

Neurological Development for Learning

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Neurological Development for Learning

The ability to learn is based on the learner's neurological development. Summarize as it relates to human learning the phylogenic, embryological and postnatal stages of neurological development. Evaluate how delays or incomplete development could affect the learning process.

Form and function of the nervous system determine the ability to learn.

“The study of learning unites education and neuroscience” (Goswami, 2004, p.1). Every thought is represented by a flow of activity in a network of neurons (Posner and Rothbart, 2004). Learning itself is the process of changes in connectivity via strengthening or weakening the connections (Goswami, 2004). It is by understanding how the brain and the nervous system develop that we can understand how to improve the neurology of learning and overcome learning and behavioral disorders.

The brain is an amazing organ. It consists of approximately 100 billion gray cells and perhaps 500 billion to a trillion supportive glial cells (Scheibel, 2002) that make up a three pound chunk of mostly fatty tissue (Carey, 2002). At the end of pregnancy the growth rate of these cells is very fast with 50,000 cells being grown every second (Scheibel, 2002). The brain continues to develop and mature through approximately age 20 as shown via time-lapsed MRI scans (Gogtay, Giedd, Lusk, Hayashi, Greenstein et al., 2004a).

The brain controls nearly every bodily activity (Carey, 2002). When the brain is functioning at a high level it can be applied to intellectual pursuits such as learning and decision making. When it is functioning at a less than healthy level, it can be the cause learning and behavioral disorders. Psychiatric disorders of childhood are becoming more and more linked to

neurodevelopmental causes (Durston, Hulshoff Pol, Casey, Giedd, Buitelaar, et al., 2001). Goswami (2004) suggests that as we learn more about the brain and its development that it will allow us to understand and implement processes for educational benefit. Understanding the development of the brain is critical to the understanding of developmental disorders of the brain and nervous system (Gogtay, Giedd, Lusk, Hayashi, Greenstein et al., 2004b).

The maturation of brain regions and their connections are necessary for all cognitive, motor and sensory development (Paus, Zijdenbos, Worsley, Collins, Blumenthal, et al., 1999). Often times learning and behavioral disorders stem from a functional disconnection of various parts of the brain due to abnormal development of the nervous system. A comparison of phylogenic, embryologic, early childhood, and gender dimorphic development will be covered. The areas of the nervous system that are vulnerable during the development process will be described and the ways that normal development can be disrupted will be discussed.

Phylogenic

Development of the human.

Charles Darwin started the philosophical argument regarding evolution in 1859 and that argument is still being waged today (BBC, 2002-2004). One need not be an evolutionist to learn from comparative anatomy. The value towards understanding the relationships between species and the functions of the nervous system and organs remains the same whether one is a creationist or one believes in the theory of evolution. It is not the purpose of this document to support, challenge or otherwise engage in this debate. It is the purpose to, as Lee (as cited by Stein, 2003;

as cited by Richardson, 2003) stated, “Absorb what is useful” and to share even challenging information and categorization in a perspective that will be useful for understanding neurological development as it relates to learning and behavior.

The human body has been looked at from an evolutionary perspective for a variety of reasons. Fitch and Giedd (1999) evaluating the developing vocal tract from an evolutionary perspective. Sepkoski (1993, in Gould, 2001, p. 39) puts it this way, “The body of any organism reads like an evolutionary chronicle.” Sepkoski provides an outline that starts at over 3,500 million years ago and spans to about 5 million years ago. There are key biological developmental gifts that he believes were donated by species to develop the human body: from bacteria, DNA and anaerobic metabolism was derived; with protists, plants, and fungi humans obtained chromosomes and aerobic metabolism; other animals donated tissues and external food; fishes gave rise to an internal skeleton; amphibians first showed four limbs; birds and reptiles offered internal fertilization; other mammals first gave hair; and primates developed finger nails (Sepkoski, 1993).

Even with these so called gifts, it would be hard to explain the leap from primate fingernails to the fingers of William Shakespeare writing *Hamlet*. Something extraordinary had to occur, the development of the human brain. It is the development of the human brain that sets man apart from all others. Andrews and Stringer (1993) state that it is from physical changes in the brain that man obtained powers that allowed both creativity and invention.

Genetic Seeds

Nature versus nurture has been argued through the ages. Quantitative genetic studies have demonstrated that it is not nature or nurture but nature and nurture, because the outcome depends on both factors (Rutter, 2002). Genetic influences have been linked to brain abnormalities and psychological traits including autism and schizophrenia (Rutter, 2002). The link appears to be that genes can influence the development of the nervous system and that genetic influence along with environmental events in a life time can determine a learner's neurodevelopmental outcomes.

The brain is such a complex piece of machinery that nearly half of the entire human genome is involved in the development of this organ, even though it will only end up with comprising two percent of the body's weight (Scheibel, 2002). Genes exert their greatest power while still in the embryo, guiding foundational development (Peterson, 2004). When activated a gene may trigger other genes to become active and this process often follows a lineage of cells (Schaffhausen, 2004). Yet Rutter (2002), claims that even a factor of 90% heritability does not necessarily mean an absolute eventuality if environmental factors do not trigger the necessary genetic response.

Developmental stability is a term that describes how resistant to change an organism's genetics are to environmental influences (Thoma, Yeo, Gangestad, Lewine, and Davis, 2002). Fluctuating brain asymmetry may be used as a measure of this kind of change because of genetic propensities towards neurogenesis and neural pruning (Thoma, Yeo, Gangestad, Lewine, and

Davis, 2002). Neurogenesis and pruning refer to the rapid growth of neurons and their subsequent pruning of redundant or unused neurons. This process assures that there are enough neurons to adapt to any environment and eliminates those not necessary for development in the environment that an organism resides in. The neurogenesis process is such that peak density occurs at eight months and pruning continues till approximately age 16 where it is maintained till the early 70's where a gradual decline is seen (Casey, Cohen, Jezzard, Turner, Noll, et al., 1995).

Intelligence is thought to have genetic links. Brain structures based on similar genes were also similar and that frontal gray matter differences in volume and structure were linked to Spearman's g (Thompson, Trone, Cannon, Narr, van Erp, et al., 2001).

Motricity drove the development of the nervous system.

