoeducation, use of client specific clinical data, and feedback from peers and clinicians regarding the impact of challenges on functional activities and the importance of adaptation.

As a final resort, some survivors, like LG, may need failure experiences in order to build insight. Another example was a legally blind client who believed firmly that he would be able to drive safely if he was put behind the wheel. He needed to take

and fail numerous driving evaluations in order to be convinced that his vision precluded him from safe driving. Allowing clients to fail as a means of building insight is a last resort and should always involve protective mechanisms to assure safety. Once a survivor becomes aware of his/her difficulties post-ABI, there is often a concomitant decline in mood (Sohlberg, Mateer, Penkman, Glang, & Todis, 1998).

Mood Challenges

Treatment of post-traumatic depression (PTD) is complex due to the organic and psychosocial factors involved. Juengst, Kumar, and Wanger (2017) report the prevalence of PTD to be 30%, with 50% of individuals with moderate to severe TBI experiencing an episode of PTD in the first year after injury. They highlight the consequences on recovery "leading to more hospitalizations and greater caregiver burden, and reducing rates of return to work and affecting social relationships, and quality of life".

Lack of uniformity in defining depression has resulted in much variability in its reported frequency post-ABI. Anxiety disorders, emotional lability, aggressive behavior, and substance abuse challenges are frequently associated with major depression post-TBI, and their copresence is a marker for negative cognitive and psychosocial outcomes (Jorge & Starkstein, 2005). The high prevalence of depression that we see clinically and the negative impact on recovery, as well as the association with suicidal ideation (Tsaousides, Cantor, & Gordon, 2011), highlight the need to come up with effective interventions. Please see Scicutella's (Chap. 10) discussion of risk factors linked to suicidal behavior after traumatic brain injury. Clinically, untreated depression presents a barrier to progress due to reduced participation and interest in therapy sessions and home activities. Lewis and Horn (2017) found that reducing symptoms of depression in clients with TBI was significantly linked to gains in cognition, communication, physical and social abilities.

There is evidence of a biphasic course in the prevalence of poststroke depression, with one peak occurring within the first year of the stroke and the second occurring during the second year. Depressed patients are often less motivated to take part in rehabilitation, have longer hospital stays, lower functional outcome, and decreased resumption of social activities following discharge from rehabilitation (Van de Meent, Geurts, & Limbeck, 2003). Ferro, Caeiro, and Figueira (2016) report that treatment and management strategies such as the administration of antidepressants and reinforcement of good coping skills have beneficial effects on stroke-associated depression, whereas anxiety and poor coping strategies have an unsurprisingly negative influence on the course of depression and are linked to unfavorable outcomes. They also report that about one-third of stroke survivors experience depression, anxiety, or apathy, which are the most common neuropsychiatric sequelae of stroke.

Neurobiological and psychosocial factors lead to a unique presentation of mood challenges in each client. Alderfer, Arciniegas, and Silver (2005) cite laterality of injury, dysfunction in dorsal frontal systems, and dysregulation of serotonergic

activity as primary neuroanatomic factors affecting mood. They also discuss various psychosocial risk factors for post-TBI depression inclusive of poor preinjury occupational status, poor premorbid social functioning, previous history of psychiatric diagnosis or alcohol abuse history, fewer years of formal education, and female gender. They report that the rate of depression is high in the first-year postinjury, although clients are at increased risk for developing depression for many decades following their injury. These researchers suggest that biological factors have an increased role in acute-onset depression, with psychosocial factors having a more significant role in delayed onset depression.

The evaluation process for all survivors with ABI should include a clinical screening for depression, with recommendations made for individual and/or group counseling in addition to neuropsychiatric consultation, as needed. This is particularly important because a delay in treatment can negatively affect emotional and cognitive gains. Neuropsychiatric consultation is always indicated in cases of suicidal or aggressive ideation and in situations where nonpharmacological treatment has been insufficient. Scicutella (Chap. 10) discusses the differential diagnosis of apathy and pseudobular affect from mood disorders.

Even subtle changes in thinking or feeling can lead survivors to feel altered. Many express sadness and frustration due to loss of certain skills or functional abilities. They frequently describe their injury as a major marker in their lives, dividing their experiences into preinjury and postinjury categories. Survivors who were high achievers prior to their injury tend to be especially frustrated by their inability to duplicate preinjury roles. Those individuals who are able to return to work but at the cost of expending significantly more effort to achieve the same result often feel deeply saddened by this loss of efficiency. Loss of status can also occur due to an altered role at work or in the family.

Survivors frequently report decreased mood due to feeling out of control. Uncertainty about the future is particularly difficult to deal with, especially for those that were very control-oriented in the past. The enormous setback that can result from severe injuries can require many years of hard work and consistent use of compensatory strategies for success.

ABI can create a giant strain on relationships and place marriages at risk for "relationship breakdown" (Blais & Boisvert, 2005). Survivors who need to deal with divorce and separation from their children, in addition to their ABI, are at very high risk for depression.

Survivors with reduced mood post-ABI benefit most from emotional support, guidance with goal direction, and overall empowerment in their daily lives. They frequently describe the significant emotional boost they derive from success, such as doing well in a course or making active progress in therapy sessions. It is also important for the clinician to keep in regular contact with the survivor's significant others to monitor mood at home and in the community. Cognitive-behavioral therapy (CBT) approaches (Beck, 1995) were designed to treat depression and anxiety in individuals without cognitive challenges. CBT has been adapted for use with individuals poststroke (Hibbard, Grober, Gordan, Aletta, & Freeman, 1990).

Tiersky et al. (2005) demonstrated that programming consisting of CBT and cognitive remediation showed promise in the treatment of depression and anxiety in individuals with mild to moderate TBI living in the community. Replacing cognitive distortions, such as, "I had a brain injury and am totally useless," with more accurate and adaptive interpretations has been found clinically useful in survivors of ABI. Mateer, Sira, and O'Connell (2005) highlighted the importance of integrating cognitive and emotional interventions in the treatment of individuals with ABI. Ashman, Cantor, Tsaosides, Spielman, and Gordon (2014) found that both CBT and supportive psychotherapy (SPT) were effective in reducing symptoms of depression following TBI. Clinically, CBT has been found more beneficial in clients with cognitive distortions and negative core beliefs. SPT is helpful with clients who are distressed by the losses postinjury but still retain constructive core beliefs about themselves and the world.

Bombardier et al. (2017) suggest that behavioral activation and improving engagement in meaningful activities can help boost mood in clients with TBI. Jones, Pryor, Care-Unger, and Simpson (2018) discuss the role of positive coping skills, resilience, and particularly spirituality in the adjustment process following an ABI. Dialectical behavior therapy (DBT) is an evidence-based cognitive behavioral treatment developed by Linehan (2015a, 2015b) that uses learned skills and strategies to help individuals build mindfulness, interpersonal effectiveness, emotional regulation, and distress tolerance. DBT has proven effective in treating personality disorders, chronic depression, anxiety disorders, eating disorders, and addictions. Some skills and strategies can be applied successfully in the clinical setting when working with clients with ABI.

Prigatano (1999) emphasized the need for a set of guiding principles in psychotherapeutic work with ABI survivors. These include working within the survivor's subjective experience, addressing disorders of awareness, considering preinjury characteristics, as well as recognizing the interaction between cognition and personality. He advised clinicians to "focus on the present but with a sophisticated understanding of how the past may have contributed to patients' behaviors."

Couples counseling is often indicated post-ABI. Primary goals include assistance with adjustment to changes and redevelopment of trust, communication, and intimacy, as well as reinforcement of empathy, flexibility, mutual support, and respect.

Group counseling, in the form of an educational/support group, can provide clients with a sense of connection to others who have gone through similar experiences. Observing progress in peers can provide a boost in level of client hopefulness. Group counseling can also help improve social interactional skills and act as a forum for mastery of strategies to regulate emotions. Survivors report much benefit from group discussions regarding adaptive ways to cope with depression and frustration. Alumni can serve as role models and mentors for survivors receiving active rehabilitation, as they can provide hope and encouragement based on their postinjury successes.

Frustration, Anger, and Behavioral Challenges

Aggressive behaviors are considered common among brain injury survivors. Baguley, Cooper, and Felmingham (2006) report that in the acute rehabilitation setting, aggression is associated with factors such as reduced communication skills, symptoms of post-traumatic stress disorder, frontal lobe injury, disorientation to place and time, and premorbid psychiatric and substance abuse history. They investigated the prevalence and predictors of aggressive behavior among clients with TBI up to 60 months postdischarge. Their primary findings were that both "depression" and a "younger age at time of injury" were the most significant predictors of aggression at 6, 24, and 60 months postdischarge, and that prevalence of aggression was at 25% following TBI at these different follow-up periods.

Survivors frequently report reduced frustration tolerance post-ABI, with overreaction to minor triggers. This represents a decrease in tolerance for levels of stimulation that were tolerated effectively in the past. As preinjury characteristics are often amplified postinjury, it isn't surprising that those who were somewhat irritable before their injury may become significantly more so following their injury. Greatest sources of reported frustration are in relation to functional loss and restriction of autonomy.

Survivors frequently experience much anger at the source of the injury. Self-anger is noted in cases where the survivor was in some way responsible for his or her injury, such as due to a faulty suicide attempt, drug overdose, impulsivity, or negligent behavior. In cases where someone else caused the injury, the survivor's relationship to that person and the interpretation of events that led to the injury will influence the reaction. For example, a roommate who assaults an individual on the head numerous times with a flashlight will effect a different emotional reaction than a stranger who collides into a pedestrian due to a sudden heart attack behind the wheel. If the individual was a passenger in a car driven by a friend or family member who was driving recklessly, his or her reaction will be different than if the friend or family member was also a victim to a second driver who was intoxicated. In cases where a survivor experiences much anger related to the surrounding events of the injury, it may take several months or years to work through the anger and move forward. Although anger and self-pity are normal reactions to an ABI, when these emotions persist they can become toxic to the recovery process. Individual and group counseling can be very helpful in allowing the survivor an opportunity to ventilate anger and obtain feedback and support from others.

Reinforcement of adaptive coping strategies is critical in improving anger control and compensating for disinhibition. Impaired regulation of mood and behavior is a serious barrier to community reintegration. Survivors often need to relearn how to slow down and self-monitor during challenging interactions to prevent inappropriate outbursts or aggressive responses. It is helpful to reinforce the first-letter mnemonic COP to remind survivors of items to remember in their attempts at anger control. The *C* stands for communication of thoughts and feelings in a nonaggressive, constructive manner. Survivors are taught to try and remove themselves from

situations where they feel they cannot communicate in an appropriate, nonhurtful manner. The *O* stands for constructive outlets used to help the individual handle frustration effectively, such as listening to music or relaxation tapes, physical exercise, or journaling. The *P* stands for preparation for dealing with triggers that evoke aggressive responses. In this way, the client can learn to both prevent escalation when triggered and to deescalate challenging situations that arise.

