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Adolescent Brain and Cognitive Developments:

Implications for Clinical Assessment in Traumatic Brain Injury

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Abstract

Adolescence is a time of significant physical, social, and emotional developments, accompanied by changes in cognitive and language skills. Underlying these are significant developments in brain structures and functions including changes in cortical and subcortical gray matter and white matter tracts. Among the brain regions that develop during adolescence are areas that are commonly damaged as a result of a traumatic brain injury (TBI). This paper summarizes major brain changes during adolescence and evidence linking maturation of these cognitive and language functions to brain development, placing consideration of both areas of development in the context of rehabilitation for adolescents with TBI.

Keywords

adolescence; brain–behavior relationships; cognitive development

Adolescence spans the developmental period from preadolescence, beginning from about age 9 years, through the end of late adolescence in the early 20s (Table 1). It is a time of significant physical, social, and emotional changes, accompanied by changes in cognitive and language skills. Intervention for adolescents with traumatic brain injury (TBI) must take into account not only important changes at this stage but also the interaction of development and injury effects on brain functions and structures.

In this paper, we review recent research on brain development in adolescence and its relation to cognitive and language developments. The effects of injury are discussed, and the combination of developmental events and injury effects are considered in light of current rehabilitation practices.

ADOLESCENT BRAIN DEVELOPMENT

Recent structural and functional imaging studies have characterized brain development from childhood to early adulthood. The results are summarized in several research reviews (e.g., Durston et al., 2001; Paus et al., 1999, 2001). The consensus of the reviewers is that

although overall brain volume is relatively constant or increases slightly through the teen years, total volume measures mask significant regional changes in gray and white matter distribution (see Table 2 for definitions of neuroanatomical terms).

White matter volume increases linearly with age until adulthood, with a net increase of about 12% from age 4 to 22 years and a greater increase in males than in females (Giedd et al., 1999). Specific white matter volume changes have been described in the internal capsule and arcuate fasciculus bilaterally (Paus et al., 1999), as well as the corpus callosum (Durstun et al., 2001), and the frontal, parietal, and occipital lobes (Sowell, Trauner, Gamst, & Jernigan, 2002).

Paus et al. (1999) postulated that increases in white matter volume reflect an increase in either the diameter or myelination of axons and underlie improvements in fine motor performance, processing of auditory information, and transfer of sensory information between anterior and posterior language areas. In addition, white matter tract size has been correlated with body height (Eyre, Miller, & Ramesh, 1991), which is increasing rapidly during this stage, so, in part, white matter tract changes might be a by-product of overall body growth. Although numerous studies address functional connectivity via white matter tracts in adults, relatively little is known about how changes in connections between brain regions relate to developments in specific cognitive functions (Paus et al., 2001), especially during adolescence.

In contrast to white matter, cortical gray matter increases steadily in volume until adolescence in most regions studied, followed by a decline that continues across the lifespan (Giedd et al., 1999). This trajectory varies by lobe of the brain, with parietal lobe gray matter reaching a peak volume at about age 10 years in girls and 12 years in boys, frontal lobe volume peaking at about age 11 years in girls and 12 years in boys, and a late peak in temporal lobe volume at about age 16 years in adolescent boys and girls (Giedd et al., 1999). The exception is occipital lobe gray matter, which continues to increase in volume throughout adolescence and the early adult years, without evidence of a plateau or decline.

Changes in cortical gray matter are thought to reflect a second wave of overproduction of synapses in the preadolescent years, followed by pruning that may be related to environmental input (Giedd et al., 1999). Thatcher (1997) found that these cycles of synaptic overproduction and pruning were associated with an increase in the synchrony of neuronal firing patterns (i.e., increased coherence of brain electrical activity). Thatcher identified three cycles of increasing electroencephalographic coherence in postnatal development, the last of which occurs during early adolescence.

Total subcortical gray matter volume declines through adolescence, with significant decreases in the volume of the thalamus, caudate nucleus, nucleus accumbens, and basomedial diencephalon (Sowell et al., 2002). Subcortical gray matter volume continues to decline at least into the third and fourth decades of life (Sowell, Thompson, Holmes, Jernigan, & Toga, 1999).