A nervous system is only necessary for an organism that has active movement; this movement is called motricity (Llinas, 2001). This occurs by sensory afferent messages that bring in information about the environment and motor efferent messages that guide the body in its interaction with the environment. In between the receiving and the acting is a decision based on the prediction of what will happen in the environment in response to the action being contemplated.

Prediction in a goal oriented way is the very essence of brain function; and it began with the need for prediction in movement (Llinas, 2001). The need to get to where the food is or the need to escape a predator, are both responses to a prediction that is made based on sensory input.

Even the act of moving towards a perception of what will be pleasurable or away from something expected to be painful, are motor responses based on the prediction of information obtained from the environment. “The evolutionary development of a nervous system is the exclusive property of actively moving creatures,” (Llinas, 2001, p. 17).

Melillo and Lesiman (2004) build on top of Llinas belief that motricity drove the development of the nervous system. They state that “...the brain probably evolved based on its dependency on information stimulation from the muscles and joints, especially spinal muscles and joints and the ability to maintain and upright posture” (Melillo and Lesiman 2004, p. 37). A timeline of concurrent development of higher levels of the nervous system providing higher prediction and a concurrent higher level of motricity (locomotion) is described in their text.

Melillo and Leisman (2004) propose that the nervous system’s tie to movement begins with sessile creatures much like a sea squirt which has no brain in its adult state but has approximately 5000 neurons in the form of a neural net (Kandel, Schartz and Jessel, 2000). Sessile creatures require little or no nervous system in their adult stationary state but in their larval state they move similarly to a tadpole. During that time they have a very primitive brain that dissolves once it finds a place to feed and becomes implanted as a mature adult version of the species. Melillo and Leisman (2004) propose that somewhere in evolution the larval state became sexually mature allowing for reproduction at that level of maturation. Thus begins the evolution of the brain from their perspective.

Moving up the evolutionary ladder is a fish that adapts to gravity as it is suspended in water and moves up and down and back and forth. The development of jaws from gill-slits allowed greater fuel supply and warranted the ability to move (Melillo, 2004). Fish have greater development of brain than the sessile creatures but it is not challenged by gravity to an appreciable extent due the support of the water against gravity, so it requires less capacity for prediction than creatures that reside out of the water living in the full gravitational effect. Even with a small amount of gravitational effect, there is evidence of brain development including the cerebellum to aid in coordinating movement activities (Melillo and Leisman, 2004).

As creatures leave the protection from gravity in their aquatic habitation, more demand for prediction is necessary and an evolution of the brain is seen. As life left the water first on a part time basis as amphibians, then reptiles followed by mammals we see significant changes that allow for further development and support for the nervous system. The addition of lungs and changes in limbs such that they were placed more underneath the creature to allow more efficient breathing, thus providing more oxygen which a requisite fuel for the cerebellum and cortex (Melillo and Leisman, 2004).

Bipedal movement separates primates and humans from other four limbed animals. This required further development of the vestibular system for the handling of unconscious balance and movement perception driving further development of the cerebellum and cortex, (Melillo and Leisman, 2004). Bipedal movement thus drove growth of the brain and gave it an exaptation, meaning an evolutionary change for one purpose that provided for an unintended benefit of an

additional evolutionary change. This exaptation was the massive cortical brain growth and particularly the formation and growth of the neocortex. In essence, this excess cortical growth allowed for more brain to brain synapsing, creating association centers and other benefits in addition to the brain to extremity and brain to organ connections present in other animals.

These brain-to-brain connections, or association centers, allowed the development of more human functions like language. Their function still relies on the brains ability to predict. Cognition can therefore be thought of as internalized movement (Llinas, 2001).

The cortex of the brain is made up of six layers (Scheibel, 2002). The process of the cortex and its association centers is essentially predicting a generalization or pattern recognition process that is handed up through the six layers of the cortex. Each layer makes a generalization or stereotype of the significance of the sensory patterns described by the previous layer creating a multilevel system of prediction (Hawkings, 2005). Hawkings, the founder of the Palm Pilot suggests that someday computers may be constructed and programmed in this manner and the reality of computers that can learn or what some call intelligence machines will become possible (Hawkings, 2005).

While the evolutionary perspective has value in understanding the value of motricity in development of the nervous system; its primary value is demonstrating the survival priority of the motor system over the cognitive system. The brains development is based on survival (Jensen, 2000). If developmentally and for survival sake the sensory-motor system developed

first, then it logically would follow that the sensory motor system must preferentially be fully developed first to allow for cognitive development. In other words, if the sensory motor system is delayed, there will likely be cognitive delays as an effect of those very same sensory motor delays. Jongmans, Smits-Engelsman and Schoemaker (2003) tie together learning disorders and behavioral disorders to perceptual and motor learning difficulties. They correlate these conditions, that are neurological functional disconnections secondary to interruptions in motor development, to the DSM-IV classification of developmental coordination disorder (Jongsman, Smits-Engelsman and Schoemaker, 2003).

Does Embryologic Development Go Through Stages Similar To Those of Phylogenic?

The concept of embryologic development being exactly the same as phylogenic was first proposed by Haeckel in the late 1800's in his concept of the law of recapitulation. Haeckel's belief that ontogeny follows phylogeny has been generally accepted as inaccurate since the early 20th century according to the University of California Museum of Paleontology (UCMP, 2005). So there is no expectation that a human embryo starts as larval stage moving to a fish to an amphibian, to a reptile, to a four legged mammal to a primate and finally arriving at a level known as human. Yet the nervous system does develop in stages that are oddly reminiscent of lesser creatures, developing lower levels of the central nervous system like the brain stem and mid brain before the higher or more human levels of the neo-cortex (Carey, 2002; Scheibel, 2002).

Human developmental stages called Carnegies stages, named after the institute which began examining and collecting human embryos at the beginning of the 19th century (Hill, 2004b), are broken down into 23 different stages based on internal or external changes (Hill, 2004c). These stages demonstrate the growth between oocyte on day one through the 60th day after which fetal development begins (Hill, 2004d). The stages show a path that is unique to human although it does present similarities to other species.

The human nervous system as complicated as it is, has humble beginnings. Of the three germinal layers, endoderm, mesoderm and ectoderm, it is the ectoderm that gives birth to the nervous system (Scheibel, 2002). During the development of the nervous system neurons multiply; migrate from their birth site to their developmental destination; connections are made; and structures are formed (Nolte, 2002).