Medd and Tate (2000) studied the effectiveness of cognitive-behaviorally oriented anger management programs involving self-awareness and self-regulation training. Participants were trained to recognize and respond more effectively to their reactions to anger inducing situations. Results showed a significant decrease in the outward expression of anger by the treatment group, suggesting improved emotional self-regulation. Hart et al. (2017) assess the efficacy of an 8 week Anger Self-Management Training (ASMT) program in clients with moderate to severe TBI that focuses on improving self-awareness, self-monitoring, and problem-solving skills. Their study demonstrated that ASMT was effective for certain aspects of anger challenges post-TBI.

Giles and Manchester (2006) discuss the value of both the operant neurobehavioral approach (ONA) and the relational neurobehavioral approach (RNA) to behavioral difficulties post-TBI. Both approaches focus on reducing disruptive behavior and reinforcing adaptive behavior. ONA involves teaching survivors to adapt to social norms within a structured environment with clear contingencies. The goal is to strengthen desirable behavior and extinguish undesirable behavior. Staff feedback is direct and authoritative. RNA targets the therapeutic relationship as a treatment variable, with the focus on promoting client motivation.

In cases where survivors are having difficulty controlling their behavior in therapy sessions, a behavioral rating scale can be very useful. The therapy team identifies the key areas interfering with the survivor's progress and those items are assessed on an hourly basis. At the end of each session, the treating therapist fills out the rating scale and gives feedback to the survivor, using a scale from 1 to 4, ranging from poor, fair, good to excellent. Common categories are "cooperation level," "ability to focus on presented tasks," "awareness of social boundaries," and "promptness." In this way, survivors can receive regular quantitative feedback on their behavioral gains and can work toward a meaningful reward if they are successful.

Claudia Osborn (1998), a physician who sustained an ABI due to a motor vehicle accident, emphasizes the importance of acceptance in the recovery process. In her chapter entitled, "Not as I wish, but as I am," she discusses the critical steps of awareness, compensation, and ultimately acceptance "that some things about us cannot be restored." Once survivors reach some level of peace with the injury, their anger level usually significantly decreases, enabling increased productivity in their daily lives.

Anxiety and Stress Management

Anxiety is very common post-TBI and has been reported at rates as high as 70%, with 29% prevalence across all severity types of TBI. The most common symptoms of anxiety post-TBI are free-floating anxiety, fearfulness, intense worry, social

withdrawal, and interpersonal sensitivity. Anxiety and depression have a high comorbidity rate (Moore, Terryberry-Spohr, & Hope, 2006). Please see Scicutella's (Chap. 10) review of the new subcategories of anxiety in the DSM-5 including panic disorder, generalized anxiety disorder and social phobia as well as the separate sections on obsessive compulsive disorder as well as posttraumatic stress disorder.

Brain injury survivors can experience an overwhelming amount of stress due to role changes and altered functional status. The life-threatening experiences they have undergone can create feelings of vulnerability and weakness. Moore et al. (2006) state that TBI is thought to break down psychological defenses and coping strategies, leaving the individual more vulnerable to previously experienced anxiety conditions.

A combination of strategies such as positive self-talk, breathing exercises, cognitive behavioral therapy techniques, imagery, as well as the use of constructive outlets such as music and physical exercise, can assist survivors in managing stress effectively.

Issues of Self-Concept

It is very common for survivors to report a reduction in self-confidence post-ABI. Most cited reasons include lessened mental acuity, a feeling of "brokenness," loss of autonomy, and decreased productivity in daily life. Additionally, survivors report reduced control over life decisions, the need to revise goals and expectations, as well as feeling devalued or stigmatized by others.

Survivors often need encouragement to avoid magnifying their errors and being overly critical of their weaknesses during the recovery process. Survivors report that gains in confidence are primarily linked to success in real-life activities and to the support and caring of significant others. Garske and Thomas (1992) found that ratings of self-esteem were most strongly related to satisfaction with family interactions, level of social contact, and positive emotional status.

Vickery, Gontkovsky, Wallace, and Caroselli (2006) examined the impact of group therapy interventions on self-concept in brain injury survivors. They discussed past research that has shown how ratings of self-concept can improve following social skills training and participation in a physical conditioning exercise program and various recreational activities. Their research explored the effectiveness of a group intervention that focused on self-concept changes following ABI by expanding knowledge of the self and reinforcement of positive self-attributes, based on the concepts of self-complexity and importance differentiation. Self-complexity involves the recognition that there are many different aspects to the self and that a person need not be defined in narrow terms. Importance differentiation is the process of recognizing that certain aspects of the self may be more valuable than others. The authors were interested in seeing if challenging the importance of affected areas of self-concept could be curative. Their research evidenced that group members showed a significant increase in self-concept ratings at the end of the group intervention.

Tomberg, Toomela, Pulver, and Tikk (2005) studied coping strategies and social support on quality of life post-TBI and found that individuals with TBI used task-oriented and emotional/social support strategies significantly less than control subjects and avoidance-oriented strategies significantly more than controls. They concluded that enhancing a survivor's well-being involves improving the quality and amount of the social support network. Anson and Ponsford (2006) found that coping strategies characterized as active, interpersonal, and problem-focused are associated with higher self-esteem following TBI.

Counseling sessions to boost self-confidence include training in assertiveness, increasing awareness of strengths, providing praise and positive feedback, and assisting in the process of reintegration to meaningful roles. Pegg et al. (2005) studied the impact of person-centered information on patients' treatment satisfaction and outcomes post-TBI rehabilitation. They found that survivors with moderate to severe injuries who were given more personalized information about their treatment exerted greater effort in therapy sessions, made greater improvement in functional independence, and were more satisfied with the treatment. They concluded that moderately to severely impaired survivors can benefit from interventions designed to enhance their sense of control and empowerment over their care.

Reintegration to Meaningful Roles

ABI sequelae present a daunting challenge to survivors who are trying to reestablish their sense of work and personal identity. Discharge planning for all clients needs to involve reintegration to productive activities. Primary discharge options include return to work, school, or vocational training, volunteer activities, active retirement, or structured day programming.

If a survivor's recovery allows for a return to a former position, appropriate timing and gradual reintegration are both critical to the success of the reentry. It is best to have a survivor start going back to work on a gradual basis, beginning with 1–2 days a week, and gradually increasing days and hours based on performance. It is important to coordinate efforts with the survivor's work supervisor, requesting reasonable accommodations as needed, and sharing best ways to facilitate the reintegration process. Supportive work supervisors are sometimes able to offer a survivor a modified position, either temporarily or permanently, to further aid the transition from rehabilitation to work.

As ABIs occur to individuals from every possible profession, the neurorehabilitation team has the challenge of assisting clients in returning to a wide variety of careers. These professions include various fields in medicine, law, business, and accounting as well as police work, maintenance, office work, and homemaking. Clients who were unemployed preinjury pose a different type of challenge, many having preinjury vocational issues which need to be addressed along with the postinjury challenges.

There are six primary challenges noted clinically that hinder return to work post-ABI: lack of readiness, suitability, support, stability, insight, and incentives. Lack of readiness refers to inappropriate timing. Survivors who return to work prematurely are at high risk for failure. Lack of suitability refers to the fact that a survivor's pre-injury work may no longer match his or her postinjury level of functioning. Clinical examples include a cab driver that became legally blind, a construction worker with balance problems, a teacher with aphasia who no longer can communicate functionally, or an accountant who struggles to perform basic calculations. Loss of work identity presents a radical challenge to an individual's self-definition. Once the survivor is emotionally ready to accept a new role, he or she may choose vocational retraining, meaningful volunteer work, or a more active role at home.

Lack of support refers to an employer's creation of barriers to a survivor's reintegration to work. Clinical experience has shown that many supervisors have difficulty accepting an employee's postinjury changes, such as reduced speed on tasks, the need for compensatory strategies to aid memory and learning, behavioral differences, reduced attention to grooming or hygiene, and difficulty understanding humor. These variations may be quite subtle and even within normal limits but are less acceptable when compared to preinjury performance standards. Despite protection provided by the Americans with Disabilities Act (ADA) of 1990, an unsupportive employer can sabotage a survivor's successful reentry. Providing education and counsel to supervisors can be very helpful in developing empathy and understanding. Lack of emotional or medical stability will prevent a successful return to work for numerous reasons. Challenges with alcohol or substance abuse have also been linked to failure in community reintegration (Dell Orto & Power, 2000). Lack of insight challenges were reviewed earlier in this chapter. Survivors with awareness deficits believe they can return to work prior to their actual readiness, and often return prematurely, resulting in failure. There can be various disincentives to return to work which surface during the rehabilitation period. These include financial (e.g., potential to jeopardize social security/disability benefits), legal (e.g., pending lawsuit), and emotional/psychosocial disincentives (e.g., adoption of the "sick" role).

In order to optimize a survivor's reintegration to work, it is strongly recommended that the appropriate steps be followed. First, it is important to integrate vocational rehabilitation goals with general interdisciplinary therapy goals while the survivor is in the neurorehabilitation program. Simulating work responsibilities is critical in assessing survivor readiness. Once the client is doing consistently well in the neurorehabilitation program and is successful on all simulated tasks or work trials, the time is appropriate to coordinate with the individual's work supervisor to begin discussion regarding a gradual return to work and any needed reasonable accommodations. The work supervisor will need input on the survivor's current strengths and weaknesses and the most effective ways to best facilitate his or her return to work.

In cases where survivors are no longer well suited for their former careers, various options are possible. They may choose to retrain through state or privately funded vocational agencies/programs for individuals with disabilities so that they can begin a new career based on their current strengths. They may be able to find

competitive or volunteer work in their field in a modified position. Survivors who were physical laborers prior to their injuries may choose to become caretakers for their children or homemakers for their families, especially in cases where they are not able to return to physical work and are not interested in returning to school or pursuing a different career. Additionally, given the wide range of volunteer opportunities in major hospitals, nursing homes, public schools, university settings, and libraries, many clients are able to find a suitable position that can be both fulfilling and flexible, in terms of hours/days.

In cases where survivors are not appropriately supported by their employers in their efforts to return to work, it is critical that support be provided by either a job coach (funded by state, county, or paid for privately) or by a primary therapist/counselor assisting the transition to work by maintaining regular contact and exchange of information with the employer. If the survivor is able to perform his/her essential job functions and the employer is not willing to provide reasonable accommodations, legal action may be necessary.

In terms of medical stability challenges in returning to work, it is critical that survivors have ongoing medical follow-up, especially in cases of seizure disorders or pain syndromes. The degree of control that a survivor has on medical challenges will greatly influence his/her attempts to successfully reintegrate to work. Continued counseling during the reintegration process can significantly assist the survivor in maintaining emotional stability and coping with difficulties that arise. Follow up counseling sessions can also assist survivors in improving their awareness level as well as coping with mood challenges that may emerge once insight increases. Counseling/psychotherapy can also help survivors overcome various disincentive barriers.