Mesial temporal structures such as the hippocampus and amygdala have been found to increase in volume with age in some studies (Durstun et al., 2001) and decrease in others

(Sowell et al., 2002). The difference in findings may be attributable to limitations of anatomical research (Durstun et al., 2001), as the influence of total volume measures mask significant regional changes in gray and white matter distribution.

Although findings of changes in brain morphology through imaging research have increased the awareness of adolescence as a developmental period, it should be noted that there are limitations in imaging research as in all research. These issues include reliability and validity of anatomical and functional measures, bias in participant selection (e.g., exclusion of particular socioeconomic groups), and sample size. In addition, macroscopic measures such as magnetic resonance imaging (MRI) do not reveal the cellular morphology that may be responsible for gross anatomical differences (Durstun et al., 2001). These limitations should be considered when contemplating the application of the research described in the following text to individual adolescents.

Variability in adolescent brain development

As at other stages of development, variability occurs among adolescents in the timing and extent of brain changes. Although the mechanisms of this individual variability are unknown, there are several possible candidates. For example, brain growth is correlated with body growth in humans (Peters et al., 1998) and body growth is highly variable among adolescents, particularly in early adolescence (Steinberg, 1999). This is evident when one considers the range of sizes and shapes among middle-school students. It has been suggested that the diameter of fibers in the corticospinal tract increases as a function of height (Eyre et al., 1991), and this may be true of cortical white matter as well.

Electroencephalographic studies of children and adolescents show age-related increases and sex differences in structural and functional differentiation of the cerebral cortex, particularly in the left hemisphere (Anokhin, Lutzenberger, Nikolaev, & Birbaumer, 2000). Thus, neurophysiological changes are likely to play a role in individual differences. Individual variation in regional neurochemistry and resultant effects on brain structure and function might also contribute to functional heterogeneity, particularly during puberty (Grumbach, 2002; McEwen, 2001; Muneoka, Shirayama, Minabe, & Takigawa, 2002), although, as Cameron (2001) noted, this is a relatively new area of inquiry.

A final possible contributor to within-subject variability is environmental input. This may be particularly true for the prefrontal cortex (PFC). Because of its prolonged developmental trajectory, it is thought that the PFC has the greatest plasticity of any brain structure (Casey, Giedd, & Thomas, 2000). Thus, it may be the most vulnerable to both environmental stimulation and the effects of toxins, hormones, and other internal and external factors (Casey et al., 2000). Perhaps, as a result of these influences, it has been hypothesized that some individuals never attain the distribution of PFC gray and white matter characteristic of most adults (Rabinowicz, 1986).

COGNITIVE AND COMMUNICATION DEVELOPMENTS DURING ADOLESCENCE

Adolescence is a time of significant development in cognition and communication. An overall increase occurs in speed of processing, with a steep trajectory from ages 5 to 11 years followed by a slower rate of improvement from 11 to 18 years (Kail & Ferrer, 2007). Changes also appear in specific aspects of cognition, primarily in functions that depend on the speed of processing and executive functions (EFs). Development in 3 areas of particular relevance to adolescents is discussed next: executive functions, social cognition, and language.

Executive function development

Executive functions have been defined as “supervisory functions” that control other modular cognitive functions (Levin & Hanten, 2005). They include self-control, abstraction, and temporal estimation and sequencing (Lezak, 1982). The coordination of these three core processes under the guidance of goal-directed behavior provides the ability to create a plan and follow that plan through to completion, making corrections as needed and modifying future behavior based on the results. Executive functions are the basis for metacognitive skills such as the ability to self-monitor performance on complex and demanding tasks (e.g., in social interactions), which undergo significant changes during later childhood and adolescence (Hanten, 2004; Steinberg, 2004). Executive functions also underlie functions such as attentional control and processing, which continue to develop throughout adolescence (Crone et al., 2006).