At approximately three weeks of development in response to chemicals released by the mesoderm, the nervous system develops into a neural plate that subsequently forms into a neural tube that will make up the central nervous system and neural crest cells that will make up much of the peripheral nervous system (Nolte, 2002; Felton and Jozefowicz, 2003). It more recently appears that this induction and patterning occurs not only from mesoderm derived guides but also from the ectoderm itself (Stern, 2001).

The central nervous system is focused on here because it is central structures that are involved with learning and behavioral disorders. From the neural tube develops a series of bulges

of which there are three that become primary vesicles: the prosencephalon or forebrain; the mesencephalon also called the midbrain; and the rhombencephalon known as the hindbrain (Nolte, 2002; Felton and Jozefowicz, 2003). These three primary bulges or vesicles will later subdivide and become secondary vesicles.

The proencephalon will eventually become the telencephalon which then becomes the cerebral hemispheres also called the cortex; and the diencephalon which will eventually become the thalamus, hypothalamus, basal ganglia and other neural structures (Nolte, 2002; Felton and Jozefowicz, 2003). The mesencephalon develops further yet remains called the same (mesencephalon); and the rhombencephalon develops into the metacephalon out of which will develop the pons and cerebellum; and the myelencephalon which will later become the medulla (Nolte, 2002; Felton and Jozefowicz, 2003).

Chronologically, beginning at three weeks the motor neurons first appear; four weeks the neural crest cells (peripheral nerves) start to migrate; at five weeks sensory nerves grow into the central nervous system, the basal ganglia, thalamus and hypothalamus begin to grow along with the beginnings of the autonomic nervous system; at 6 and 7 weeks the basal ganglia, cerebellum, optic nerve and the beginnings of the cortex develop; at 8-12 weeks the neurons further proliferate and migrate along with cerebellar and cortical growth; at 12-16 weeks neurons continue to proliferate and migrate and the corpus collosum develops to connect the left and right cortex; and finally at 16-40 weeks many synapses are formed and some neurons develop their myelin sheaths (Nolte, 2002; Felton and Jozefowicz, 2003). At approximately 25-28 weeks the

fetus weighing less than three pounds is viable although full term is considered 40 weeks (A.D.A.M., 2003).

Post Natal: Sign Posts to Normal Development Neurologically

After birth synaptogenesis occurs creating approximately 150% of the number of synapse as adult levels during the first 12 months; this is followed by pruning the unused or redundant synapses (Goswami, 2004). Carey (2002) suggests that only 50% of the neurons created during synaptogenesis will survive to adulthood, with many succumbing to internally generated suicide processes. This pruning appears to occur secondary to the dominance and use of certain pathways and patterns during development.

The Institutes for the Achievement of Human Potential (IAHP, 2003) have developed a series of sign posts for development based on the expected time frames regarding the stage of brain development, motor ability, language capability and manual dexterity. The system profiles slow, average and superior developmental levels in seven stages that cover the average development between birth and age six. Within each stage the slow stage approximates twice the monthly age of the average age which is in turn approximately twice the monthly age as the superior level.

This profile is valuable in understanding how the development of the nervous system and the use of language is intimately related motoricity. This developmental profile which links cognitive ability and motricity appears to be useful for the clinician as well as to the educator for

its early recognition and early intervention value. Interestingly the stages of mobility in this profile are somewhat similar to the evolutionary progress in motricity from mammals to primates and primates to humans as described by Melillo and Leisman (2004). A later version of IAHP profile links visual, auditory and tactile progress to brain development (IAHP, 2005). This profile may be more appropriate for the educator as the three modalities of visual, auditory and kinesthetic are more commonly used in a typical teaching environment.

More familiar to educational readers is Piaget's theory of cognitive development. Piaget created four stages of development that were designed to encompass physical growth and psychological growth of the learner. The stages are for infancy: sensorymotor; for toddler and early childhood: the pre-operational stage; for elementary and early adolescence: the concrete operational stage; and from adolescence into adulthood: the formal operational stage (Huitt and Jummel, 2003). These stages have garnered much more interest and support from the educational and psychological communities than from the neuroscience community upon which the theory is supposedly based.

Development of Gender Specific Neurology

Behavioral and learning disorders are in general more common in boys than girls (Pasteur and Ruben, 2002). The differences in their different neurological development and thus susceptibility to developmental disruptions may explain why this occurs. Differences do exist between men and women neurologically.

Male and female brains are significantly different. For example, total brain variability and volume is generally larger in males, most specifically the cerebrum and superior temporal gyrus; females tend to have larger size of the internal capsule components including the putamen, caudate, hippocampus, amygdale, and globus pallidus (Lange, Giedd, Castellanos, Vaituzis, and Rapoport, 1997). Male nervous systems tend to mature slower than females; this is consistent with both auditory and visual memory tests that demonstrate gender advantage to 6-10 year old girls (Vunotela, Steenari, Carlson, Koivisto, Fjallberg, et al., 2003).

BrainPlace.com (2003), a website hosted by the Amen Clinics describes differences in male and female brains. Dr. Amen is a medical physician who is board certified in psychiatry and imaging. His website states, “men have 4% more brain cells than women, and about 100 grams more of brain tissue.” They also state that there are differences in cellular connections. They describe, “even though a man has more brain cells, it is reported that women have more dendritic connections between brain cells.” Finally the Amen website describes that women have a larger deep limbic system than males do.

Mitchell, Free, Merschhemke, Lemieux, Sisodiya et al. (2003, p. 410) state that corpus callosal cross-section area was larger in women. Gur, Gunning-Dixon, Bilker and Gur (2002, p. 998) found that women have larger orbital frontal cortices than men. They also describe that there is a significant difference in the ratio of cortex to amygdale, the larger difference in women they postulate is devoted to modulating emotions. Cahill (2003, p. 163) found that “Both the hemispheric involvement of the human amygdale in memory for emotionally arousing events

and the impairing effect of B-andrenergic blocade on memory for emotional events exhibit pronounced sex-related differences.”