Survivors who are high-school students receiving neurorehabilitation can benefit when they obtain both their tutoring and therapy sessions at the same location, so that tutors and therapists can coordinate efforts. Educating tutors about the best ways to work around the student's weaknesses and to utilize strengths is very helpful. Survivors returning to high school following an ABI may benefit from a gradual shift from the neurorehabilitation program to part-time classes, with resource room as needed.

In working with a survivor returning to college or graduate school, it is best to coordinate efforts with the Office of Disability Services at the university. Reasonable accommodations most commonly recommended include extended time on tests in a private room and permission to tape lectures as well as to use the services of a note taker. Enlarging handouts and tests as well as providing tests in multiple choice format can be helpful for certain clients.

Volunteering is an excellent next step for many survivors who are not returning to school, work, or homemaking responsibilities following rehabilitation. Within many hospital settings, there are many structured opportunities ranging from filing and basic clerical work to maintenance, paper delivery, food preparation, mail room responsibilities, and gift shop work, to higher level positions, such as reading to children in pediatric wards or acting as a patient advocate. The clinical team and the

coordinator of volunteer services can try to match a position to a survivor's abilities and interests.

For survivors who are unable to return to preinjury work but are good candidates for reentry to competitive employment, coordination of efforts with a state or privately funded vocational program can sometimes lead survivors to appropriate career changes. For instance, survivors who cannot return to the physical work of construction may become trained in construction management or computer programming, or a survivor who was an emergency medical technician (EMT) can be retrained as a lab technician.

Post-ABI Growth

CD was a 33-year-old electrician receiving neurorehabilitation subsequent to a cocaine-induced brain aneurysm. During the initial meeting, CD was agitated and devoid of insight, stating that he only used cocaine and alcohol infrequently "at weddings." He was in denial regarding his drug and alcohol problem and was totally unaware of neurocognitive changes following his injury. CD's only focus was on discharge and returning to his former life. Once a positive therapeutic alliance was gradually developed and insight grew, CD became more willing to explore and reevaluate his preinjury life. He was able to see that his daily "partying" had hurt his self-esteem, career opportunities, financial status, interpersonal relationships, and finally his health and thinking abilities. The client had difficulties with attention, short-term memory, and processing speed, but was able to master compensatory strategies to work around his difficulties effectively.

CD spoke about the positive changes he wanted to institute in his life and the fact that his injury led him to personal growth. He ultimately used his injury and subsequent neurorehabilitation experience for the purpose of post-ABI growth. He began his own business, started a family, worked around his difficulties by consistent use of strategies, and stayed away from substances.

The concept of post-ABI growth developed as a result of working with hundreds of survivors like CD who demonstrated constructive life changes following an ABI. Post-ABI growth refers to any positive byproduct resulting from an ABI, such as an improvement in sense of self, an increase in appreciation of friends and family, a termination of destructive habits, or introduction to new vocational or avocational pathways.

Substance Abuse Challenges

Coping with both ABI and substance abuse challenges results in "a multidimensional disability and presents a unique set of problems related to dual diagnosis." Alcohol is involved in the acquisition of 36–73% of all TBIs (Beaulieu-Bonneau

et al., 2018). They note that there is an early decrease in substance use after a TBI, with substance use tending to gradually increase after the first year following the injury. Their study revealed that younger age, not being in a relationship, and intoxication at the time of the TBI were associated with the presence of a post-TBI substance use disorder. Once an individual has sustained an ABI, continued alcohol abuse is linked to increased likelihood of seizures, poor impulse control, and heightened cognitive deficits. Clinically, we see a majority of individuals who abused different substances leading to their ABIs become clean and sober following a moderate to severe injury. The lengthy hospitalization and rehabilitation process coupled with postinjury challenges often provide clients with the awareness and opportunity to examine their preinjury habits and the serious impact on their physical, cognitive, and emotional functioning. Counseling aimed at assisting clients in understanding the reasons and repercussions of the abuse, and ways to prevent relapse through consistent use of adaptive coping outlets and constructive communication with others are critical.

Some individuals may be predisposed to sustaining a TBI due to self-destructive behaviors as a result of depression or substance abuse. The higher the number of preinjury vulnerabilities (such as a history of depression or substance abuse, lack of family support, or limited education), the more challenging the recovery process with a more guarded prognosis. Individuals with complex histories will require the collaboration of specialists in neuropsychiatry and substance abuse treatment to increase the likelihood of a favorable recovery.

Screening for alcohol and substance abuse during a survivor's initial intake to a neurorehabilitation program is critical. Findings suggest that CAGE (Cut down, Annoy, Guilty, and Eye Opener) may be a useful screening tool for alcohol abuse and that the Substance Abuse Subtle Screening Inventory (SASSI-3) may be useful for assessing drug abuse in individuals with TBI (Ashman, Schwartz, Cantor, Hibbard, & Gordon, 2004). CAGE is a four question-screening tool that addresses the drinking experience of the individual. Two or more positive responses are viewed as an indication of an alcohol problem (Ewing, 1984). Following screening, case management appears to have a beneficial effect on survivors with both TBI and substance abuse challenges (Heinemann, Corrigan, & Moore, 2004).

Conclusion

The experience of counseling individuals post-ABI is always dynamic and demanding. The beginning challenges involve creating a favorable rapport, developing trust, establishing an environment where the survivor feels comfortable sharing personal issues, and assisting in empowerment of the individual in the process of recovery. Later challenges may involve effecting confrontation without alienation, keeping a goal-oriented approach, weaving significant others actively into the process, reinforcing strategies to improve mood, and frustration tolerance, as well as ensuring that the survivor is on a productive path emotionally and socially. The final

objectives are to keep the individual hopeful about the future despite awareness of residual weaknesses and to assist in the process of transitioning successfully to a next step that involves continued structure and stimulation, such as work, school, or a volunteer role.

The counselor/psychotherapist is oftentimes the survivor's primary partner in the neurorehabilitation process, due to the clinician's awareness of the individual's fears, motivations, vulnerabilities, and triggers. It is highly important to integrate efforts with all other team members to assure that emotional and behavioral difficulties are not interfering with the survivor's progress in any domain. This type of close team communication and collaboration enables the survivor to receive consistent feedback from different team members and also highlights the fact that the team is working in concert, guiding the survivor toward goal achievement and reintegration to meaningful roles.

References

Alderfer, B., Arciniegas, D., & Silver, J. (2005). Treatment of depression following traumatic brain injury. *The Journal of Head Trauma Rehabilitation, 20*(6), 544–562.

Anson, K., & Ponsford, J. (2006). Evaluation of a coping skills group following traumatic brain injury. *Brain Injury, 20*(2), 167–178.

Ashman, T., Cantor, J. B., Tsaosides, T., Spielman, L., & Gordon, W. (2014). Comparison of cognitive behavioral therapy and supportive psychotherapy for the treatment of depression following traumatic brain injury: A randomized controlled trial. *The Journal of Head Trauma Rehabilitation, 29*(6), 467–478.

Ashman, T. A., Schwartz, M. E., Cantor, J. B., Hibbard, M. R., & Gordon, W. A. (2004). Screening for substance abuse in individuals with traumatic brain injury. *Brain Injury, 18*(2), 191–202.

Baguley, I. J., Cooper, J., & Felmingham, K. (2006). Aggressive behavior following traumatic brain injury. *The Journal of Head Trauma Rehabilitation, 21*(1), 45–56.

Beaulieu-Bonneau, S., St-Onge, F., Blackburn, M. C., Banville, A., Paradis-Giroux, A. A., & Ouellet, M. C. (2018). Alcohol and drug use before and during the first year after Traumatic Brain Injury. *The Journal of Head Trauma Rehabilitation, 33*(3), E51–E60.

Beck, J. S. (1995). *Cognitive therapy: Basics and beyond*. New York, NY: Guilford Press.

Blais, M., & Boisvert, J. (2005). Psychological and marital adjustment in couples following a traumatic brain injury: A critical review. *Brain Injury, 19*(14), 1223–1235.

Bombardier, C. H., Fann, J. R., Ludman, E. J., Vannoy, S. D., Dyer, J. R., Barber, J. K., & Tempkin, N. R. (2017). The relation of cognitive, behavioral, and physical activity variables to depression severity in TBI: Reanalysis of data from a randomized controlled trial. *The Journal of Head Trauma Rehabilitation, 32*(5), 343–353.

Cicerone, K. D. (1989). Psychotherapy interventions with traumatic brain injury patients. *Rehabilitation Psychology, 34*, 105–114.

Crosson, B., Barco, P. P., Velozo, C. A., Bolesta, M. M., Cooper, P. V., Werts, D., & Brobeck, T. C. (1989). Awareness and compensation in post-acute head injury rehabilitation. *The Journal of Head Trauma Rehabilitation, 4*(3), 46–54.

Dell Orto, A. E., & Power, P. W. (2000). *Brain injury and the family*. New York, NY: CRC Press.

Evans, C., Sherer, M., Nick, T., Nakase-Richardson, R., & Yablon, S. (2005). Early impaired self-awareness, depression, and subjective well-being following traumatic brain injury. *The Journal of Head Trauma Rehabilitation, 20*(6), 488–500.

Ewing, J. A. (1984). Detecting alcoholism: The CAGE questionnaire. *Journal of the American Medical Association, 252*, 1905–1907.

Ferro, J. M., Caeiro, L., & Figueira, M. L. (2016). Neuropsychiatric sequelae of stroke. *Nature Reviews Neurology, 12*, 269–280.

Garske, G. G., & Thomas, K. R. (1992). Self-reported self-esteem and depression: Indexes of psychosocial adjustment following severe traumatic brain injury. *Rehabilitation Counseling Bulletin, 36*, 44–52.

Giles, G. M., & Manchester, D. (2006). Two approaches to behavior disorders after traumatic brain injury. *The Journal of Head Trauma Rehabilitation, 21*(2), 168–178.

Gray, J. A., & McNaughton, N. (1996). *The neuropsychology of anxiety*. Nebraska: University of Nebraska Press.

Ham, T. E., Bonnelle, V., Hellyer, P., Jilka, S., Robertson, I. H., Leech, R., & Sharp, D. J. (2014). The neural basis of impaired self-awareness after traumatic brain injury. *Brain, 137*(10), 586–597.

Hart, T., Brockway, J., Maiuro, R., Vaccaro, M., Fann, J. R., Mellick, D., … Tempkin, N. (2017). Anger self-management training for chronic moderate to severe traumatic brain injury: Results of a randomized controlled trial. *The Journal of Head Trauma Rehabilitation, 32*(5), 319–331.

Hart, T., & Sherer, M. (2005). Disorders of self-awareness. *The Journal of Head Trauma Rehabilitation, 20*(4), 287–367.

