

Neuroplasticity: The Impact of Age and Injury

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### Abstract

**Background:** Neuroplasticity is an ongoing process of the brain that allows for learning, changing, and adapting to every day changes as well as to trauma. As we age, the rate of neuroplasticity (that combats the ramifications of brain injury) starts to decline. This has been seen throughout many different species and is the justification for why adult systems have more devastating deficits from injury than children. The brain can spontaneously recover from injury but for improved long-term results, speech and language therapy in conjunction with spontaneous recovery is ideal for maximal recovery of function and language.

**Purpose:** In this review, the primary goal is to discuss past and present research on neuroplasticity, neural aging and the effects of injury on the language centers of the brain.

**Results:** I have discussed neuroplasticity and peak neuroplasticity in children known as the critical periods and sensitive periods, discussion of normal aging on neuroplasticity, the results of prenatal strokes in comparison to the results of adult strokes, and the different types of recovery that occurs post-stroke/traumatic brain injury (TBI).

*Keywords:* Communication Disorders; Aphasia; Stroke; Neuroplasticity; Transcranial magnetic stimulation; Transcranial direct current stimulation; Neurorehabilitation; Interhemispheric interactions, Neurodevelopment; Synaptic plasticity; Neuromodulation; Critical period; Sensitive period; Transcranial magnetic stimulation, Developmental plasticity; Brain stimulation; Cerebral palsy; Perinatal stroke; Clinical trials, Critical period; Sensitive period; Neurodevelopmental disorder (NDD); Neurotypical; Synapse; Time-window; Neurotoxicology; Children; Development; Psychology

Our knowledge of the human brain is miniscule. It is a complex and incredible organ. Most of what we know about the brain's functions have been a consequence of injury or disfunction to specific areas of the brain. Instances of individuals who survived traumatic brain injuries (TBI) or had a cerebral vascular accident (CVA), more commonly known as a stroke have been ideal candidates for research to understand how the cortex specializes. The function of the cortex is associated with higher brain function such as thinking, perceiving, producing and understanding language. Researchers do not have the technology and understanding of the brain's circuitry needed to restore loss of function and develop new neurons and neural connections (Kleim, 2008). Depending on the age in which a person experiences a TBI or a CVA as well as its location and lesion size, will yield drastically different results in terms of deficits in language comprehension/understanding and recovery of language. Neuroplasticity's role in language recovery during spontaneous recovery and language intervention therapy.

Understanding how the brain works is a complicated task. The brain is a repertoire of behaviors, the ability to learn, remember and forget requires a sequence of steps and maturation. The process of brain development is determined by gene expression, intrinsic neuronal activity as well as external factors that include resources from the mother during embryonic stages (Meredith, 2014). The brain transports all the processed information on "highways" called neuro-pathways. These pathways are created by neurons that are formed to complete a task. In order to accomplish different functions, the neurons form specific pathways. It was only recently determined that these pathways and connections are subject to change in adult life (Hallett, 2001). These changes have been identified as brain plasticity. The purpose of this paper is to analyze what neuroplasticity is, how and when neuroplasticity changes with typical human

aging, how the onset of injury effects brain recovery as well as neuroplasticity's involvement in ongoing injury.

### *Neuroplasticity*

From birth, the brain begins to make connections and eventually creates specialized regions. These connections are neural synapses and pathways that are altered as a result of behavioral, environmental and neural changes. These changes give the brain its ability to learn, this process is called, neuroplasticity. According to Johnston (2004), "Plasticity, derived from the Greek word 'plaistikos' meaning 'to form' refers to the brain's ability to learn, remember and forget as well as its capacity to reorganize and recover from injury" (p. 73). For decades, researchers have asked themselves, what is neuroplasticity? How does it occur? and why does it occur? Some of these questions have been answered while others are debated and accepted as currently incomplete. This means that it has been accepted among the scientific community that understanding all aspects of the brain and neuroplasticity are currently beyond our medical and technical capabilities. With new technology, research may eventually produce a better understanding of these topics in the future.