Some evidence suggests that executive function-related functions, such as cognitive flexibility and goal setting, remain relatively stable following rapid preadolescent growth (Anderson, Anderson, Northam, Jacobs & Catroppa, 2001; De Luca et al., 2003). What changes significantly is the functional integration of these components. For example, performance on tests such as the Tower of London, which measure complex problem solving, shows improvements into later adolescence (Asato, Sweeney, & Luna, 2006; De Luca et al., 2003). Overall, adolescence may be characterized as a stage at which qualitative differences in executive functions combine with increased speed and capacity for dealing with multiple, competing concepts and stimuli. The net result is an increase in the ability to achieve complex, integrated thought and action.

Social cognition development

The predominance of social concerns is a defining characteristic of preadolescence and adolescence. Although parents continue to exert an indirect influence at this stage (Brown, Mounts, Lamborn, & Steinberg, 1993), the main focus is on peer social relationships (Blakemore, 2008). By age 10 or 11 years, preadolescents are acutely aware of themselves and others in their social world, and this awareness is associated with a variety of positive social outcomes (Bosacki, 2003).

Many of the skills required to execute social behaviors successfully are included under the umbrella term of *social cognition*, which refers to a set of cognitive processes that are

thought to be specific to social functioning (Schulkin, 2000). Although the specific cognitive functions that are included in social cognition continue to be debated (Beer & Ochsner, 2006), most authors agree that they include, at minimum, the ability to recognize emotions using affective cues, as well as theory of mind (ToM). *Theory of mind* is defined as the ability to make inferences about the mental states of others and use these inferences to interpret others' behaviors (Premack & Woodruff, 1978). The development of ToM is thought to be complete in preadolescence, signaled by the comprehension of faux pas (Bosacki, 2000). A full understanding of faux pas requires the recognition that one's words were inappropriate, given the knowledge and feelings of others, and that this necessitates some form of conversational repair (Baron-Cohen et al., 1999; Bosacki, 2000). Emotion recognition continues to develop into later adolescence (Tonks, Williams, Frampton, Yates, & Slater, 2007), and this might improve social cognition performance as well. Social performance as a whole continues to improve into adulthood, but it is unclear if this reflects changes in social cognition per se or the continued development of cognitive functions that are not specific to social behavior, such as declarative knowledge, metacognition, speed of processing, and working memory. Our understanding of ToM development after early childhood—including when development ends, what aspects change, and how it should be measured—is still in a primitive stage, with much remaining to be learned.

Language development

The preadolescent and adolescent years are characterized by major development in language form, content, and use (Nippold, 1998, 2000). It is during these years that speakers master sophisticated syntactic functions such as appositive constructions, postmodification of noun phrases, perfect and passive tenses, and modals (Scott, 1988). Sentence length and clause subordination also increase (Scott, 1988), and skills emerge in genres such as persuasive writing (Nippold, 2000). With regard to language form, adolescents are developing a “literate lexicon” (Nippold, 1988), which includes the vocabulary they will need in academic and employment settings. While some of these developments appear to reflect declarative knowledge gains (e.g., in vocabulary and knowledge about language), others, such as the use of complex embedded clause structures, the ability to resolve ambiguous sentences, and the comprehension of proverbs in written text, have been linked to improvements in working memory and abstract reasoning (Felser, Marinis, & Clahsen, 2003; Moran, Nippold, & Gillon, 2006). Evidence suggests that improvements in language processes such as metaphor comprehension and inference reflect gains in working memory (Moran et al., 2005), but they also likely reflect improvements in executive functions such as abstraction and cognitive flexibility.

With regard to language use, adolescents are developing the skills required to communicate in the increasingly diverse social, vocational, and educational contexts in which they are expected to interact. They are learning to regulate their own verbal behavior, negotiate, and use language to achieve complex goals (Turkstra, McDonald, & Kaufmann, 1996). Taken together, these skills typically are considered “higher level language functions,” and increasingly they are viewed as reflecting developments in cognitive abilities that are not specific to language, but also to executive functions and social cognition (Blakemore, 2008;

Blakemore & Choudhury, 2006). These cognitive abilities, in turn, appear to depend on environmental mediation over time to mature fully (Klahr, McClelland, & Siegler, 2001).