Heinemann, A. W., Corrigan, J. D., & Moore, D. (2004). Case management for traumatic brain injury survivors with alcohol problems. *Rehabilitation Psychology, 49*(2), 156–166.

Hibbard, M. R., Grober, S. E., Gordan, W. A., Aletta, E. G., & Freeman, A. (1990). Cognitive therapy and the treatment of post-stroke depression. *Topics in Geriatric Rehabilitation, 5*, 43–55.

Jones, K. F., Pryor, J., Care-Unger, C., & Simpson, G. K. (2018). Spirituality and its relationship with positive adjustment following traumatic brain injury: A scoping review. *Brain Injury, 32*(13-14), 1612–1622.

Jorge, R. E., & Starkstein, S. E. (2005). Pathophysiologic aspects of major depression following traumatic brain injury. *The Journal of Head Trauma Rehabilitation, 20*(6), 475–486.

Juengst, S. B., Kumar, R. G., & Wanger, A. K. (2017). A narrative literature review of depression following traumatic brain injury: Prevalence, impact and management challenges. *Psychology Research and Behavior Management, 10*, 175–186.

Kortte, K. B., Wegener, S. T., & Chwalisz, K. (2003). Anosognosia and denial: Their relationship to coping and depression in acquired brain injury. *Rehabilitation Psychology, 48*(3), 131–136.

Leung, D. P. K., & Liu, K. P. Y. (2011). Review of self-awareness and its clinical application in stroke rehabilitation. *International Journal of Rehabilitation Research, 34*, 187–195.

Lewis, F. D., & Horn, G. J. (2017). Depression following traumatic brain injury: Impact on post-hospital residential rehabilitation outcomes. *NeuroRehabilitation, 40*(3), 401–410.

Linehan, M. M. (2015a). *DBT skills training manual* (2nd ed.). New York, NY: The Guilford Press.

Linehan, M. M. (2015b). *DBT skills training: Handouts and worksheets* (2nd ed.). New York, NY: The Guilford Press.

Mateer, C. A., Sira, C. S., & O'Connell, M. E. (2005). Putting humpty dumpty back together again: The importance of integrating cognitive and emotional interventions. *The Journal of Head Trauma Rehabilitation, 20*(1), 62–75.

Medd, J., & Tate, R. L. (2000). Evaluation of an anger management therapy program following acquired brain injury: A preliminary study. *Neuropsychological Rehabilitation, 10*(2), 185–201.

Moore, E. L., Terryberry-Spohr, L., & Hope, D. A. (2006). Mild traumatic brain injury and anxiety sequelae: A review of the literature. *Brain Injury, 20*(2), 117–132.

Osborn, C. L. (1998). *Over my head*. Kansas City, MO: Andrews McMeel Publishing.

Pegg, P. O., Auerbach, S. M., Seel, R. T., Buenaver, L. F., Kiesler, D. J., & Plybon, L. E. (2005). The impact of patient-centered information on patients' treatment satisfaction and outcomes in traumatic brain injury rehabilitation. *Rehabilitation Psychology, 50*(4), 366–374.

Prigatano, G. P. (1999). *Principles of neuropsychological rehabilitation*. New York, NY: Oxford University Press.

Prigatano, G. P., & Schacter, D. L. (Eds.). (1991). *Awareness of deficits after brain injury*. New York, NY: Oxford University Press.

Sherer, M. (2005). Rehabilitation of impaired awareness. In W. M. High, A. M. Sander, M. A. Struchen, & K. A. Hart (Eds.), *Rehabilitation for traumatic brain injury* (pp. 31–46). New York, NY: Oxford University Press.

Sohlberg, M. M., Mateer, C. A., Penkman, L., Glang, A., & Todis, B. (1998). Awareness intervention: Who needs it? *The Journal of Head Trauma Rehabilitation, 13*(5), 62–78.

Tiersky, L. A., Anselmi, V., Johnston, M. V., Kurtyka, J., Roosen, E., Schwartz, T., & DeLuca, J. (2005). A trial of neuropsychologic rehabilitation in mild-spectrum traumatic brain injury. *Archives of Physical Medicine and Rehabilitation, 86*, 1565–1574.

Toglia, J., & Kirk, U. (2000). Understanding awareness deficits following brain injury. *NeuroRehabilitation, 15*, 57–70.

Tomberg, T., Toomela, A., Pulver, A., & Tikk, A. (2005). Coping strategies, social support, life orientation and health related quality of life following traumatic brain injury. *Brain Injury, 19*(14), 1181–1190.

Tsaousides, T., Cantor, J. B., & Gordon, W. A. (2011). Suicidal ideation following traumatic brain injury: Prevalence rates and correlates in adults living in the community. *The Journal of Head Trauma Rehabilitation, 26*(4), 265–275.

Van de Meent, H., Geurts, A. C. H., & Limbeck, J. V. (2003). Pharacologic treatment of post-stroke depression: A systematic review of the literature. *Topics in Stroke Rehabilitation, 10*(1), 79–92.

Vickery, C. D., Gontkovsky, S. T., Wallace, J. J., & Caroselli, J. S. (2006). Group psychotherapy focusing on self-concept change following acquired brain injury: A pilot investigation. *Rehabilitation Psychology, 51*(1), 30–35.

Villalobos, D., Bilbao, A., Espejo, A., & Garcia-Pacios, J. (2018). Efficacy of an intervention programme for rehabilitation of awareness of deficit after acquired brain injury: A pilot study. *Brain Injury, 32*(2), 158–166.

Youngjohn, J. F., & Altman, I. M. (1989). A performance-based group approach to the treatment of anosognosia and denial. *Rehabilitation Psychology, 34*, 217–222.

Chapter 13
Acquired Brain Injury and the Family: Challenges and Interventions

Jean Elbaum

Introduction

In clinical practice, the neurorehabilitation team encounters families that have experienced trauma, turmoil, and significant losses. Injuries are generally ill-timed and families are frequently ill-prepared for the length, uncertainty, and challenges of the post-injury process. In the literature, much has been written about "caregiver burden" and the stressful impact an acquired brain injury (ABI) can have on family systems. There is increasing awareness of the importance of identifying family needs and establishing effective and comprehensive interventions.

In some ways, families may actually experience more distress than the actual survivors of ABI. Crimmins (2000) described how accidents divide life into "the great Before and After." Survivors tend to have amnesia for the very time period that was most traumatic, whereas loved ones are clearly able to remember those unfortunate events. In the early months post-ABI, the entire family system tends to focus, almost exclusively, on the needs of the person who was injured. The amount of distress that a particular significant other will experience depends on a variety of variables, such as the quality of the relationship prior to the injury, the specific commitment to the injured loved one, the amount of time and extra responsibilities involved, other simultaneous life stressors, and the adaptiveness of coping skills. Each family subsystem confronts unique challenges, and each unit requires knowledge, partnership, and understanding from the team. In the best situations, the survivor has good awareness, is hardworking, has a constructive attitude, makes very active gains, and can reintegrate to productive roles at home and in society. In the most challenging situations, the survivor has poor awareness, severe impairments, requires long-term care, and will not be able to integrate successfully to home or community settings.

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Dell Orto and Power (2000) summarized various frustrations of primary caregivers, such as trying to balance multiple roles, feeling overwhelmed due to the lack of assistance by other significant others, feeling restricted socially, and dealing with the reality that their pre-morbid loved one may be permanently different.

Family Needs

There are commonalities in caregiver needs that we see clinically each day. Despite the type of acquired brain injury and regardless of age, gender, background, and other differences, caregivers need education, coping tools, and hope. They need to feel respected and empowered by the clinical team because their situation has led them to feel powerless and deeply concerned about how the future will unfold.

Powell et al. (2017) looked at the primary concerns of caregivers of adults with traumatic brain injury (TBI) during the initial months following discharge to home. Caregivers were most concerned about managing the client's emotional and behavioral changes as well as balancing their time properly to make sure their own needs were met. In the clinical world, families report initial concerns upon client's discharge home to be safety related, assuring that medical and rehabilitation needs are properly met, and learning to cope with role changes. Carlozzi et al. (2015) describe how caregivers have most difficulty meeting their needs for social and emotional health. Powell et al. (2017) describe how caregivers gradually return to increased participation in everyday activities over time, similar to what is seen in the literature regarding caregivers of stroke survivors (Cameron & Gignac, 2008).

Hart et al. (2018) reconceptualize moderate and severe TBI as a chronic condition and do a scoping review of the literature. They describe the variability in educational materials used across sites and lack of uniformity in training of caregivers. They highlight the importance of developing evidence-based educational interventions and recommend the use of a Self-Management Training model which has been effective for other chronic conditions (Lorig & Holman, 2003) due to inclusion of problem-solving strategies and goal setting in addition to provision of education.

Research, going back 40 years, highlighted that caregivers of individuals post-ABI are at increased risk for depression, anxiety, and physical illness (Oddy, Humphrey, & Uttley, 1978) which we still see clinically today. The Family Needs Questionnaire (FNQ; Kreutzer, Serio, & Bergquist, 1994) is a classic tool that was developed to quantify the multiple needs of family members in order to identify families at high risk and target interventions appropriately. The six categories identified were needs for health information, emotional support, instrumental support (refers to the need for respite and practical everyday assistance), professional support, community support, and involvement in care. The scale was developed based on extensive family interviewing and a comprehensive literature review (Serio, Kreutzer, & Witol, 1997). Research has shown that medical needs are perceived to be most important as well as most frequently met. In contrast, emotional needs were most often perceived as unmet (Serio, Kreutzer, & Gervasio, 1995).

Witol, Sander, and Kreutzer (1996) reported that families are generally satisfied with the information and support provided by professionals at early and late post-injury intervals. However, family members have difficulty getting their emotional and instrumental support needs met, a situation worsening over time in the case of emotional support.

Unpublished data from 2006 involving comparison of FNQ results in three different settings reflected more unmet needs in families attending a long-term stroke support group than in families of survivors in outpatient or inpatient neurorehabilitation programs. The low percentage of needs being met in families of individuals in the long-term stroke support group may be due to a selection factor, as the stroke group participants are not representative of all people post-stroke. The stroke group is a social/recreational program that meets on a weekly basis for survivors and caregivers. The families studied had been caregiving for an average of 7 years.

Serio et al. (1995) reviewed the predictors of family outcome by studying injury, patient, and family characteristics. They concluded that time since injury influences family reactions as caregivers report more unmet needs over time, consistent with our unpublished data. These authors also reported that patients' emotional and behavioral problems correlated with increased caregiver stress. Family members' perceptions of patients' problems are more important predictors of needs than test results. In terms of family characteristics, they found that spouses had more difficulty adjusting to the injury of a loved one than parents, reporting more depression, anxiety, isolation, and distress.