In order to understand neuroplasticity, it is key to understanding how to define it. As previously stated, neuroplasticity refers to the ability to change and mold; however, there is more than one type of neuroplasticity that occurs in the brain. These different types include, developmental neuroplasticity, impaired neuroplasticity, excessive neuroplasticity, plasticity that makes the brain vulnerable to injury and adaptive neuroplasticity. As an infant begins to grow, the brain also grows and changes, as observed in the achievements of developmental milestones. This type of plasticity is known as developmental neuroplasticity. Developmental neuroplasticity

is based on several components, these include genetic encoding and a time-dependent, sequenced maturation process that is regulated by homeostasis. “Developmental neuroplasticity is an inclusive term that involves fundamental changes in neurogenesis, neuronal cell migration, synapse formation and structural and functional neuronal networks specialization leading to behavioral acquisition of motor and non-motor developmental milestones and adaptation to a constantly changing environment through learning and memory” (Ismail, 2017, p. 25).

Impaired neuroplasticity occurs in situations in which genetic or acquired disorders disrupt plasticity pathways. This type of neuroplasticity is commonly associated with neurological disorders that have disruptions with learning and memory. Excessive neuroplasticity can lead to disability through reorganization of new, maladaptive neuron signals. This type of neuroplasticity is associated with partial seizures and focal dystonia. Neuroplasticity that makes the brain vulnerable happens in situations where mechanisms designed to enable neuroplasticity to become over-stimulated which results in excitotoxic neuronal damage (Johnson, 2004).

Adaptive neuroplasticity, which was most critical for the scope of this paper, is the brain’s ability to change or adapt based on environmental change, emotional change, or change to the integrity of the structure. Johnston (2004) defines adaptive neuroplasticity as the changes in neuronal circuitry that enhance a special skill with practice. It also allows the brain to adapt or compensate for injuries or changes in sensory input.

Once researchers were able to understand how neuroplasticity should be defined, it was easier to research, track, and evaluate thus leading into the next question, how does neuroplasticity occur in the brain? Hallett (2001) observed “Their [cellular mechanism] physiology has been studied in model systems, and we have only incomplete knowledge as to which mechanism applies to which phenomenon” (p. 107). Despite having incomplete

knowledge on the subject, researchers were able to observe how the central nervous system (CNS) contributes to brain plasticity. “On a molecular level, CNS receptors undergo major alterations to achieve adult-like patterns and are thought to regulate the critical and sensitive periods of development... These biological processes are subjected to genetically programmed, time- limited periods, called the critical period or sensitive periods, during which the brain is most amenable to change” (Ismail, 2017). Critical periods and sensitive periods in the literature have been separated by slight differences in their definition which will be discussed in further depth in the following section. But, the critical period and sensitive period are time related periods in which neuroplasticity undergoes the greatest changes to the structure of the brain.

It is important to understand that neuroplasticity does not only occur during specific ages or moments in development. Rather, neuroplasticity occurs over the course of a lifetime, always changing in response to new experiences. There is overwhelming evidence that suggests the brain remodels and changes its neural circuitry continuously in order to encode new experiences and enable behavioral change (Kleim, 2008). What was once thought to be a single isolated event has now been dismissed, resulting in a new theory that neuroplasticity does not stop after puberty. Regardless of age, the brain is flexible and capable of change. It has the capacity for functional and structural neuroplasticity throughout the human life span Raymer (2008). “Neuroplasticity reflects the capability of the brain to modify throughout life by adapting at different levels, to environmental exposition. It underlies the processes of learning and memorizing and the damage-induced processes of brain recovery and reorganization. Neuroplastic mechanisms appear to be greatest during infancy, the so-called critical period, at a time when brain maturation has a faster pace” (Simona, 2015). Although neuroplasticity does occur over the lifetime, that does not discount that there are heighten peaks at which

neuroplasticity is most abundant. These early periods of neuroplasticity are often linked to the concept of the critical periods and sensitive periods.

### *Critical and Sensitive Periods*

There is a subtle distinction between the terms critical period and sensitive period. “Historically, critical periods have been used to describe brain circuit-based phenotypes including ocular dominance in the visual system or synaptic plasticity in the developing somatosensory cortex. Sensitive periods, on the other hand, are often referred to as time windows during which exposure of the organism to external factors or experience modulates the emergence of specific behaviors” (Simona, 2015). Consequently, a critical period can be defined as a developmental period in which a skill or characteristic is acquired. A sensitive period on the other hand is a period of maturation where the environment heavily influences the development of an organism. For the context of this paper, the terms critical period and sensitive period will be used interchangeably as a term that refers to heightened periods of neuroplasticity.