Relation of brain changes to cognitive changes

Maturation of cognitive, emotional, and behavioral processes has been linked to the observed gray and white matter developments described earlier in this paper (Barnea-Goraly et al., 2005; Paus et al., 1999; Shaw et al., 2006; Sowell, Delis, Stiles, & Jernigan, 2001; Sowell et al., 1999). Spear (2000) increased axon size and myelination improve signal transduction between neurons and improve the networking capability and cross talk among areas of the brain. It is this improved organization of white matter tracts that is believed to underlie the behavioral developments observed in adolescence (Barnea-Goraly et al., 2005), including improvements in functions such as response inhibition, emotional regulation, planning, and organization (Sowell et al., 1999). A few studies have also revealed correlations between specific anatomical changes and improvements in cognitive functions. The most consistent finding is that executive functions are closely linked to the development of the PFC. For example, functional imaging has shown increased activation of dorsolateral PFC with increasing age in children, associated with improvements in working memory task performance (Crone et al., 2006). Activation in this same region has been shown to increase with age on tasks that require response inhibition and switching rules, with continued development into early adolescence (Crone, Zanolie, van Leijenhorst, Westenberg, & Rombouts, 2008). Although these studies suggest that executive functions can be localized to specific subparts of the PFC, several researchers have theorized that the integrity of the entire brain is necessary for efficient executive functions (Anderson, 1998; Spanos et al., 2007), particularly given the presence of executive function impairments in a wide variety of clinical groups. This has been supported by imaging data from adults with TBI, in whom executive function impairments were more correlated with overall white matter loss than with the presence of focal frontal lesions (Kennedy et al., 2009).

Other cognitive functions have been studied using structural and functional imaging techniques. Structural studies in children have provided evidence that changes in cortical thickness are correlated with improvements in visuospatial memory (Sowell et al., 2001) and general intelligence (Shaw et al., 2006). It also has been shown that changes in myelination (measured as white matter volume) are the main predictors of increased processing speed with age (Mabbott, Noseworthy, Bouffet, Laughlin, & Rockel, 2006).

Improvements in social cognition have been linked to changes in what Brothers (1990) referred to as the “social brain,” which includes frontal, medial temporal, and parietal lobe regions in a densely connected network. Kolb, Wilson, and Taylor (1992) observed that improvements in emotion recognition, which occur at about age 10 years and then again at age 14 years, are associated with periods of brain growth spurts identified by Thatcher (1997). Baron-Cohen, Wheelwright, Hill, Raste, and Plumb (2001) observed similar phases of improvement in the ability to read emotion from eyes.

Although research linking specific behaviors to brain regions and networks is in the early stages, the findings to date support the notion that structural changes provide the architecture for specific changes in cognitive abilities. As is discussed later, current research has also

shown how disruption to this architecture can lead to disruption of normal development in adolescent cognition and behavior.

INJURY EFFECTS ON THE BRAIN

Effects of preadolescent TBI on brain structure and function

On the basis of the “Kennard principle” (Kennard, 1940), it was widely accepted until fairly recently that an early brain injury would result in better outcome than a similar acquired lesion in an adult. This idea was challenged earlier by Hebb (as reviewed by Kolb et al., 2000) and has been further challenged with recent findings regarding the trajectory of brain development and factors that influence plasticity (reviewed by Kolb et al., 2000). Specifically, the idea has been reconsidered that all aspects of language and cognition are affected uniformly by an early lesion (Tranel & Eslinger, 2000). Growing evidence indicates that early damage, especially to prefrontal areas, can have drastic consequences for the continued development of brain structures and functions (Jacobs, Harvey, & Anderson, 2007), including effects on the development of personality, moral reasoning, social cognition, and executive functions (Hanten, Bartha, & Levin, 2000; Tranel & Eslinger, 2000). In a few extreme cases (e.g., Anderson, Bechara, Damasio, Tranel, & Damasio, 1999), adolescents with early focal lesions have presented with a profile of behavior described as “acquired sociopathy,” in which moral reasoning had failed to develop. Although the potential effects of TBI on moral reasoning and personality in adults are well documented (see discussion that follows), the link between these functions and frontal lobe injury in a developing system is only beginning to be understood, and the effects of subtler lesions are not well known.