The Brain Injury Family Intervention (BIFI) is an evidence-based intervention that was designed to mitigate family challenges by providing education and teaching effective coping tools. Using the FNQ, significant gains were found in the areas of health information, emotional support, professional support, and involvement in care following training with the BIFI (Kreutzer, Stejskal, Ketchum, & Marwitz, 2009). Meixner, O'Donoghue, and Hart (2017) demonstrated that use of the BIFI following a 3 day training protocol boosted providers' knowledge and confidence in working with clients with TBI and their caregivers.

Kitter and Sharman (2015) describe the financial strain that caregivers deal with due to the high costs of care and high caregiver demands limiting their ability to work. Sabella, Andrzejewski, and Wallgren (2018) come up with a brief scale of financial hardship for caregivers of clients with TBI. Their goal was to identify financial hardships in family and preliminary findings indicate it may be a useful scale.

Family Coping

Families are the neurorehabilitation team's primary partners in facilitating survivor progress, and they play a major role in client recovery. Consequently, meeting caregivers' needs and reinforcing positive coping skills is critical (Serio et al., 1997).

The literature on family coping post-ABI includes many references to Kubler-Ross's (1983) stages of denial, anger, depression, and adjustment following a major loss. Each member of the family as well as the entire family unit as a whole goes through an adjustment period following the ABI of a loved one. In clinical practice, many survivors and families have rebelled against the terms "adjustment" and "acceptance" of an injury, assuming that these words denote "giving in" to the injury instead of overcoming it. In reality, these terms refer to the ability of the client and family to go forward in a productive manner despite the sequelae of the injury.

Lezak's classic article (1978) "Living with the Characterologically Altered Brain Injured Patient" describes the qualitative challenges that families face in dealing with loved ones who have behavioral difficulties following an ABI. She discusses several categories of behavioral alterations that create the greatest adjustment challenges for families: reduced empathy and self-centered behavior, reduced self-regulation, increased impulsivity and silliness, reduced executive functioning abilities, such as difficulties with initiation and planning, increased or reduced sexual interest, and difficulty with social learning. Over time, there has been more emphasis on quantitative measures of family needs and coping styles.

Nabors, Seacat, and Rosenthal (2002) described various factors that are powerful predictors of family adjustment, such as pre-injury family functioning, level of financial stress, perceived level of burden, coping mechanisms used, and availability of social support systems. They highlighted the importance of ongoing assessments of family needs.

Man (2002) identified several types of adaptive coping strategies used by families of ABI survivors, inclusive of positive appraisal, resource requisition, family tension management, and acquisition of social support.

Although there are meaningful and rewarding aspects of being a primary caregiver, at times the role can be very overwhelming, lonely, and thankless. It is not uncommon for survivors to unfairly direct a significant amount of post-injury frustration and anger toward their loved ones, by verbal attacks or disrespectful behavior. Caregivers usually have to bear the brunt of their loved one's frustration. It is important for significant others to learn how to deescalate stressful encounters and how to cope constructively with a loved one's unreasonable behavior. Without proper training to handle these challenging situations, many caregivers can turn toward destructive coping strategies.

Cameron, Cheung, Streiner, Coyte, and Stewart (2006) found that caregivers experience more symptoms of depression when they care for survivors of stroke who exhibit memory and comprehension symptoms. Their study demonstrated that 45% of caregivers reported elevated levels of symptoms of depression. They emphasized the importance of addressing the needs of caregivers by providing needed information and resources on how to handle neurocognitive difficulties.

Family Subsystems

Spouses, parents, siblings, and children are frequently the caregivers who are involved in the recovery process as primary caregivers. Sady et al. (2010) report that most caregivers of clients with TBI are parents and spouses.

Individuals who are dealing with the ABI of a marital partner are often dealing with financial, social, and personal stressors in addition to adjusting to the particular physical, cognitive, and psychosocial changes in their spouse. A spouse whose loved one has been severely injured may lose a confidante, sexual partner, household comanager, and childrearing assistant all at once (Serio et al., 1995).

Kreutzer, Gervasio, and Camplair (1994) stated that caregiving spouses reported greater family dysfunction and increased likelihood of depression compared to parents who acted as primary caregivers. Spouses of individuals post-ABI face a loss of peer-based, reciprocal relationships when they take on caregiving roles, compared to parents who return to roles familiar from child-rearing years (Wood, Liossi, & Wood, 2005). The concept of social limbo experienced by spouses after the ABI of their partner refers to the fact that spouses may not only lose the equal partnership with their loved one but they also lose the ability to maintain friendships outside of the marriage. This is the case because of lack of time or rejection by peers due to the survivor's neurobehavioral changes (Lezak, 1978). Wood et al. (2005) found that unpredictable behavior on the part of the survivor imposed the greatest burden on marital relationships and contributed to relationship breakdown.

Blais and Boisvert (2005) examined the factors associated with marital adjustment following ABI. They found that frequent use of problem-solving skills and positive reinterpretation of problems encountered, in addition to low use of avoidant coping skills were associated with higher adjustment levels.

Katz, Kravetz, and Grynbaum (2005) researched the impact of wives' "coping flexibility" and duration of time since the husband's traumatic brain injury (TBI) on perceived burden. They focused on wives in particular because their literature review demonstrated that TBI creates more difficulties for wives than for other family members. These authors defined coping flexibility as the ability to modify an ineffective coping approach and to seek a more appropriate and adaptive coping strategy. They found that only in cases of wives who had reduced coping flexibility did perceive burden increase with time since injury.

Each survivor's particular combination of neurobehavioral, cognitive, and physical difficulties in addition to the effects of medications can pose unique challenges to intimacy. Problems with focused and sustained attention can interfere with both social and physical aspects of sexual activity. Frontal lobe disinhibition can lead to socially inappropriate sexual behavior. At the other extreme are survivors who are very withdrawn or passive following their ABI and who demonstrate a very decreased libido. Functional deficits, such as communication problems, mobility loss, perceptual problems, loss of sense of smell, reduced sensation and tremor, can all contribute to intimacy challenges. As survivors with ABI may no longer have the full capacity for empathy and interpersonal sensitivity, they may no longer be able

to satisfy their partner's need for affection or intimacy (Lezak, 1978). Spouses may struggle with shifting from caregiver to sexual partner, especially in cases where the injured spouse has become very childlike.

Screening survivors and families for concerns regarding changes in intimacy should be part of the initial clinical interview, so that difficulties can be identified and addressed early on.

When parents are the primary caregivers, the role shift is different from that of a spouse. In cases where there are two parents, both can help each other assist their child. There is some familiarity in caring for a grown child as parents once did in the past, in contrast to a spouse who has always been an equal with his or her marital partner. Additionally, parents generally feel unconditional love and responsibility toward their child regardless of the level of challenge presented. In the case of a marriage partner, there is always the option of divorce. Testa, Malec, Moessner, and Brown (2006) reported that caregivers had more complaints at 2 years post-injury than at 6 months, which they speculate may underlie the high rate of divorce, up to two-thirds, at 2 years post-injury. Their study noted a strong relationship between neurobehavioral problems and impaired family functioning.

Benn and McColl (2004) stated that parents who viewed the ABI of their child as a manageable family challenge instead of a catastrophe were able to adapt more successfully. These researchers discussed the process of redefinition of stressful events to make them more meaningful. They emphasized that coping strategies that allowed parents to reframe stressors and to obtain social support helped to reduce stress.

MS sustained a severe TBI due to a fall at age 27. He had a substance abuse history and had preinjury challenges relating to self-esteem, anxiety, and depression. D, his mother, was his only significant other and primary caregiver. D was bright, highly educated, and very eager to help her son improve. She suffered post-traumatic stress disorder (PTSD), activated by the events that started when she received the frightening phone call from the police department on the night of her son's injury. Each time MS had a setback, D's PTSD was reactivated. MS's lack of insight, anger control problems, and poor attitude in addition to physical and cognitive difficulties were very upsetting to his mother, who was hopeful that the injury could bring about post-ABI growth (Chap. 12).

D attended counseling sessions aimed at reducing symptoms of post-traumatic stress disorder and teaching strategies to set limits with her son, communicate her thoughts in a constructive fashion, and improve her sense of control and use of adaptive outlets for frustration. Fortunately, MS gradually made remarkable gains in his insight, attitude, and functional status. D also recovered from the emotional trauma and was able to foster her son's continued growth and reintegration to school and work.

Parents of young children or adolescents post-ABI frequently report significant distress and fear regarding their child's altered skills and their recovery. The family unit can become more cohesive or more at risk for breakdown following a child's injury, depending on the family unit's preinjury closeness, and the amount of support and guidance they receive. Their primary challenges include making certain

that their child receives the appropriate neurorehabilitation programming and that he or she is reintegrated successfully to school at the appropriate time. Preventing social isolation and boosting self-esteem are usually important goals for children post-ABI that the clinical team can help address. Parents of young children are usually very eager to help the team by carrying over goals to the home setting and reinforcing home exercise programs. It isn't uncommon for parents to be in conflict relating to how to discipline their child with ABI, and how lenient or strict to be in rule setting and expectations. It is helpful for the clinician to train both parents in providing the same message to their child so as to increase clarity about parental expectations. Family counseling with the emphasis on parents working together as a team for the benefit of the family unit is usually very productive.

Parents of grown, married children who sustain an ABI can be very helpful to the survivor's spouse, who is usually exhausted by the demands of caregiving in addition to his or her other usual responsibilities. It is in everyone's best interest when different family members come together to best meet the needs of an injured loved one. In families where there is much tension or lack of cohesiveness, the survivor will ultimately suffer. Frequently, competition arises between the parents of the injured individual and his or her spouse regarding decision-making and treatment planning. A counselor can be very helpful in assisting different family members in working together for the survivor's benefit despite different viewpoints.

Elderly parents who become the primary caregivers of an adult child status post an ABI are in a particularly difficult situation. They suddenly have increased pressures and responsibilities instead of fewer daily activities and the enjoyment of retirement. In cases where their child will need long-term programming, parents usually gain greater peace of mind if they are able to locate a long-term residential program or supported living during their life time, thereby reassured that their loved one is being taken care of properly. It is important that elderly parents be encouraged to secure the assistance of an aide or other support services to ensure some respite.

Young children of a parent with an ABI often deal with the challenge of receiving less attention than they did prior to their parent's injury, as one parent suddenly has increased needs and the other parent consequently has less time. The child may feel an internal void due to the lack of attention and parental support. Additionally, in cases where the survivor has become childlike or attention seeking after the injury, competition may arise between the child and the injured parent for the other parent's time. Also, the child may be saddened or embarrassed by the fact that the parent is acting silly or inappropriate. Young children whose parent was injured frequently express guilt relating to somehow contributing to their parent's injury, and often require reassurance that they were not responsible. Additionally, children tend to express fear that their uninjured parent will get hurt, as they tend to feel more vulnerable following their other parent's injury. It is important to alert the child's principal, teachers, and school psychologist about the parent's injury and the need for the child to be given extra support and attention. It is also important for the child to be educated, based on his or her age level, on ABI sequelae, for better understand-

ing of the parent's injury. This type of training should be offered through the neurorehabilitation program that the parent attends.