The critical period length varies from individual to individual. Thus, the inconsistency of the critical period length creates debate about its existence. The debate over the critical periods existence has other components to it besides duration. Components such as age-related factors, language development, and second language acquisition can be confounded with experiential and motivational factors (Gervain, 2015). The critical period holds important medical implications on the medical pathology of brain research (Ruben, 1997). Testing the existence of the critical period would cross ethical boundaries; however, the critical period has been observed throughout many different species. The data that have been collected in animals provides viable information when relating to human development. What we can gather from these sensitive periods provides

a framework for how the brain changes and grows normally (Meredith 2014; Simona 2015).

Knowing when the brain grows and what changes in the brain during those times of growth is important to help determine the timeline of events that make up the critical period.

In 1967, Lenneberg popularized the idea of the critical period in his paper “Biological Foundations of Language”. Lenneberg claimed that the critical period for humans began during infancy and extended into late childhood and possibly until puberty. Grimshaw (1998) examined indirect data that confirmed and expanded on Lenneberg’s initial hypothesis by investigating the exact timing and nature of the critical period.

Grimshaw was not entirely correct in her analysis claiming that the critical period had an exact time frame, as noted earlier there is a range of duration amongst individuals. When thinking about the critical period in terms of language acquisition, there were incorrect assumptions in the early research. At first, language was thought to be an innate process that humans were born with. The view of language as an innate function prevailed from ancient times until the beginning of the 19<sup>th</sup> century, this was dissociated from ideas of neuroplasticity. From then on, cases and evidence were reported that challenged the view of language as being an entirely inborn capacity (Ruben, 1997). Cases of children never acquiring language due to extreme deprivation of language exposure, the concept that language was innate was brought into question. Today the consensus is that language is acquired during this critical period where the brain is most plastic. Lenneberg used children who experienced a cerebrovascular accident (CVA) as examples of the existence of the critical period by examining language recovery post CVA. Grimshaw (1998), on the other hand, disagreed with his method and felt that an accurate test of the critical period is to test the ability to acquire language after the “closing” of the critical period. Using extreme deprivation as suggested by Grimshaw as a way to directly test of the

critical period is fortunately not common. The reason that injury is not a true test of the critical period is because some, all or portions of language (depending on age of onset) had already been typically acquired and when injury disrupts that typical language development, other areas of the brain have to compensate. “These findings reflect the potential for nonlanguage areas of the brain (either in the right or left hemisphere) to take over language functions after insult, and the time-limited nature of this recovery. They do not address the existence of a critical period during which normal dedicated language areas of the left hemisphere are predisposed to the acquisition of language” (Grimshaw, 1998, p. 238). Grimshaw (1998) argued that Lenneberg was not testing for the critical period in his study, but rather he was testing the brain’s ability to recover previously learned language function after an injury.

Ruben (1997) observed, “The current view of critical periods/sensitive periods for language acquisition is based primarily upon psychophysical observation, many of which are anecdotal, and only a few of which include quantitative data and/or have been reproduced” (p.202). Because there are few cases of extreme neglect reported in this literature, the data is also limited, thus making it difficult to conclude that the critical period has a cut off. Evidence contradictory to the idea of the closing of the critical period can be found in work of Ruben (1997). For instance, Ruben (1997) comments that there is research that examines the phonemic critical period, “Other studies indicate that the phonemic critical period may be severely constricted or closed by the end of the first year of life” (p.203).

However, as mentioned previously, neuroplasticity continues throughout the lifespan (Kleim, 2008). Thus, this “closing” of the critical period may no longer be an accurate term. An article written by Balari and Lorenzo (2015) asked other researcher professionals in the field if adopting a gradient view of the critical and sensitive periods rather than the traditional window

view would be more accurate, especially when referring to language acquisition. “It has to do with the suitability of the idea of ‘gradient of language’ which replaces here the concept of ‘faculty of language’, as well as the associated ‘critical point(s)’ concept, to accommodate certain intriguing findings” (Balari, 2015). Knowing that neuroplasticity occurs over the course of a lifetime, the gradient view of neuroplasticity would account for neuroplasticity seen in adulthood, as well as the changes that occur in early childhood until puberty, and the wide age range observed in typical development in which the critical period is presumed to exist. The gradient view also accounts for the idea that neuroplasticity does not close, but rather changes form as we age.

After puberty, the critical period and sensitive period begin to decline. This declining process can be referred to as adult neuroplasticity. The adult system will continue to change and mold to sensory input and continue make modifications to behavioral responses but, the responses will not be as drastic as the child’s system neuroplastic responses. I will discuss in later sections, this declining of neuroplastic mechanisms is the reason we see drastic deficit differences in recovery of language function after injury in the adult and child systems. The recovery of language in the adult system further supports this idea of a gradient view of neuroplasticity rather than a closing of the critical period after puberty.