The use of structural and functional imaging techniques in the pediatric population, including adolescents, has provided a framework to investigate the impact of TBI on brain–behavior relationships during this critical period. Structural imaging techniques, including computerized tomography (CT) and MRI, have been used for some time in both the acute diagnosis of TBI (Munson, Schroth, & Ernst, 2006) and attempts to predict longterm outcomes (Bigler, 1999). More recently, functional imaging techniques, such as functional magnetic resonance imaging (fMRI), have emerged as a promising tool for identifying recovery mechanisms that are specific to pediatric TBI (Munson et al., 2006), investigating issues of plasticity, and measuring the impact of behavioral interventions on brain function (Strangman et al., 2005). In theory, the use of imaging technology will also support the exploration of the relationship between brain development and age of injury, although to date this relationship has received relatively little research attention.

Structural imaging studies

The use of structural imaging in TBI, outside of the realm of clinical diagnosis and medical management, has provided a good foundation of information regarding patterns of brain injury (including focal vs. diffuse damage) and injury mechanisms, and their relation to outcome. For example, Wilde et al. (2005) conducted an MRI volumetric study to evaluate brain volume differences between the whole brain and prefrontal, temporal, and posterior regions of the brain of children after moderate to severe TBI. Compared with a control group

that was matched for age, the TBI group had significantly reduced whole-brain volume as well as reduced prefrontal and temporal region tissue volumes, accompanied by an increase in cerebrospinal fluid volume. More detailed analysis of each of these regions revealed differences in both gray and white matter volumes in the superior medial and ventromedial PFC (also found by Berryhill et al., 1995), white matter differences in the lateral PFC, and gray matter, white matter, and cerebrospinal fluid differences in the temporal regions. In this study, the location of the lesion was an important variable in that gray matter loss in the frontal areas was primarily attributed to focal injury, whereas the white matter loss in frontal and temporal regions was related to both diffuse axonal injury and focal lesions. The degree of PFC atrophy, related to either focal or diffuse injury, was related inversely to functional recovery. While recognizing that further research was needed to relate these findings to specific behaviors, the authors speculated that the specific cognitive and behavioral difficulties that follow frontotemporal lesions might result in a decreased adaptive ability, reflecting impairments in executive functions. In light of the differences between gray versus white matter volume changes, the authors noted the need for further consideration of the effects of TBI on the developmental time course of myelination in the frontal and temporal lobes. This highlights the potential impact of TBI in preadolescence on the developing brain and supports the idea that mechanism of injury and site of lesion are important factors in predicting outcome.

In addition to the structural images that are obtained by CT or MRI scans, diffusion tensor imaging (DTI) is a technique that allows an in vivo view of brain connections. Specifically, DTI is an imaging technique that assesses the microstructure of cerebral white matter on the basis of the movement of water molecules and has been used to characterize damage to white matter in a wide variety of clinical disorders, including pediatric TBI (Hanten et al., 2008; Kraus et al., 2007; Wilde et al., 2006). Researchers have used DTI as a tool not only to describe the integrity of white matter but also to explore the relationship between white matter integrity and behavioral performance. A study conducted by Wilde et al. (2006) focused specifically on the corpus callosum. The results reflected those found in the adult TBI literature, that is, children and adolescents with TBI showed decreased integrity of the corpus callosum compared with typical peers as indicated by a decrease in fractional anisotropy (i.e., anisotropic diffusion of water molecules along white matter tracts). This finding was independent of the presence of any overt structural lesion. This measure of the integrity of the corpus callosum correlated significantly with functional outcome as measured by the Glasgow Outcome Scale (Jennett et al., 1981) and with the performance on a measure of reaction time with interference. Similarly, Kraus et al. (2007) found that DTI changes correlated significantly with the performance on measures of executive function, memory, and attention, even in children and teens with mild TBI.