It is very difficult for a parent who sustained an ABI to reestablish his or her status at home after a lengthy post-injury absence due to hospitalization and acute rehabilitation. Oftentimes, the household developed new norms while the survivor was away and it's very common for children to have become accustomed to having all their needs met by the uninjured parent. The injured parent usually is frustrated by his or her loss of power and may feel unimportant or excluded from the rest of the family. He or she may have particular difficulty setting limits and demonstrating good frustration tolerance. Hypersensitivity to noise and motion may also negatively affect parent–child interactions. Confidence may be very reduced as the parent post-injury is dealing with the loss of status at home in addition to other post-ABI changes.

Adult children tend to become the significant other for their parent in cases where the other parent is deceased, disabled, or not involved. As grown children usually are in the process of developing their career and busy with their own family needs, time management usually becomes exceedingly difficult. These family members will require substantial support and training from the neurorehabilitation team to facilitate positive coping.

Siblings become the primary caregiver in cases where the injured brother or sister was single and their parents are either deceased, in another state, or don't speak English. Clinically, it has been noted that siblings tend to be very devoted significant others and usually stricter and less protective than parents. It is emotionally very draining for siblings to try to care for their injured loved one while trying to manage their own daily responsibilities, and they will require a lot of team support and guidance.

Young siblings whose brother or sister was injured have to deal with reduced attention by parents, increased tension at home, change in the quality of the relationship with the injured sibling, and embarrassment in cases where the injured brother or sister display neurobehavioral difficulties. It is important to alert the school about the challenges that the sibling is experiencing so increased attention and support can be offered.

Family Training

One of the significant benefits of educating families on the repercussions of ABI is the reduction in number of common errors or misunderstandings. For instance, families tend to confuse a survivor's reduced executive functioning skills with laziness. Also, families tend to misinterpret a survivor's emotional lability or pseudobulbar affect with depression, and they confuse organic lack of awareness with psychological denial.

Lezak (1988) found that families cope more effectively with a loved one's injury when they have knowledge about the nature of the emotional changes associated with ABI.

Reinforcement of adaptive coping skills and positive problem-solving abilities is very important during family training. Grant, Weaver, Elliot, Bartolucci, and Giger (2004) found that a negative orientation toward problem-solving and a lack of preparedness for the caregiver role were variables that were associated with a higher risk of depression in caregivers of stroke survivors.

Family-based psychosocial intervention after stroke has been shown to increase efficacy and control, optimize social support, improve family cohesion, and promote effective problem-solving (Glass et al., 2000).

Palmer, Glass, Palmer, Loo, and Wegener (2004) discussed the value of educating survivors and families about depression post-injury and reviewing ways to adaptively solve psychosocial challenges that can contribute to depression. They also reviewed ways to recognize, identify, and manage symptoms of depression.

Dell Orto and Power (2000) highlight the value of group counseling for families by describing it as a "counterforce to helplessness, isolation, and desperation." Group counseling provides a forum for families to learn from each other's successes and failures and obtain training regarding productive ways to respond to survivor's behaviors and needs. Families can also benefit from being paired with other families who are further along in the process, who can act as mentors based on their first-hand experience with similar challenges.

"Rehabilitation is an equalizing process. No one much cares what you did before; they're focused on what you can do now and how you can learn to live independently again" (Crimmins, 2000). In running family groups, the equalizing process that Crimmins refers to become very apparent. For example, an unexpectedly close kinship developed between two women who were members of a family educational/support group. One was a pediatrician and the other was a tollbooth operator. Prior to their husbands' injuries, the two would likely not have become friends due to differences in age as well as cultural and educational backgrounds. However, within the group they were equals and they shared strategies and resources in a supportive manner. Their husbands who also had little in common except their ABIs were also group equals in their attempts to regain their autonomy and proceed to the next step of the process.

No one can understand and relate to a family whose loved one was injured the way another family in a similar situation can. Group counseling can be a great source of peer and professional support.

SL sustained a stroke ending his career as a college professor. His primary difficulties included expressive aphasia and rigid thinking. He and his wife, J, were both in their sixties and were struggling to keep their relationship intact despite post-injury challenges.

In a stroke caregiver's group, J shared her plan to sell the family home of many years due to her own physical difficulties that made the upkeep of the house too cumbersome. Her husband was very against the move, due to his desire to remain in a familiar setting and exert some level of control over his life. He also was experiencing

post-injury rigidity of thinking and difficulty with change. Despite J's attempts to reason with her husband and have others, including professionals and family members, try to convince him of the merits of the move, he remained uncompromising and hostile. J was very emotionally distraught by her husband's lack of support of her needs since she had always been very dedicated and supportive of him. J benefited greatly from ventilating her mixed emotions to the group, who could relate to her challenging situation and provide reassurance, emotional support, understanding, and helpful advice. Over time, J felt empowered by the support and advice of the group to go forward with the sale of the house.

How Families Can Help

Families need to know that it is a priority that they address their own physical and emotional health so that can successfully persist in caregiving. The following suggestions can optimize their effectiveness.

1. Reinforce carryover of learned strategies to the home setting to help survivors generalize progress to real life situations. If the client is resistant to doing same, speak to the therapy team and get ideas regarding how to address this obstacle.
2. Praise survivors on progress and provide only constructive criticism to help rebuild self-confidence. This is very vital as survivors tend to struggle with reduced sense of worth.
3. Help provide structure for survivors when they are not in rehabilitation so as to facilitate continued gains.
4. Keep survivors stimulated and active to help prevent regression but make certain they aren't overstimulated. Speak to the therapy team if you aren't sure how much stimulation is sufficient.
5. Be in regular contact with the neurorehabilitation team to exchange relevant information. Find out about available resources. Come to meetings prepared with questions.
6. Find an appropriate balance between over- and underprotectiveness to assure that the survivor is safe but also not prohibited from making gradual gains in self-reliance.
7. Encourage survivors to do home exercises. Assist the client in doing home exercises if they can't do alone.
8. Be flexible in creating a useful and realistic role for the survivor in the family. Families that identify meaningful roles for the survivor help clients rebuild a sense of purpose which boosts overall self-esteem.
9. Learn how to deescalate stressful situations so that the home is an environment of peace instead of chaos.
10. Participate in a family support group to obtain continued support and information.

11. Keep hopeful but realistic about continued progress.

The primary value of structured educational/support groups for families is that they provide knowledge about ABI and reinforce best practices for facilitating a loved one's recovery. It is comforting for families to meet others who can truly relate to the daily challenges they experience. Exchanging helpful ideas about how to handle difficult situations that arise can provide much support and reassurance to significant others. An agenda that is structured, facilitated by different team members, and that provides information about medical, physical, cognitive, and neurobehavioral sequelae of ABI best meets the needs of caregivers. A sample agenda includes:

1. Introduction—Review of the role of all team members and what to expect from rehabilitation.
2. Discussion regarding emotional and social changes post-ABI.
3. Discussion regarding neurocognitive changes post-ABI.
4. Discussion regarding the specialized roles of the physical therapist, speech/language pathologist, and occupational therapist.
5. Stress management training.
6. Problem solving how to adaptively handle challenging situations that arise.

Conclusion

A primary mission of the neurorehabilitation team is to alleviate family distress by providing information and support in a clear and sensitive manner. The team needs to aid and never impede a caregiver's attempts to cope constructively with the injury of a loved one. When the team is ineffective in meeting family needs, caregiver stress and frustration will be increased.

It is important for neurorehabilitation team members to be aware of the effects of prolonged caretaking on caregivers. The long-term implications of ABI can generate severe strain and tension on the survivor's family unit. Knowledge of the specific challenges that each family is dealing with and their particular strengths and vulnerabilities will help guide recommendations. All team members are responsible for prevention of caregiver breakdown and in providing families with skills, knowledge, support, and a sense of mastery.

References

Benn, K., & McColl, M. (2004). Parental coping following childhood acquired brain injury. *Brain Injury, 18*(3), 239–255.

Blais, M., & Boisvert, J. (2005). Psychological and marital adjustment in couples following a traumatic brain injury: A critical review. *Brain Injury, 19*(14), 1223–1235.

Cameron, J. I., Cheung, A. M., Streiner, D. L., Coyte, P. C., & Stewart, D. E. (2006). Stroke survivors' behavioral and psychologic symptoms are associated with informal caregivers' experiences of depression. *Archives of Physical Medicine and Rehabilitation, 87*(2), 177–183.

Cameron, J. I., & Gignac, M. A. M. (2008). Timing it right: A conceptual framework for addressing the support needs of family caregivers to stroke survivors from the hospital to the home. *Patient Education and Counseling, 70*, 305–314.

Carlozzi, N. E., Kratz, A. L., Sander, A., Chiaravalloti, N. D., Brickell, T., Lange, R., … Tulsky, D. S. (2015). Health related quality of life in caregivers of individuals with traumatic brain injury: Development of a conceptual model. *Archives of Physical Medicine and Rehabilitation, 96*(1), 105–113.

Crimmins, C. (2000). *Where is the mango princess*. New York, NY: Vintage Books.

Dell Orto, A. E., & Power, P. W. (2000). *Brain injury and the family*. New York, NY: CRC Press.

Glass, T., Dym, B., Greenberg, S., Rintell, D., Roesch, C., & Berkman, L. (2000). Psychosocial intervention in stroke: The families in recovery from stroke trial. *American Journal of Orthopsychiatry, 70*(2), 169–181.

Grant, J. S., Weaver, M., Elliot, T. R., Bartolucci, A. A., & Giger, J. N. (2004). Family caregivers of stroke survivors: Characteristics of caregivers at risk for depression. *Rehabilitation Psychology, 49*(2), 172–179.

Hart, T., Driver, S., Sander, A., Pappadis, M., Dams-O'Connor, K., Bocage, C., … Caj, X. (2018). Traumatic brain injury education for adult patients and families: A scoping review. *Brain Injury, 32*(11), 1295–1306.

Katz, S., Kravetz, S., & Grynbaum, F. (2005). Wives coping flexibility, time since husbands' injury and the perceived burden of wives of men with traumatic brain injury. *Brain Injury, 19*(1), 59–66.

Kitter, B., & Sharman, R. (2015). Caregivers' support needs and factors promoting resiliency after brain injury. *Brain Injury, 29*(9), 1082–1093.

Kreutzer, J. S., Gervasio, A. H., & Camplair, P. S. (1994). Primary caregiver's psychological status and family functioning after traumatic brain injury. *Brain Injury, 8*, 197–210.