### *Language Acquisition*

It has been known that the young brain has a greater capacity for language than in the adult counterpart. This is because of the young brain’s repertoire of neuroplastic responses (Ismail, 2017), seen in the capacity for learning and memory that is reflected in the ability to learn to play musical instruments or become proficient in sport (Johnston, 2004), and to learn

non-native language and linguistic systems (Balari, 2015). Young children are predisposed for acquisition of language and exhibit sophisticated speech and language perception and learning from birth (Grimshaw, 1998; Gervain, 2015).

Language acquisition is part of the developmental milestones children move through as they grow and develop (i.e., moving from crying, to babbling to words to phrases to sentences). Children begin to experience the world as non-linguistic beings. They attain the mastery of the intricate properties of human languages within the first few years of life, traversing along the way a series of distinctive stages and milestones (Balari, 2015). The first stage of typical language development is the pre-linguistic stage in which infants under the age of one are expected to cry, coo use pseudo language or babbling that mimics the native language in its intonation and form. Next is holophrases which involves using many single word utterances, after holophrases are two-word utterances. These include combining two words together to form a short sentence. This stage may appear between ages 18-22 months. By age three, children should have nearly 1,000 words and are combining sentences. Gervain (2015) found that the speech processing network that newborns have shown similarities with the adult network, thus indicating that there is an early brain specialization for language in infants.

### *Injury to the Brain*

Injury to the brain can happen at any age. The two major types of injury are called a traumatic brain injury (TBI) or a CVA which is commonly known as a stroke. TBI's are any major outside force that causes brain injury. These can be gunshot wounds, concussions, and other accidents that result in a blow to the head. A stroke or CVA is a when an area of the brain is cut off from blood flow usually do to a clot. Stroke is a common disorder that can often lead to

significant disability. The neurologic deficits are worse at onset and in most patients, there is some recovery of function (Hallett, 2001).

Typical childhood development can be disrupted when a TBI or a CVA occur. Although, strokes are often associated with older adults and the elderly, “You will not likely incur a higher period of risk for ischaemic stroke than the week you are born. A term newborn carries a risk >1:3500, three- fold higher than a week in the life of a diabetic, hypertensive, smoking adult and eight- fold above all adults. An additional 50% of perinatal stroke presents later in infancy” (Kirton, 2017, p. 76). It was also found that approximately 75% of perinatal stroke survivors have neurodevelopmental deficits; these deficits include, deficits in language, vision, cognition, behavior and epilepsy also occur, present in 20-60% of arterial strokes (Kirton, 2017).

Deficits for both infants and adults are established based on the following criteria, the size of the lesion, it’s location and the timing in which the cerebral vascular accident occurred. Depending on the timing of the insult, the brain can be affected in one of two ways for prenatal infants. The first being during periods of neuronal proliferation, migration, and cortical organization, resulting in brain malformations. This occurs during the first or second trimester of pregnancy. The second is during periods of selective vulnerability of white or gray matter and result in periventricular or cortical and deep gray matter gliotic or cystic lesions that occur during the third trimester (Simona, 2015). For adults, deficits are more prominent especially with age, this is due to the declining neuroplastic mechanisms. Factors that produce different results of the study include, age of onset of the injury, the type of injury, and the location of the lesion(s). Cerebral reorganization is most interesting in congenital brain lesions as they naturally encompass the different combinations of distribution (focal and diffuse) and timing (early gestation, late gestation, and term) of damage (Simona, 2015).

Aphasia is a common consequence of a stroke, it is a communication disorder that results from damage to the language centers of the brain, usually in the left hemisphere. The injury must affect an extended network of cortical and subcortical structures perfused by the middle cerebral artery in the left hemisphere to see deficits in language, speech or both (Hamilton, 2011). Luckily, children who suffer from strokes and childhood aphasias have a recovery rate that is not usually seen in the adult counterpart. A left hemispherectomy performed for epilepsy as late as the second decade indicated that children's recovery of receptive language was a great demonstration of the early brains ability to recover after injury (Johnston, 2004). Perinatal stroke is an ideal human model for the study of developmental neuroplasticity because of the combination of distinct differences in timing and location of lesions (Kirton, 2017).