In a different study using DTI, Wilde et al. (2006) examined white matter integrity and postconcussive symptoms in adolescents with mild TBI. This study demonstrated that DTI techniques are better suited to examine clinically meaningful cognitive, somatic, and emotional changes than traditional imaging measures (i.e., CT and MRI). Measures of fractional anisotropy and radial diffusivity (i.e., diffusion of water perpendicular to white matter tracts) along the corpus callosum were found to correlate significantly with postconcussive and emotional distress levels. These findings indicate that compromise to

white matter tracts along the corpus callosum can be associated with mild TBI in adolescents.

In a study that specifically focused on young adolescents (mean age = 13.87 years) at 3 months postinjury, Hanten et al. (2008) found a strong relationship between white matter integrity in the cingulate gyrus bilaterally, dorsolateral PFC bilaterally (although left more than right), and left temporal lobe to performance on the interpersonal negotiation strategies (INS) task, which measures social problem solving. The scores on INS were related to the presence of focal frontal lesions in younger participants but not in older adolescents. These findings speak to both the specific vulnerability of the late-developing frontal lobes and the importance of white matter integrity to cognitive processing, a point that was mentioned previously in this paper when discussing the development of executive functions.

Together, these structural studies provide a foundation for understanding the most likely areas of damage in TBI and how these areas are affected specifically by the injury. Additional well-documented neuropathological changes after TBI include (1) damage to and associated atrophy of the frontal and temporal lobes; (2) diffuse axonal injury and related exvacuo dilation of the ventricles (dilation that is a result of brain tissue loss); (3) decreased volume of the corpus callosum; and (4) generalized cerebral atrophy in the chronic phase, even in the absence of structural findings at the time of the injury (Barkley, Morales, Hayman, & Diaz-Marchan, 2007; and see review in Bigler, 1999). Such structural changes have been observed in both pediatric and adult populations, with adolescents included in both types of studies. Additional structural differences, specifically after moderate to severe TBI, include (1) reduced growth of the corpus callosum 3 years postinjury (Levin et al., 2000); and (2) decreased hippocampal volume, specifically following pediatric TBI (Di Stefano et al., 2000). In addition, Tasker et al. (2006) found that when pediatric TBI was complicated by increased intracranial pressure, there was a disproportionate hippocampal growth reduction 5 years postinjury, which was most notable on the ipsilateral side to the site of impact. These results indicate that there are widespread, yet consistent, areas of structural damage following TBI, providing a foundation from which to consider links between lesion location and performance on tasks that are specific to adolescents.

Functional imaging studies

Although structural imaging does provide vital information, there are instances in which a person with behavioral deficits after TBI does not present with identifiable structural lesions on either a CT scan or an MRI scan. A “normal” structural scan during the acute phase of injury does not rule out subsequent structural or functional damage (Munson et al., 2006). In these instances, functional imaging begins where structural imaging leaves off. *Functional imaging*, as indicated by the term, refers to a group of techniques that provide information about brain function, typically by measuring blood flow, brain electrical activity, or brain chemistry. For the study of adolescents, functional imaging has the added benefit of providing insight beyond what might be possible when pairing structural imaging with performance on behavioral measures, by providing a more complete window into the function of a developing system.

The use of functional imaging techniques has become increasingly common in research on typical populations and has begun to be used to study issues related to plasticity and relearning in clinical populations. In contrast to structural imaging techniques, which have a long history of use in clinical settings, functional imaging currently is used most frequently as a research tool (Ricker & Arenth, 2007). The functional imaging technique that appears most frequently in the pediatric TBI literature is fMRI, which is reported to be a powerful tool for investigating biological models of recovery and rehabilitation (Matthews, Johansen-Berg, & Reddy, 2004). Although functional imaging research in general has grown, few studies have been published on use of this technology in the TBI population, and even fewer studies have used functional imaging to focus on pediatric injury or to consider adolescents as a distinct population. Of the pediatric studies that have been conducted, most have focused primarily on working memory, language, and social cognition.