Another related concern to gender development of the nervous system is the effect of sex specific hormones on the production of dopamine receptors. Anderson, Thompson, Krenzel and Teicher (2002, p. 683) state that “Males, but not females, over produce dopamine receptors in the striatum...” They also point out that this is followed by an elimination period during young adulthood. Mozley, Gur, Mozley and Gur (2001, p. 1492) describe the significance of dopamine to the subject by describing the learning effects and the structures involved. They say “Sex hormone levels seem to be related to some cognitive abilities, particularly memory.” They later add that “Relationships between dopamine availability in the caudate and putamen and executive and motor functioning were observed in women, but not in men.” This is very significant because it describes a piece of the pathway linking motoricity to learning.

McEwan (1999, p. 7128) says, “For the most part, brain sex differences are thought to arise in perinatal development through the actions of testosterone secreted by the developing testes, and these sex differences are believed to persist in the absence of gonadal hormones in adult life, very much like the basic plan of the male and female reproductive tracts, which are also developmentally determined.” This is supported by Shors and Miesegaes (2002, p. 13955) who state state, “Exposure to sex hormones prenatally and early in development organizes the brain for many behavior patterns that manifest throughout adulthood.” “Early work in this area, primarily in rats, focused on the effects of sex steroid hormones on brain morphology during

critical periods of early development,” (Goldstein, Seidman, Horton, Nikos, Kennedy et al. 2001, p.490).

Many of these studies were on rodents and other mammals and yet it appears to be reasonable to transfer what has been learned to humans. Goldstein, Seidman, Horton, Nikos, Kennedy et al's. (2001, p.490) paper confirms most animal studies in sexual dimorphism to be validated in live humans via magnetic resonance imaging. He reports that “A permutation test showed that, compared to other brain areas assessed in this study, there was greater sexual dimorphism among brain areas that are homologous with those identified in animal studies showing greater levels of sex steroid receptors during critical periods of brain development.” Later he states, “Postmortem work in humans also identified sexual dimorphisms in brain regions involved in the neural control of sexual and maternal behavior and gonadotropic secretion.” Completing his argument with the statement “With the advent of magnetic resonance imaging (MRI) to examine in vivo brain anatomy an increased acceptance of the idea of sex differences in the human brain, there are a growing number of in vivo studies on sexual dimorphisms in adults.” Thus it would appear to be reasonable that the dimorphic changes seen in males and females in mammals and humans are similar.

These changes include amygdale, hippocampus, hypothalamus, corpus collosum, anterior commissure and posterior temporal cortex, (McEwan 1999). Many of these structures deal with two important areas of behavior, learning and emotions. Lemaire, Loehl, Le Moal, and Abrous

(2000) found that prenatal stress can cause a reduction in neurogenesis of the hippocampus causing a lifetime of impairment.

According to Michael, Zumpe and Bonsall (1992), “It is thought that testosterone acts on the brain via estrogen and androgen captors to organize the development of sexually dimorphic (gender unique) neural structures that underlie sex differences in behavior.” If so then a change in testosterone or the precursors that would affect testosterone availability would have an effect on development of brain and thus learning along with emotion maturity. Because of the importance in the relationship of these structures to learning and the developing brain, the literature is rich with information about the effects of maternal and environmental stressors on the developing fetus’ nervous system.

Dawson, Cheung and Lau (1975) found that in animals masculinized females (those given injections of testosterone), had higher spatial learning (a male trait) and that feminized males spatial and other activity roles were also reversed. Fitch, Berrebi, Cowell, Schrott and Denenberg (1990) found that corpus collosum changes could be induced by altering testosterone during early developmental stages. Estrogen blockers created defeminizing effects in females. They concluded that the presence of estrogen was necessary for the feminization of cortical tissue in the female brain. Guilamon, Vaencia, Cales and Segovia (1986) also found that they could reverse learning abilities related to sexual dimorphism with altering testosterone. The literature is rich with evidence of sexual dimorphic (gender based) changes in learning and behavior.

While the link of motricity and cognition through phylogenic, embryological, fetal, postnatal, and through sexual dimorphic development may be theoretical, what we know about human anatomy is not. The link of cognitive performance and the development of the sensory-motor systems, becomes more certain when it is recognized that the motor and cognitive functions use the same anatomy. It is the development or the disruption of the development of these shared anatomical structures that determines that explains how learning may be affected.

An overview of the Anatomical Structures and their Function

Changes in function and anatomical differences have been linked to a variety of learning and behavioral disorders. Magnetic resonance imaging has been used to describe neurological origins of attention deficit hyperactivity disorder (AD/HD), obsessive compulsive disorder (OCD), Tourette syndrome (TS), schizophrenia, depression, autism and anorexia (Durston, Hulshoff Pol, Casey, Giedd, Buitelaar, et al., 2001). Neuroimaging studies regularly demonstrate changes in volume of both gray and white matter of the brain in many learning and behavioral disorders and also changes in the metabolic function of those regions (Nakamura, 2003). The following is a brief overview of what has been found via imaging in school aged children.

Areas of involvement.

The areas involved in learning and behavioral disorders include the cerebellum, basal ganglia, thalamus and the frontal lobes (Alpiner, 2003). Executive function is suspected of being a key a variety of neurodevelopmental disorders (Hughes and Graham, 2002). The prefrontal

cortex controls the executive functions of the brain and is broken down into three areas of involvement that are commonly affected.

Specific cortical areas involved include the dorsal lateral prefrontal lobes that give the ability to attend and pay attention; the orbital frontal lobe that suppresses or dilutes emotional outbursts from the amygdale; and the ventromedial prefrontal lobe that controls motivation (Melillo, 2004). Attention is the core to our awareness of the world and determines how we respond to it; it also affects our literacy and perhaps even our IQ (Posner and Rothbart, 2004). Disruptions in executive function have been connected to AD/HD (Gioia, Isquith, Kenworthy, and Barton, 2002).

Attentional networks include three functions: orientation that is modulated by the neurotransmitter acetylcholine in the superior parietal, temporal parietal junction, frontal eye fields and the superior colliculus; alertness governed by norepinephrine in the locus coruleus, right frontal and parietal cortex; and the executive attention areas sensitive to dopamine including anterior cingulate, lateral ventral prefrontal cortex, and the basal ganglia (Posner and Rothbart, 2004). Casey, Gied, Vaus, Vaituziz, Hamburger, et al. (1997) also found the anterior cingulate and the right hemisphere linked to attention.