Balari (2015) observed that research conducted by Lenneberg in 1967 was indirect, yet informative. It was concluded that children who experienced trauma before the age of 3, reacquired language as if they had not suffered any trauma at all. However, children who experienced trauma after the age of three had lingering patterns of impairment. This predicted pattern of recovery and residual deficits was observed in individuals ages 4 to 10. After 10 years of age the gradual reacquisition of language became unpredictable as it would be of a typical adult with aphasia associated with a stroke (Balari, 2015).

### *Spontaneous Recovery and Therapeutic Language Intervention*

The brain can heal spontaneously, as well as with language intervention therapy. In months following a CVA or TBI, the brain begins to heal on its own. This natural healing is called spontaneous recovery. When the lesion affects the language centers of the brain, causing

aphasia, the traditional treatment is to provide intervention as early as possible. It was suggested that providing as early as possible suggesting treatment in chronic stages would be less effective. However, research has found that patients who delay their aphasia treatment by 3 months caught up to other individuals who had begun aphasia therapy in the subacute phase (Raymer, 2008). Other research has also suggested that spontaneous recovery occurs within the first two to three months post-aphasia onset (Raymer, 2008; Hallett, 2001; Hamilton, 2011). However, this recovery is influenced by a number of factors. These factors include, “lesion site and size, and the existence of prior strokes” (Hamilton, 2011). Spontaneous recovery occurs due to plastic changes in the brain and the sprouting of new axon terminals to form new synapses (Hallett, 2001). The brain begins to heal by making new synaptic connections in different areas of the brain that take on the role of the language centers of the brain.

Spontaneous recovery is most prominent the first few months post stroke but, this does not mean recovery is by any means complete. There is long term recovery as a result of therapeutic language intervention. “I believe that rehabilitation is potent. Otherwise, why would we get up every day and do what we do?” ... so what is the evidence that treatment is “potent?” (Rothi, 2001, p. 118). There are several types of therapy to initiate neural activity, these include, (1) Recruitment of lesioned and perilesional left hemisphere regions for language-related tasks, (2) acquisition, unmasking or refinement of language processing ability in the nondominant right hemisphere, and (3) dysfunctional activation of the nondominant hemisphere that may interfere with language recovery” (Hamilton, 2011). Language intervention therapy expands on those synaptic language connections made during spontaneous recovery, thus improving language function beyond spontaneous recovery. Language intervention therapy also develops long term recovery of language by strengthening those new synaptic connections.

There is a need for both spontaneous recovery and language intervention therapy to produce the best long-lasting recovery of language. After the brain spontaneously heals, speech language pathologists (SLPs) implement therapeutic interventions that are patient centered and grounded in research evidence. These goals may include using the process of learning, compensatory strategies, and other evidence-based therapies. In neuroscience research, there are two main categories in which improving function after brain damage fall into. The first effort is to limit the severity of the initial injury to minimize loss of function and the second effort is to reorganize the brain to restore and compensate for function that has already been compromised or lost (Kleim, 2008). After the initial injury is treated by medical professionals, it is the role of the SLP to help the brain reorganize language function to other areas of the brain (i.e. moving language typically found in the left hemisphere into the right hemisphere).

The SLP should know the difference between adult neuroplasticity and childhood neuroplasticity, how the lesion location effects deficits seen in adults and children, and how a client's personality and willingness to learn will affect the type of treatment is used. Brain injury is incredibly individualized which can make the SLP's job challenging in finding an ideal evidence based treatment plan. "If, as speech pathologists, we accept the premise that behavior emanates from the brain and if we accept as our goal reconstitution of behavioral functioning after loss due to injury/disease through rehabilitation, then it is important that we understand what is happening to the brain structurally and physiologically during recovery and in response to rehabilitation" (Rothi, 2001, p. 117).

*Post-Stroke Recovery: Children Compared to Adults*

In the wake of injury, there is a substantial difference between how the child system recovers from injury in comparison to how the adult system recovers from injury. In the developing brain is much less vulnerable than the adult brain to detrimental effects of injury. Observation of this is in the wake of localized brain injury, children are more resilient in that the brain recover faster and with less lingering deficits, therefore the developing system must be more plastic and responds to injury in ways that the adult system cannot (Stiles, 2000). This is because the neuroplastic mechanisms are in abundance and seamlessly make adjustments in synaptic pathways. A child who sustained focal brain injury can compensate for that injury and recover differently than an adult who has a similar injury. There is overwhelming data that suggest that focal brain injury sustained in childhood results in more limited patterns of behavioral and cognitive deficits than comparable injury in adulthood. These less devastating outcomes following early childhood injury are attributed to the developing brain's capacity for plastic reorganization and that reorganization declines with maturation (Stiles, 2000).