A case study of working memory in pediatric TBI recovery used a combination of fMRI and behavioral measures to examine recovery of function following brain injury (Williams, Rivera, & Reiss, 2005). In this study, a 9-year-old boy with severe TBI was tested at 30 days and 15 months postinjury on measures of intelligence and behavior and then completed a working memory task during functional imaging. At 30 days postinjury, the fMRI results revealed a significant decrease in areas of brain activation between an easier version of the working memory task versus a more difficult version; however, at the second follow-up visit, the patterns of activation resembled those of typical individuals. These improved patterns of performance were accompanied by improved behavioral performance, demonstrating the possible uses of functional imaging for understanding recovery processes for preadolescents including plasticity. It is important to note, however, that this was a single case study, the results of which are not easily generalizable.

Two functional imaging studies have focused on language skills after pediatric TBI (Chiu Wong et al., 2006; Karunanayaka et al., 2007). Karunanayaka et al. (2007) used fMRI to study patterns of brain activity during a verb generation task and found differences in activation patterns in the perisylvian language zones between young children with TBI ($n = 8$; mean age = 7.9 years) and their peers who were matched for age and sex but who had sustained only orthopedic injuries ($n = 9$; mean age = 7.1 years). In the TBI group, there was a significant association between fMRI results and behavioral measures, such as verbal fluency and Glasgow Coma Scale scores. This association was present even in the absence of focal lesions because more than half of the participants had no evidence of lesions on structural imaging (Karunanayaka, et al., 2007). The second study, conducted by Chiu Wong et al. (2006), used single photon emission computed tomography to study eight pediatric TBI patients 3 years postinjury with scans obtained during a complex discourse task. The results of this study revealed positive correlations between discourse abstraction abilities and amount of right frontal perfusion (blood flow). In addition, increased perfusion to the left frontal regions was associated with decreased discourse abstraction abilities, indicating that a pattern of supportive plasticity-induced change would involve preferential recovery of right frontal perfusion versus maladaptive plasticity-induced change that appeared to be associated with increased left frontal perfusion.

Together, these functional imaging studies provide a foundation to consider issues of plasticity and associations among behavioral performance, patterns of brain activation, and injury severity. Although this is a new area of inquiry and not yet directly applicable to clinical intervention for individual clients, a group picture is beginning to emerge regarding injury in the developing nervous system. Studies that include older children have focused thus far on preadolescents, but the results provide a starting point to understand the potential delayed effects that TBI can have on brain development during adolescence. The limitations of these studies, however, must be addressed before applying specific results to individual patients. To date, no studies have been large enough to explore the combined effects of development and brain injury to show how development, injury severity, and outcome are related to patterns of brain function and recovery of function. Furthermore, no studies have considered adolescents as a unique group.

Because the application of these techniques is so new, there are additional limitations that must be addressed, including issues such as within-age variability and possible interactions of age with sex (Blakemore, 2008; Chiu Wong et al., 2006; DeBellis et al., 2001). Other issues relate to the limited inclusion of individuals from groups such as children in poverty, who are at risk for negative environmental influences on development. There also are limitations that are common in the TBI literature in general, such as the exclusion of individuals with developmental learning disabilities, who are overrepresented in the TBI population and may have a complex interaction of developmentally atypical function and acquired impairments (Donders & Strom, 1997). Growing evidence shows differences in brain structures and functions between children with learning disabilities and their typically developing peers (Holland et al., 2007); however, to the authors' knowledge, only one study (Donders & Strom, 1997) described outcome in children with a combined diagnosis of TBI and learning disability, and this study included only 10 children.

IMPLICATIONS FOR THE ASSESSMENT OF COGNITIVE--COMMUNICATION SKILLS

As discussed, changes in social, emotional, and cognitive functions during adolescence reflect the interaction of neural maturation and environmental factors. Traumatic brain injury clearly has the potential to cause deviations in the expected neurodevelopmental trajectory; this, combined with typical adolescent heterogeneity, variation in the general population in the types of skills that are developing in adolescence (e.g., executive functions), and variability among adolescents in the timing, location, and magnitude of brain damage, pose significant challenges for assessment (Blosser & DePompei, 1994; Ciccia & Turkstra, 2002; Snow & Douglas, 2000; Turkstra, 1999). In short, it can be exceptionally challenging to distinguish "different" from "disordered" when it comes to adolescents. Although brain development in adolescence is a relatively new area of inquiry, the literature reviewed here suggests three factors to consider when assessing adolescents with TBI: (1) the nature of brain and cognitive developments during adolescence (i.e., what to test), (2) the identification of age-appropriate contexts in which to assess performance (i.e., how to test); and (3) the timing of assessment relative to ongoing brain and cognitive developments (i.e., when to test).