The temporal lobes have extensive connections with the amygdale and the hippocampus (Giedd, Vaituzis, Hamburger, Lange, Rajapakse, et al., 1996). These structures combine to serve functionally with emotions, memory and language (Giedd, Vaituzis, Hamburger, Lange,

Rajapakse, et al., 1996). Abnormalities of the temporal lobe include autism, affective disorders, and schizophrenia; these conditions are increasingly being looked at as developmental disorders of the nervous system (Giedd, Vaituzis, Hamburger, Lange, Rajapakse, et al., 1996).

Pre-Frontal Cortex

Theory of Mind is used as a hypothesis in the understanding of autistic learners (Edelson, 1995). Wikipedia (2005) describes the phrase Theory of Mind, used in this context, as having the capacity to recognize that others have their own thoughts, reasons, and desires. Theory of Mind has been linked to the frontal lobes capacity for self awareness and it is suggested that the disruption of frontal function could have both congenital and injury origins (Stuss and Anderson, 2004).

There have been links made connecting emotional behavior to cortical hemispheres. The more cortical capacity contributes to better coordination of multiple systems that are involved in emotional processing via parietal involvement in recognizing emotional triggers and anterior systems processing that recognition (Pollak and Wismer Fies, 2001). A lack of neurological development can hinder this processing. For example, obsessive compulsive disorder has been linked to the abnormal function of the ventral medial portion of the prefrontal cortex (Cummins and Ninan, 2002). AD/HD, OCD, depression and schizophrenia have all been linked to abnormal function of the prefrontal lobe (Casey, Cohen, Jezzard, Turner, Noll, et al., 1995).

Hemispheric specialization.

The development of more human capacities and behaviors rely largely on the specialization of function between the two cortical hemispheres. For example, the right hemisphere is associated with visuo-spatial and attention activities, while the left is associated more with phonological storage and rehearsal (Kwon, Reiss and Menon, 2002). These systems develop concurrently together during childhood and adolescence (Kwon, Reiss and Menon, 2002). Another example of specialization is how the left hemisphere is linked to approach behaviors while the right hemisphere is associated with withdrawal behaviors (Olko and Turkewitz, 2001). One way that this has been demonstrated is with the use of olfactory (smell) sensation in infants. In their study the researchers isolated hemispheres taking advantage of the fact that olfactory sensation has relatively direct access to the emotional centers of the brain and arrives on the same side of the brain as the nostril projecting the smell; this combined with the fact that the transmission between the two hemispheres is inefficient in infants allowed the infants to be evaluated as split brain test subjects. (Olko and Turkewitz, 2001).

Task specialization appears to be differently wired for efficiency. For example at the neuron level, distal synapses between neurons may lose out to more proximal neuronal synapses (Steratt and Van Ooyen, 2002). On a more macro scale, simple tasks are more efficiently handled by a single hemisphere where as more complex tasks are more efficient when tasks are distributed across the hemispheres (Monaghan and Pollman, 2003). An example of this includes timing tests which are also measures of synchronicity of the different brain components as they work together. Helmuth and Ivry (1996) found that on the side of cerebellar weakness the timing

when finger tapping was performed slower than on the side of the healthy cerebellum; yet when both hands fingers tapped the two sides synched up with and maintained pace with the healthy cerebellum.

This synchronicity and balance of the two cortical hemispheres is necessary. In a study related to hostile behavior and grip strength it was found that right hemisphere disorders were associated with both weakness of the right grip strength and with hostile behavior (Demaree, Higgins, Williamson, and Harrison, 2002). This study points to the link between motor function and behavioral controls. Another example of problems from hemispheric asymmetries is neglect (Monaghan and Shillcock, 2004). Neglects demonstrate a deficient processing of stimulus in the deficient field. Neglects can be visual or other sensory input. The thalamus is thought to provide an internal clock designed to maintain synchronicity of the brain (Llinas, 2001; Melillo, 2004).

Corpus callosum.

Between the hemispheres resides the corpus callosum. It is the major conduit between the hemispheres (Giedd, Rumsey, Castellanos, Rajapakse, Kaysen, et al., 1996); and is made up of 200 million fibers (Rajapakse, Gied, Rumsey, Vaituzis, Hamburger, et al., 1996). Processing speed can be greatly affected by injuries to the corpus callosum and quantitative measurement of the corpus callosum is a better measure of psychological effect than measures of ventricular dilation in children (Verger, Junque, Levin, Jurado, Perez-Gomez, et al., 2001). Developmental delays of the corpus callosum would likely cause similar delays in cognitive processing speed. This is because of the necessity for unified functions of cognitive, motor and sensory activities

(Rajapakse, Gied, Rumsey, Vaituzis, Hamburger, et al., 1996). Disconnection syndromes, AD/HD, and schizophrenia have been associated with developmental problems of the corpus collosum (Giedd, Rumsey, Casellanos, Rajapakse, Kaysen, et al., 1996). The corpus collosum has also been implicated in dyslexia and other learning disorders (Giedd, Snell, Lange, Rajapaskse, Casey, et al., 1996). The corpus collosum has been found to be 40% smaller in depressed patients and is associated with decreased glial cells (Ongur and Price, 2000).

Amygdala.

The amygdale is also tied to the theory of the mind and it has been pointed out that early injury to the amygdale is associated with inappropriate language and that later injury to the amygdale was not; additionally there is belief that the amygdale is involved in developmental impairments including autism (Shaw, Lawrence, Radbourne, Bramham, Polkey, et al. (2004). Fear and anxiety have been linked to the amygdale and the limbic system and may affect learning (Jensen, 2000; Cummins and Ninan, 2002). When processed contextually fear has also been linked to the hippocampus (Cummins and Ninan, 2002).

Internal capsule.

The caudate nucleus and the basal ganglia have been linked to attention deficit hyperactivity disorder (AD/HD), obsessive compulsive disorder (OCD), Tourette syndrome, and schizophrenia (Durstun, Hulshoff Pol, Casey, Giedd, Buitelaar, et al., 2001). The fact that movement itself is governed by these structures and that hyperactivity and the tics of Tourette syndrome are in themselves' movement disorders, makes the connections between these

conditions more obvious. The connections are made to and from the internal capsule to higher and lower levels via the thalamus which serves as distribution center for information up and down the nervous system.