Kreutzer, J. S., Serio, C. D., & Bergquist, S. (1994). Family needs after brain injury: A quantitative analysis. *Journal of Head Trauma Rehabilitation, 9*(3), 104–115.

Kreutzer, J. S., Stejskal, T., Ketchum, J. M., & Marwitz, J. H. (2009). A preliminary investigation of the brain injury family intervention: Impact on family members. *Brain Injury, 23*(6), 535–547.

Kubler-Ross, E. (1983). *On children and death*. New York, NY: Macmillan.

Lezak, M. D. (1978). Living with the characterologically altered brain injured patient. *Journal of Clinical Psychiatry, 39*(7), 592–598.

Lezak, M. D. (1988). Brain damage is a family affair. *Journal of Clinical Experimental Neuropsychology, 10*, 111–123.

Lorig, K., & Holman, H. (2003). Self-management education: History, definition, outcomes and mechanisms. *Annals of Behavioral Medicine, 26*(1), 1–7.

Man, D. (2002). Family caregivers' reactions and coping. *Brain Injury, 16*(12), 1025–1037.

Meixner, C., O'Donoghue, R., & Hart, V. (2017). Impact of the brain injury family intervention (BIFI) training on rehabilitation providers: A mixed methods study. *NeuroRehabilitation, 40*(4), 545–552.

Nabors, N., Seacat, J., & Rosenthal, M. (2002). Predictors of caregiver burden following traumatic brain injury. *Brain Injury, 16*(12), 1039–1050.

Oddy, M., Humphrey, M., & Uttley, D. (1978). Stress upon the relatives of head injured patients. *British Journal of Psychiatry, 133*, 507–513.

Palmer, S., Glass, T. A., Palmer, J., Loo, S., & Wegener, S. T. (2004). Crisis intervention with individuals and their families following stroke: A model for psychosocial service during inpatient rehabilitation. *Rehabilitation Psychology, 49*(4), 338–343.

Powell, J. M., Wise, E. K., Brochway, J., Fraser, R., Temkin, N., & Bell, K. R. (2017). Characteristics and concerns of caregivers of adults with traumatic brain injury. *The Journal of Head Trauma Rehabilitation, 32*(1), E33–E41.

Sabella, S. A., Andrzejewski, J. H., & Wallgren, A. (2018). Financial hardship after traumatic brain injury: A brief scale for family caregivers. *Brain Injury, 32*(7), 926–932.

Sady, M. D., Sander, A. M., Clark, A. N., Sherer, M., Nakase-Richardsn, R., & Malec, J. F. (2010). Relationship of preinjury caregiver and family functioning to community integration in adults with traumatic brain injury. *Archives of Physical Medicine and Rehabilitation, 91*(10), 1542–1550.

Serio, C. D., Kreutzer, J. S., & Gervasio, A. H. (1995). Predicting family needs after brain injury: Implications for intervention. *Journal of Head Trauma Rehabilitation, 10*(2), 32–45.

Serio, C. D., Kreutzer, J. S., & Witol, A. D. (1997). Family needs after traumatic brain injury: A factor analytic study of the Family Needs Questionnaire. *Brain Injury, 11*(1), 1–9.

Testa, J. A., Malec, J. F., Moessner, A. M., & Brown, A. W. (2006). Predicting family functioning after TBI: Impact of neurobehavioral factors. *The Journal of Head Trauma Rehabilitation, 21*(3), 236–247.

Witol, A. D., Sander, A. M., & Kreutzer, J. S. (1996). A longitudinal analysis of family needs following traumatic brain injury. *NeuroRehabilitation, 7*, 175–187.

Wood, R., Liossi, C., & Wood, L. (2005). The impact of head injury neurobehavioral sequelae on personal relationships: Preliminary findings. *Brain Injury, 19*(10), 845–851.

Chapter 14
Postrehabilitation After Acquired Brain Injury

Allison Muscatello and Jean Elbaum

As triumphant as the survivor, family, and neurorehabilitation team may be on the day of a successful discharge, it is premature to forecast continued growth or even stability at a later date. There are often ongoing challenges experienced by both survivors and caregivers following severe acquired brain injuries (ABIs), which can persist years after the injury, and long after rehabilitation ends, as is illustrated by the case of LV, below.

The accident struck me like a lightning bolt. A speeding motorist rear-ended my car, fracturing my skull, causing a severe traumatic brain injury. I was comatose for 35 days. I would not be returning to my previous life. My eyes crossed. I lost my sense of smell. I experienced severe coordination and balance difficulties. I had major visual challenges as well as cognitive and emotional difficulties.

Before the accident, I balanced my family (husband and two young sons) and career as a dentist successfully. I was just 49 years old, youthful and dynamic.

Post injury, following the prolonged coma, I started the neurorehabilitation journey, which has been my primary job since the accident. I followed all the steps from acute to postacute inpatient programming, shifting to daily, intensive outpatient therapies, and gradually reducing to part-time outpatient rehabilitation as my condition improved and my needs changed. It took 21 months to finally return home and become an outpatient.

I had three corrective surgeries, the last in 2015, to reposition both eyes for accident-induced esotropia and diplopia. Esotropia was eliminated but diplopia persists to this day. My double vision has been corrected through the use of prismatic lens eyeglasses. The improvement in my vision has also helped to increase my balance. I struggled to be cleared to return to driving and was reauthorized to drive again, in 2006, 8 years following my injury. I drove for 12 years from reauthorization to the present, which increased my independence and sense of control. I just

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recently decided to discontinue driving, as I recognized that my safety on the road was becoming an issue.

I was in a wheelchair for approximately 1 year followed by 2 years of using a walker. I then and still to this day use a cane to help me ambulate due to residual balance difficulties. I have made enormous gains in all areas, including my thinking skills, especially memory, emotional status, and in the use of compensatory strategies to work around my weaknesses.

As a result of some of my residual difficulties, a return to my profession wasn't possible until 2010. I did succeed in returning to my dental practice again for 6 years. I was deeply gratified to see a number of my previous, preinjury patients who were loyal and sought continued services with me. Before returning to my field, I worked hard to obtain an appointment as a clinical assistant professor at a school of dental medicine which I held for 9 years. I spent many years as a volunteer faculty member at the dental school, where I've instructed students in digital radiography techniques and interpretation.

I believe that my excellent recovery so far has been due to the combination of the intensive, integrative therapies I received over the years as well as my strong determination. My gains would have been impossible without either component.

After years of physical therapy, personal trainers, and health club memberships, my home has now become a near-professional gym, with free weights, a high-end stationary bicycle, and an elliptical machine, and I have become my own coach. This equipment provides the best fitness exercises for me, since I am unable to run. In the near future, I also plan to include yoga and meditation in my daily schedule. In addition to regular exercise through my physical fitness program, I have incorporated a healthy, nutritious diet and adequate sleep and rest into my routine.

My cognitive skills are honed by voracious reading of fiction, nonfiction, and periodicals. I continue to challenge my fine motor skills, compromised by the accident, through sewing, gardening, and cooking.

Additionally, my eye–hand coordination skills benefit from a return to playing the piano. Despite 8 intensive years of lessons, as a youngster, I will probably never achieve my preaccident, advanced level of piano, due to residual coordination deficits. Instead, I simply enjoy playing the works to which I was exposed in my youth to the best of my abilities.

What lies ahead? I hope that I can continue to establish new pathways in the unused portions of my brain to help further boost my walking, talking, vision, fine and gross motor skills, as well as balance. These days my cognitive and emotional status is at a pretty good level.

My greatest challenge over the past 20 years has been to stop bemoaning my losses and get on with my life as fully as possible. I know I will always bear some scars from the brain injury, but I continue to be optimistic about the future. I hope to continue to be an example of fortitude and perseverance in the face of the unexpected. I've learned that you always have to be prepared because you never know how life's rudders will twist and turn your pathways.

Preparing for Discharge

It is essential for the neurorehabilitation team to project survivor/family needs beyond their current status, and begin to lay the groundwork for next steps. The outpatient team must identify community/work reintegration options such as competitive employment, vocational training, or volunteer work, and begin working toward those goals while the client is still in rehabilitation. Postdischarge medical and psychosocial supports must be identified and discussed with the survivor/family, along with education regarding the importance of developing community-based support systems, in order to maintain health and wellness once formal rehabilitation services have ended. University speech clinics are excellent sources of long term continued speech therapy and postrehabilitation occupational and physical therapy programming can be very helpful as a next step following discharge from restorative therapies. Many survivors also benefit from alternative wellness activities such as yoga, tai chi, and creative arts. Survivor support groups that meet regularly are also a useful resource and can provide long term support and education. Clients who require more long-term care may be candidates for Medicaid Waiver-based programs. In New York State, these prior approval services include service coordination, independent living skills training, home and community support services, structured day program (social model), positive behavioral interventions, environmental modifications, assistive technology, community integration counseling, and more.

For clients who are not ready for reintegration to work or school and who need supervision and continued work on social and cognitive challenges, structured day programming (SDP) is an excellent option.

Social/Leisure Involvement Post rehabilitation: Structured Day Programming

ABI (Acquired Brain Injury) survivors with persistent difficulties in areas such as focus, short term memory, executive functions, functional communication, vision, and balance will pose particular challenges with regard to maintaining or redeveloping an active social life following brain injury.

Postrehabilitation and structured day programs can fulfill a tremendous need in this area and are often an untapped resource for a variety of reasons such as location, transportation, clients' lack of awareness, or barriers in finding a consistent, reliable, and helpful aide to accompany them to program.

Day programming offers a structured setting and reinforcement of compensatory strategies to boost cognitive and interpersonal skills. Group and individualized sessions can focus on areas such as social skills, independent living activities, awareness and adjustment, goal setting, cognitive fitness, coping skills, psychoeducation, creative arts and alternative wellness activities, as well as prevocational readiness.

The three cases below highlight the benefit of structured day programming and other postrehabilitation services for individuals who are recovering from severe acquired brain injuries requiring long term support, structure, and supervision.

Case 1 *Ms. A was in her late 40s, single, and an active member of her community until sustaining anoxia following a sudden cardiac arrest in 2011. Prior to her injury, she had owned her own salon where she had regular high-profile clientele well-known in the arts and entertainment world. She also volunteered as an EMT. Ms. A was very artistic, spontaneous, and open to learning new things. The oldest of four, Ms. A was used to being the go-to person for her younger siblings. Since surviving her injury however, Ms. A has struggled with severe cognitive deficits in areas such as memory and executive functioning skills in addition to impulsivity and reduced frustration tolerance. Having to deal with significant role changes including her siblings becoming her legal guardians has been very challenging. Ms. A also consistently struggles with the need for 24 h oversight and supervision.*

In 2014, Ms. A was discharged from a neurobehavioral center and began attending the structured day program three to four full days per week. The routine of the program has gradually benefitted her greatly. She has enjoyed the social support and interaction. She has developed a constructive outlook and is very supportive of peers with more severe challenges.