Production and comprehension of language is often affected in the wake of a left hemisphere stroke or brain injury. Children with unilateral lesions recover language functions more successfully than do similarly afflicted adults. The current research in focal lesions (FL) explores their effects on language recovery (Grimshaw, 1998). Studies of language acquisition in FL began with populations with the earliest stages of language acquisition up until the middle to end of the school-aged period. It was predicted that children with LH injury would have more pronounced deficits than children with RH injury; production deficits would be associated with anterior injury and comprehension deficits would be associated with posterior injury (Stiles, 2000). The evidence concluded that there was pervasiveness of language acquisition deficits in the FL population and that there were no significant differences between the LH and RH

populations. They also found that older children had persistent, subtle disorders in pragmatics which persisted into the school aged period (Stiles, 2000). The children with FL injuries were able to compensate for injury with minor deficits. Even though acquiring a prenatal or perinatal injury is more optimal than acquiring an injury later in life, an uninjured brain is always the ideal system to function on.

In the adult system, the neuroplastic mechanisms begin to declining with age. The adult synaptic pathways have strong connections to carry out specific jobs (i.e. speech production residing in Broca's area and language function residing in Wernicke's area). When those synaptic connections are broken or damaged, it is harder for the adult system to compensate because of the strong synaptic pathways that have had years of reinforcement and are less adept to take on another function.

Adults appear to demonstrate a different trajectory of recovery post brain injury. Adults who experience focal brain injury early in life do not manifest the same extent and magnitude of cognitive and affective impairment as adults with comparable, but later occurring injury (Stiles, 2000). Again, this is due to the decline of neuroplastic mechanisms. If the injury or damage occurs early in life, the recovery of language and other functions is more obtainable than if the injury occurs later in adulthood. This is why adults with similar lesions but experienced damage at different stages of life do not have similar deficits.

The resilience of the brain can be observed in both child and adult systems in its ability to reorganize functions typically found in the left hemisphere into the right hemisphere. When the left hemisphere (LH) is damaged and language function is affected, the brain will often reorganize language function into the right hemisphere (RH). Researchers continue to investigate

the effects of the right hemisphere reorganization on language recovery. Patients with early injury to traditional LH language areas do have substantial preservation of language, and linguistic functioning appears to be mediated by the RH in a significant proportion of cases (Stiles, 2000). This occurs because the brain develops two systems, the primary language system that resides in the LH and a secondary system. If the primary language system is injured or lost, the secondary systems are available to mediate language (Stiles, 2000).

The right hemisphere is not the only location in the brain that can reorganize language. “There is considerable evidence that perilesional areas of the left hemisphere acquire or reacquire language ability in the weeks and months following injury. It has long been accepted that the size of the left hemisphere infarctions in perisylvian language areas correlates with initial aphasia severity and inversely with aphasia recovery (Hamilton, 2011, p. 41). The localization of lesion, the size of the lesion and the individual’s age when the injury has acquired plays a role in how the brain will recover language function in different areas of the brain through spontaneous recovery. “Plasticity is a central process in both brain development and in the processes, that underlie neural reorganization in older organisms. The data suggest that the alternative patterns of brain organization observed following early injury do not require special, transient plastic mechanisms, these data indicate what is observed following early injury in a perturbation of a normally operating system” (Stiles, 2000, p. 255).

### *Concluding Remarks*

Brain injury is the most direct way of learning about the brain. The brain's ability to cope with injury is referred to as neuroplasticity. The point of heightened neuroplasticity are called the critical periods and sensitive periods. Language acquisition resides during the critical period

which changes over time. Injury that occurs prenatally and perinatally have less detrimental effects after a TBI or a CVA than seen in the adult counter-part. Infants are at a higher risk of stroke than adults with diabetes, heart disease, and other high-risk factors. This is due to the molecular and maturational development of the brain during childhood. The adult system is not as resilient as the child's system; however, recovery is still possible for those who suffer a TBI/CVA later in life. The brain will spontaneously recover after a stroke within the first few months after an injury, however this does not negate the effects of therapy post stroke. Especially with individuals who acquire aphasia post-stroke.

Knowledge of brain function, brain plasticity, and the critical period are still incomplete. However, the knowledge we have acquired on these topics have helped develop therapy strategies that are effective and have led to more research questions. Consistent research and therapy seek to improve the lives of those who have suffered from brain injury regardless of age.

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