Cerebellum.

The cerebellum plays a very important role in motor and non-motor activities. It has connections with many parts of the nervous system providing both feed forward and feed back information. Specific areas of connection include: hypothalamus; limbic and paralimbic regions; septal region; hippocampus; cingulate gyrus; prefrontal; posterior; parietal; temporal; parahippocampal; and cingulated cortices (Schmahmann, 2001). The cerebellum through the mentioned connections affects the autonomic nervous system, the limbic (emotional) systems and the cognitive regions of the brain, provides planning and correction of movement as well as cognitive correlates to movement. At least one subtype of dyslexia has been associated with cerebellar dysfunction (Francks, Fisher, Marlow, McPhie, Taylor, et al. 2003).

Evaluation of How Delays or Incomplete Development Could affect the Learning Process.

Disorders of the nervous system may develop differently because of the cause, area disrupted or timing of the disruption of development. This can occur because the components of the brain grow at different rates. Thus it is seen that nature and nurture share in the quality of neurological development of a learner.

Genetic

Genes can affect natural propensity with normal development or susceptibility in abnormal development. First degree relatives have a five fold increase in the likelihood of developing AD/HD (Vaidya, Austin, Kirkorian, Ridlehuber, Desmond, et al., 1998). The dopamine D4 receptor gene has been associated to novelty seeking and AD/HD (Lynn, Lubke, Yang, McCracken, McGough, et al., 2005).

Normal development provides a range of possibilities for expression. The genome is responsible for setting down predetermined patterns towards self organization (Bednar and Miikkulainen, 2000). Posner and Rothbart (2004) found variances associated with genetics in activation of the anterior cingulate which is a major factor in the ability to pay attention.

Genetic defects are likely to be problems throughout the brain and they may include three classes: those that affect brain size which represents the number of neurons themselves or the number of synapses between neurons; neural migration; and neurotransmission effecting neurotransmitters or binding processes (Thomas, 2003).

An interesting perspective of an outward measure of the homobox genes in humans is its ties of brain development to cranial facial characteristics and handedness. Specifically it is suggested that brain growth is a good indicator of dental development and that dental development may be prolonged due to prolonged brain development; thus linking facial structure to cognitive capacity (Dayi, Gungormus, Okuyan, and Tan, 2002).

Abnormal development of the hind brain can be caused by defective genes resulting in disruption of the normal crossing of the tracts leading from the cortex down and tracts related to the cerebellum (cortical spinal and superior cerebellar), causing a condition known as Joubert syndrome (Parisi, Pinter, Glass, Field, Maria, et al., 2004).

Inutero Complications

Maternal derived abnormalities in neural development can occur from fetal alcohol syndrome; rubella viral infection; folate deficiency causing neural tube defects; prescription drugs such as Thalidomide affecting limb development and maternal thyroid conditions (Hill, 2004a). While there is vulnerability at all phases of development, Felton and Jozewicz (2003) describe the third and fifth weeks as particularly sensitive to toxic, developmental, vascular and other insults and Scheibel (2002) describes problems that can occur during the neuronal migration process including schizophrenia, epilepsy and dyslexia. Carey (2002) suggests that this migration period has multiple mechanisms that must occur including the recognition of the correct path to follow. If this migration process is disrupted, it may lead to mental retardation and epilepsy (Carey, 2002).

Stress related hormonal changes.

Maternal stress causes prenatal changes in fetal brain development, per Lemaire, Koehl, Le Moal and Abrous (2000, p. 11032), who say “Indeed, prenatal stress results in an enhanced production of stress hormones by the mother during critical periods of fetal brain development

and provokes a definitely longer corticosterone response to stress in offspring associated with a reduction in the number of hippocampal corticosteroid receptors.” Schors, Pickett, Wood and Pacynski (1999) demonstrated that stress exposure immediately and persistently changes the estradiol and glucocorticoid in female rats. Their study also demonstrated that these stresses also reduced associative learning. This is significant because it demonstrates that changes in maternal stress hormone, creates change in estrogens that will affect testosterone in the developing fetus. These changes in maternal hormones have documented changes in offspring. Kerchner and Ward (1992) also found that maternal stress created incomplete masculinization of neurology in prenatally stressed rats. The changes included areas of executive function residing in the medial preoptic area of the frontal lobe.

Ischemia

Even with good genetics, there can be complications during embryologic and fetal development. For example, in a condition called intrauterine growth restriction (IUGR) there is decreased blood supply arriving to the placenta (Scherjon, Briet, Oosting, and Kok, 2000). IUGR may cause adaptive responses that while designed for survival can cause decreased cognitive ability later in life including lowered IQ and a significant decrease in brain gray matter (Scherjon, Briet, Oosting, and Kok, 2000). Ischemia alone has been linked to cerebral palsy (Volpe, 2003).

Nutritional deficiencies

Nutritional deficiencies such as folic acid (Nolte, 2002) can cause a variety of defects related to the neural tube. Postnatal deficiencies in B vitamins and omega-3 fatty acids have been associated with symptoms of AD/HD (White, 2003). Diets too high in refined sugars have also been associated with AD/HD in a small percentage of cases (White, 2003).

Bipolar disorder is frequently comorbid with AD/HD and has been associated with genetic and environmental induced neurological causes (Kidd, 2004). Nutritional support through the use of omega-3 fatty acids, DHA, EPA, vitamin C, vitamin E, B vitamins (B3, B6, B12 and folic acid) along with the amino acids L-tryptophan and 5-hydroxytryptophan have been found helpful in treating bipolar disorder (Kidd, 2004). It is conceivable that maternal deficiencies may have a negative effect when combined with genetic predisposition.

Toxins.

There are times where the developing nervous system is more susceptible to toxins than others. According to the Canadian Association of Physicians for the Environment (CAPE) one time is weeks three through five post conception when the early structures are being developed (CAPE, 2000a). Another time includes weeks three through five and goes all the way up to six weeks post birth; this is because the blood brain barrier that provides partial protection to the brain does not develop until approximately six months after birth (CAPE, 2000a). Thus there is increased vulnerability during this time.