Ms. A has enjoyed and advanced through the structure and stimulation provided by the program. In addition to social groups, she has been especially engaged in special sessions facilitated by former clients such as a baking group led by a survivor who is a pastry chef or a yoga/meditation group run by another survivor certified in this area.

While at the day program, Ms. A consistently works on her goals of increasing her overall independence, working on her social skills, learning and maintaining compensatory strategies for her impaired memory, and reframing her identity and life role expectations.

Case 2 *Mr. M experienced anoxia following cardiac arrest in 2012 at the age of 23, while riding home on the train from NYC after a night out with friends. He was taken to a trauma center for immediate care and then transferred to a rehabilitation hospital for acute inpatient care. He required psychopharmacological treatment for agitation. A month later, he was transferred to a facility about 90 min north of NYC for inpatient subacute rehabilitation, but was moved to the neurobehavioral unit due to extreme agitation and confusion. He remained in this facility for five months with intensive 1:1 supervision when his family opted to take him home with supports through the NYS TBI Medicaid Waiver program, including postrehab Structured Day Program (SDP) services.*

When Mr. M first began SDP he struggled due to confusion, disorientation, severe memory difficulties, and suspicious thinking. He became extremely verbally aggressive and at times physically aggressive with staff, which led to his needing to be discharged from program while he worked with a neuropsychiatrist on stabilization through medication.

After a few months, Mr. M was able to restart SDP on a gradual schedule from one morning a week to gradually full-time programming as he progressed.

With the addition of intensive behavioral supports, adjustment-related counseling, and constant repetition and use of memory strategies, Mr. M has continued to improve over the past few years. He recently began attending a prevocational program in the community. He also recently worked with an organization where he received a therapy canine which has been an additional support in his overall recovery. He still attends part-time structured day program for the social aspect as well as a postrehab weekly exercise group. His family is also in the early process of exploring independent living with Medicaid Waiver supports.

Case 3 *Mr. P is a 62 years old male who had a stroke in 2016, which resulted in severe expressive and receptive language challenges. History prior to the stroke indicated that he was on disability due to mental health challenges.*

Mr. P engaged in restorative services for speech for about 6 months post-CVA (cerebrovascular accident); however, his participation and progress were inconsistent.

He was referred to SDP to continue to work on communication and socialization in a supportive, group setting. While Mr. P has challenges in SDP such as inconsistent participation in groups and frustration due to severe expressive communication challenges, he benefits from the structure as well as social stimulation and support.

Mr. P has developed friendships at the program and obtains a lot of support from his peers who try to engage him in different activities. He also has worked on speech-related exercises with a volunteer who is an ABI survivor. Additionally, he has shown an interest in creative activities such as painting and drawing, which allow him an opportunity to express himself despite the aphasia.

In addition to SDP, Mr. P has received ongoing aphasia support through a local university postrehabilitation speech clinic. He also has an extremely supportive aide, who takes him into the community regularly to participate in social activities he enjoys such as the library, shopping, and eating out.

For clients who are not ready or able to return to work or school due to residual challenges, involvement in postrehabilitation services providing structure and meaningful activities is vital so that client can continue to make gradual progress and prevent decline over time.

Chapter 15
Successful Transitions After Acquired Brain Injuries

Jessica Moskowitz

Recovery stories have threads, twists, and commonalities. The beginning of a personal tale might involve a surprise diagnosis or a tragic accident. The middle part of the journey has obstacles, such as memory loss, physical or emotional changes, and a struggle for independence. There are also the moments of hope when, at first, words are remembered, ideas are recalled, and small tasks are accomplished. Gradually, progress is seen as physical movements get less painful and more graceful. Psychosocial or cognitive groups become comfort zones where you realize you are not alone. The endings to our stories are never finite because humans are always encountering new challenges and searching for ways to cope and move forward.

Our acquired brain injuries have impacted each of the six of us with different outcomes. It's taken years of work to get to the stages of recovery we currently inhabit. Some of us have worked hard to ensure we can move on our own, whether it's relearning to drive a car or managing train routes. Some of us have returned to our old lives as new people. We are all familiar with setbacks and struggles. But we also have talented doctors and therapists who have provided excellent coping skills. Amazingly, all of our families have shown up and been there for us when we needed them most.

We all have strong drives to be productive and we focus on what we can do versus what might stop us. We compensate for our difficulties and our injuries disappear because we have devised ways to work around them. While there are a lot of different circumstances that brought our stories to come together here, the power of resilience is the takeaway ahead.

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Traumatic Brain Injury From a Car Accident

Martin is 26 years old and experienced a life changing event in 2012. He was a passenger in a car accident and the driver, his friend, was killed. Martin emerged from a coma with a traumatic brain injury and C1 and C2 fracture. “I never thought a simple walk across the street would be so challenging,” he wrote in a recent personal essay.

But since completion of years of outpatient interdisciplinary therapies, he has used fitness and the gym as his way forward, writing: “It takes the anxiety right out of me and helps me to end my day knowing I’ve done something to improve myself.” After a long road to recovery, he has advice for those who may struggle: never let anyone define your path ahead. “Believe in yourself and keep fighting for what you want and you can overcome a lot,” he writes.

Martin does have a lasting vocal communication issue. His has a speech disorder called dysarthria that makes it hard to understand him. To compensate, he uses his phone to type in what he wants to say to communicate with others. With this compensatory tool, he works full time in the financial industry and maintains a very independent life.

He thanks his mom for all the sacrifices she’s made. He shares: “I wouldn’t be where I am today if it wasn’t for her love, commitment and hard work.”

Encephalitis

A seizure in 2010 sent Chris, a sales manager, falling into a glass table in his living room. There was a misdiagnosis, then a diagnosis of viral encephalitis. After all of that, Chris began outpatient therapy and set on his path of recovery. He writes that he asked a lot of questions during his 6 months of therapy and a year and a half of weekend group memory sessions. Staying informed also helped him focus on the process of getting better. “I had to really force myself to be present in the moment,” he writes. It “made me feel better than always thinking about: what’s next?”

While the ringing in his head and throbbing pain persisted, Chris fought for all of his memory gains. He worked tirelessly with his therapists to use “CRAVE” strategies to chunk, repeat, associate, visualize, and elaborate his way to broader memory functionality. He set goals, writing: “I want to live independently again, to get back to work, and figure out a way to help someone else.” Chris was able to return to work and independent living successfully. Chris has worked with the “Big Brother, Big Sister” program since 2013. In 2015 he joined “Best Buddies,” to volunteer his time with someone with mental challenges. “I realize how lucky I am,” he writes.

Traumatic Brain Injury from a Fall

Robert also knows how life can change from one instance to the next. He was 20 years old, a student in his junior year of college in Pennsylvania, when he fell down the stairs and crashed into a steel door at the bottom. He needed emergency surgery to save his life. Robert spent 3 months in the hospital without skull flaps.

During his rehabilitation, Robert writes: "I needed to relearn how to do everything." His time was divided between physical, occupational, and speech therapy 5 days a week. He lost 30 lb. "It has been almost 5 years now since the injury and I still have problems with my memory, attention and some physical impairments," he writes. But he shares with others who may just be beginning their recovery: "It is a long road … but you can never give up. You have to keep working. Hard word does pay off, but it doesn't stop after you are done with therapy."

Robert now works as a per diem rehabilitation therapy aide as he was so inspired by the therapists that played a role in his recovery.

A Benign Brain Tumor

My own story involves a brain tumor that was deemed slow growing—probably in my head for a decade—before it made itself known. It was discovered, and shortly after, removed with deep brain surgery from the back left ventricle. It took months of outpatient physical, speech, and occupational therapy, but I slowly returned to independent living.

My recovery was made possible with the love and support of my family. My mother, in particular, deserves a massive thank you. She put her own life on hold to drive me to my appointments and take care of me when I was my most vulnerable.

"The experience has given me more perspective than before on the importance of living in the present, because things could always be worse—completely out of nowhere and totally unexpectedly," I wrote in an essay a few months after I returned to a very demanding career in broadcast journalism in 2015.

The past few years have been an adjustment because once recovery from the surgery ended, the seizures began. Now that I have seizures, I don't drink alcohol, I work out five times a week, I make sure to get plenty of sleep, and I use a meditation app on my phone to control anxiety. With all this cautious behavior, I still experience auras and breakthrough seizures a few times a year. Each time something happens that feels out of my control, it's a major emotional setback. However, once these times pass I remember I have so much to be thankful for. I might feel powerless for a short period of time but then my medicine will work as it should. I'll have wonderful experiences with friends and family and I return to the realization that life goes on.

Arteriovenous Malformation

Patti is a highly educated engineer with an active lifestyle and is raising three young children. She experienced an arteriovenous Malformation (AVM) in 2014 and her life was thrown into a tailspin. Patti spent about 5 months in the hospital after her brain surgery. She was pregnant with her youngest during her time in the neuro-intensive care unit. When she was able to, she started outpatient speech, physical, and occupational therapies.

Patti writes that she gave birth to a healthy baby girl about 5 weeks after being released from the hospital. While she faces struggles using her right hand, she has learned to work around it. She sees a psychotherapist once a month to reinforce positive coping.

Her baby just celebrated her third birthday. "I was truly very humbled and honored by the number of people who both visited me in the hospital and were there for my family in that time of need," she writes. Her husband never left her side. Her mom "dropped everything to care for my family." Patti had no say in whether she would have an AVM or not, but she is a survivor. Her advice to families and patients recently diagnosed with the condition is to be patient with yourself. "The recovery process is much more like a marathon than a sprint," she conveys.

Hepatic Encephalopathy

There is one man who wants to contribute his recovery story but wishes to remain anonymous. His is a bit of a cautionary tale: prioritize your health. This patient says he knew he was not OK when he attended an event with jaundiced eyes and a tight chest, but he put off seeing his doctor. Everything caught up to him one day when he vomited blood and passed out. His diagnosis is as follows hepatic encephalopathy, toxins in his bloodstream had traveled to his brain.

He admits he was abusing alcohol and food. "My life had gotten out of control," he writes. After several months of recovery, he lost 80 lb and was learning to value his health over his initial inclination to get straight back to work. In therapy, he worked on sharpening his memory and math skills, balance, and strength. Counseling brought a reduction in anxiety and boost in confidence. He's abstained from alcohol in the 4 years post his injury. His family continues to grow as he's had a fourth child since the medical emergency. "I am grateful about how things turned out," he writes. He currently works full time and is very successful in commercial real estate.

Random, tragic, surprising, awful events have happened to the people you've read about here. I hope you have also seen the larger thread of humanity too. We have all experienced some of the most challenging events life can put in front of you, but we are survivors. We have overcome. We thank those who love us and stood by us in our times of need. We aim to make the most of the time we have on this planet because we have all seen just how limited that time can be.

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