Toxins can include solvents, pesticides, persistent organic pollutants and heavy metals (CAPE, 2000b). White (2003) adds molds, fungi, food additives and other sensitivities to the classification of toxins as a cause of neurobiological developmental disorders. The following is a brief summary of how toxins can be disrupting to the developing nervous system, causing the learning process to be affected.

Maternal vices.

Some toxins and stressors on neurological development are related to the external environment. Other stressors are internal and include alcohol which McGivern, Raum, Salido and Redei (1988) found to have changes in stimulation of testosterone due to prenatal exposure. Environmental stressors that have been related changes in the development of sexual dimorphism include alcohol, (Hard, Dahlgren, Engel, Larsson, Liljequist et al. 1984) who found that prenatal exposure to alcohol lowers testosterone. Cocaine has also been found to lower estrogen and causing feminizing effects in males and defeminization in females brain sexual differentiation by Raum, McGivern, Peterson, Shryne and Gorski, (1990). These changes in gender specific neurological development may cause learning and behavioral difficulties later because of the disruption in normal development of the brain.

Fetal alcohol syndrome and the effects of other recreational drugs have become common knowledge to have detrimental effects in the developing nervous system, yet few are fully aware of the risks of smoking. Maternal smoking is associated with increasing the risk of physical aggression in their children's behavior (Tremblay, Nagin, Seguin, Zoccolillo, Zelazo, et al.,

2004). Antisocial behavior, conduct disorder and delinquency are also associated with maternal smoking (Wakschlag, Pickett, Cook, Benowitz, and Leventhal, 2002). Smoking can also be a source of lead poisoning (CAPE, 2000c).

Lead and other chemicals.

Even low levels of exposure to lead has been found to cause decreased achievement in school, lowered IQ and behavioral difficulties (CAPE, 2000c). Lead paint is often considered a source of lead exposure but it may also be found in lead water pipes, lead solder with copper plumbing, canned foods, crayons, candlewicks, mini-blinds and plastic toys (CAPE, 2000c). Lead poisoning has been linked to AD/HD (CAPE, 2000c).

Other toxins are in the category of persistent organic pollutants. These include halides, pesticides, solvents and other chemicals such as PCB's, DDT, hexochlorobenzines, dioxins and others (CAPE, 2000d). If the mother has an exposure to these chemicals whether environmental or ingested via fish or other foods, they may cross the placenta and alter normal development of the nervous system inutero (CAPE, 2000d). Lead and PCB's have not only been tied to children's brains but also to adult disorders such as Parkinson's disease (Weiss and Landrigan, 2000).

One chemical that may effect up to 25% of mothers is phthalate (Swan as cited by Kristiansen, 2005). The chemical which is often in plastics can cause changes in male reproductive organs with very little maternal exposure (Swan as cited by Kristiansen, 2005). The

change in male reproductive organs can cause changes in testosterone which can affect growth of the brain dimorphically as described earlier.

Mercury, amonia and iatrogenetic causes.

Mercury is a very dangerous and toxic substance to the nervous system. Mercury can come from a variety of possible sources. Industrial waste, thermometers, thermostat switches, florescent lightbulbs, batteries, fish from polluted waters, and amalgam from dental fillings have all been suggested as potential sources. Of particular concern for many is that health care uses of mercury could cause iatrogenetic problems, meaning the treatment is the cause of a new problem. If mercury is a factor it may be the accumulative effect from a variety of sources rather than from a single source.

There has been much floated around in the lay media regarding mercury in vaccines as a cause for learning and behavioral disorders like autism (Vlahos, 2005). There are lay articles that imply conspiracy on the part of the pharmaceutical industry, FDA and CDC (Kennedy, 2005) and there are also studies in the peer reviewed literature that express some concern about the use of mercury in Thimerosal used as a preservative in vaccines (Nelson and Bauman, 2003). Yazbak (2003, p. 107) said “Emerging evidence suggests some relationship between MMR and thimerosal-containing vaccines and regressive autism.” The contrary opinion also exists. It has also been suggested that the link between vaccinations and autism does not exist in both the lay literature (Vlahos, 2005b) and in the peer reviewed literature (Nelson and Bauman, 2003).

Ammonia has also been associated with being an iatrogenic cause of autism (Fallon, 2005). It is suggested that the manufacturing process of Augmentin™, a common antibiotic for pediatric ear infections, uses ammonia via urea in the fermentation process and the residues of this ammonia when ingested may cause inflammation or other changes in susceptible developing brains (Fallon, 2005). The FDA has added convulsions as a potential neurological side effect to Augmentin™ in 1998 but autism is still not listed as a possible side effect of its use (MEDWATCH, 1998).

Immunological.

Inflammation is an immunological response by the body. It frequently occurs following injury or infection. Brain inflammation has been linked as a cause of autism (Varguas, Nascimbene, Krishnan, Zimmerman, and Pardo, 2005). Neuroinflammation has been linked to a wide variety of neurological disorders (Mrak and Griffen, 2004).

Infectious agents and the body's reaction to perceived attacks can alter neurological development. Cummins and Ninan (2002) found that antibodies made by the basal ganglia in response to beta hemolytic streptococcal bacteria can cause obsessive compulsive disorder in genetically vulnerable populations. Anxiety in children has also been linked to other immunological responses. Allergies like hay fever can also trigger alterations in temperament in children (Cummins and Ninan, 2002). Lesions related to white matter development have been linked to cerebral palsy secondary to infection which apparently increases the vulnerability (Volpe, 2003).

Stress and sexual maturation.

Similarities in incomplete neurological development as seen in sexual dimorphism and AD/HD are significant in that they share similar developmental anomalies. As discussed previously, structures that are involved with dimorphic learning seem to be effected. Both seem to be effected by the alteration of maternal cortisol effecting estradiol which effects testosterone development, which then alters the development of the brain from its normal pattern of neurological development. Rice and Barone, (2000, p. 511) tie the components together as they state “Furthermore, various clinical disorders in humans (eg schizophrenia, dyslexia, epilepsy, and autism) may also be the result of interference with normal ontogeny of developmental processes in the nervous system.”

Premature Birth

Premature birth can cause a variety of health concerns. Survival itself can be at stake. Brain development can be interfered with by premature birth (Rivkin, 2003). Rivkin (2003) further points out that complications associated with preterm births can also affect the development of both gray and white matter. These conditions include bronchopulmonary dysplasia, multiple gestation pregnancies, intraventricular hemorrhage, periventricular leukomalacia and treatment with steroids (Rivkin, 2003). Cerebral palsy has been linked to even mild ischemic conditions when concurrent with an infection in premature births (Volpe, 2003).

Developmental Overview

Disruptions in plasticity can occur from a variety of origins in the young learner. Conditions observed by pediatric neurologists include: “neurofibromatosis, tuberous sclerosis, Fragile X syndrome, other inherited forms of mental retardation, cretinism, Coffin-Lowry syndrome, lead poisoning, Rett syndrome, epilepsy, hypoxic-ischemic encephalopathy and cerebral palsy” (Johnston, 2004, p. 73). These disruptions of plasticity called functional disconnections can also cause schizophrenia (Friston, 1996).

Other forms of plasticity may also be tied to developmental difficulties. Dayi, Gungormus, Okuyan and Tan (2002) linked handedness, facial structure and brain development together. Bonvillian, Gershoff, Seal and Richards (2001) found there was a decrease in right handedness in the autistic population, suggesting some neurological developmental tie to the condition.

Myelination.

Myelination is the process of laying down a fatty insulation sleeve around neurons that allow them to conduct messages much faster. Between the ages of 4-18 there is a great deal of myelination of the neurons occurring that is the cause of the increase in white matter while there is simultaneous decreases in gray matter due to synaptic and neuronal pruning (Rajapakse, DeCarli, McLaughlin, Gied, Grain, et al. (1996). The supportive white glial cells outnumber the gray neurons by as much as 10 to one (Giedd, Snell, Lange, Rajapakse, Casey, et al., 1996). This myelination is thought to continue through as many six decades in life (Giedd, Snell, Lange,

Rajapakse, Casey, et al., 1996). Disruption of white matter growth has been linked to AD/HD and may be an underlying cause of a metabolic origin of functional disconnections (Castellanos, Lee, Sharp, Jeffries, Greenstein, et al. 2002). Disruption of myelination has been associated with abnormal cerebellum development which may effect both cognitive and motor ability (Collin, Usiello, Erbs, Mathis and Borelli, 2004).

Cortical lobe maturity.

The various prefrontal lobe regions mature separately (Hooper, Luciana, Conklin, and Yarger, 2004). The parietal lobe peaks in gray matter growth at age 12, the temporal lobe peaks at age 16 and the occipital lobe continues to increase in gray matter up to about age 20 (Giedd, Blumentha, Jeffries, Castellanos, Liu et al., 1999). The associative neocortex and the corpus collosum continue to develop through the third decade of life (Giedd, Snell, Lange, Rajapakse, Casey, et al., 1996).

Schizophrenia is a severe and debilitating disorder of the mind. It has been linked to genetic, environmental and developmental causes (Thompson, Vidal, Giedd, Gochman, Blumental, et al., 2001). It appears to develop in late adolescence with changes visible through imaging starting in the parietal lobes, progressing to the temporal lobes and then the sensory motor areas of the prefrontal cortex with the final changes showing in the dorsolateral frontal lobes and superior temporal gyri (Thompson, Vidal, Giedd, Gochman, Blumental, et al., 2001).

Nurture.

Environmental stimulus or lack of stimulus can create variations in development and these can have significant changes in cognitive function (Goswami, 2004). Proximal environments have a lot of power in determining the outcome of genetic expression (Odom and Worley, 2003). For example, early exposure to television has been linked to attention problems by age 7 (Christakis, Zimmerman, DiGiuseppe, and McCarty, 2004). Pisecco, Wristers, Swank, Silva and Baker (2001, p. 451) link the quality of friendships to learning disorders like ADD/ADHD when they state “there is compelling evidence suggesting that deviant peer environments seem to contribute to the development and the continuation of antisocial behavior.

During development of the nervous system disruptions of normal development can cause problems that affect the ability to learn and control behaviors. This is especially true because of younger learners who are more vulnerable to losses of inhibition due to a lack of maturation of frontal and striatal regions (Durston, Thomas, Yang, Ulug, Zimmerman, et al., 2002). The vulnerability due to lack of maturation is complicated by other factors such as plasticity. In general, young learners have an extra capacity for brain plasticity compared to adults (Johnston, 2004).

There are critical periods in the development of the nervous system where there are vulnerabilities to disruption in the developmental process; and different times of disruption will cause different effects (Carey, 2002). Specific windows of opportunity include development of emotional balance from birth to age 2; math and logic birth through age four; language from

birth through age 10 and music from age three to ten (Afifi and Bergman, 2004). Another example of a biological window of opportunity is the increase in striatal activity of nearly 10-15% in school age children over preschool children (Henriksen, Greisen, and Schneider).

Thoughts are things.

Stress can be a factor hindering learners. It can also be a factor in the developing nervous system. The basal ganglia and the frontal cortex have been linked to maturation secondary to adolescent cognitive input (Durstun, Hulshoff Pol, Casey, Giedd, Buitelaar, et al., 2001). A lack of development of the basal ganglia and prefrontal cortex have been linked to a lack of inhibition as is found in AD/HD (Durstun, Hulshoff Pol, Casey, Giedd, Buitelaar, et al., 2001).

Bruce Lipton (1998), molecular biologist describes that thoughts can literally change genetic code. His belief that the neurotransmitter and other chemical changes that occur in the brain during the thought process stimulates receptors on individual cells that cause alterations in the cell membrane that cause changes intracellularly to unshroud genetic code production including the ability to make new genes to respond to new circumstances or environmental challenges.

Concluding Thoughts

There are various times where the development of the nervous system can be compromised affecting the ability to learn and control behaviors. Genetics play a role and so does the environment throughout a learner's life. Disruptions in normal nervous system can

occur inutero, post-natally, early childhood, adolescence and through out life. This paper has shown that they can occur from toxins, ischemia, vices, stress, and a variety of other causes.

In reviewing the literature it is overwhelming how many minor factors can make such very significant differences in the nervous systems development. Many of these factors can not be controlled or eliminated. In fact, even living in plastic bubble would appear to have risks associated with phthalates. It therefore appears society should put emphasis on the ability to correct these developmental changes to the nervous system to avoid epidemic effects of functional disconnections associated with learning and behavioral disorders.

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