What cognitive--communication functions should be assessed?

Most of the aspects of language, executive functions, and social cognition that can be measured reliably with existing standardized tests are those that mature at around the onset of puberty. These include functions such as basic spoken language forms, emotion recognition, and self-regulation in structured environments. As the review of cognitive developments earlier in this paper indicates, adolescence is characterized by further development of skills in integrating and applying basic cognitive, language, and social functions in progressively more complex contexts, and these are much more difficult to measure than are changes in language skills such as vocabulary.

The assessment of adolescent communication ability must address complex skills such as higher level written language skills, which improve, in part, as a function of developments in working memory and complex social cognition (Kamhi et al., 2007; Proctor, Wilson, Sanchez, & Wesley, 2000; Singer, 2007), as well as self-regulation, self-monitoring, and motivation (Singer, Kamhi, Masterson, & Apel, 2007). For this reason, tasks such as homework assignments, peer conversations, and daily scheduling are likely to be more revealing of challenges in students with TBI than scores on standardized language tests. Given the wide variability in what is considered successful, or typical, performance on these tasks, it is critical to have comparison data from peers, which should be possible when authentic curriculum-based contexts are used for assessment and classroom data are available.

How and where should cognitive--communication skills be evaluated?

The limitations of most standardized tests for the assessment of cognitive, language, and social skills in adolescents apply equally to the assessment context, that is, skills related to the integration and application of information must be assessed in contexts in which they will be used. This includes activities and contexts of daily living, including social contexts and real-world academic situations. This will help identify deficits that clinical testing may mask owing to the artificial structure of many clinical tests and the absence of competing stimuli (Lezak, 1982; Sohlberg & Mateer, 2001). By observing students in classroom contexts, clinicians can gain appreciation of the scope of the impact of the injury. It is the convergence of multiple processes, including working memory and response inhibition, as well as judgment of performance and flexibility of applying newly acquired information, that are important for successful communication functioning in everyday life. In other words, the ability to balance the demands of an adolescent life successfully and the ability to meet these demands in facilitative environments with adequate structure are two separate skill sets. This is particularly true for adolescents with TBI because of the high prevalence of executive function impairments in this group and the high risk for failure to advance in age-typical executive function development postinjury. If a new injury has impaired the ability to use previously learned information in novel contexts, then performance on tests of preinjury knowledge in structured contexts is likely to cause teachers and others to overestimate the adolescent's capacity for succeeding in everyday life.

When should cognitive--communication skills be evaluated?

The evidence supporting a reciprocal relationship between brain and behavioral developments during the teen years suggests that ongoing reevaluation is warranted when a child or adolescent sustains a TBI, even if he or she has been discharged from services after meeting goals at one point in development. Increasing environmental demands and expectations for cognitive functioning necessitate ongoing reevaluation of the adolescent's ability to communicate and interact successfully. The environment can influence not only expectations but also actual skill development, as evidenced by the finding that "higher" cognitive skills—including social cognition, executive functions, and metacognitive skills—depend on environmental input over time to mature fully (Klahr et al., 2001).

The protracted developmental time course for the frontal lobes and the high sensitivity of developing neural structures to environmental influences necessitate a shift to a life-span approach to intervention. There is growing recognition that the effects of TBI are revealed at later points in development and that the injury may derail neural development in progress so that negative effects are not fully apparent for months or years afterward (Kolb, Gibb, & Gorny, 2000). Early injury may change not only the capacity for brain development but also the brain's ability to respond to environmental input, which, in turn, may limit future developments. Thus, an adolescent who cannot control his or her attention on a task might fail to develop complex divided attention skills as well as miss learning the information on which he or she is meant to focus. Our approach must take into consideration not only disorder-specific effects but also the complex relationship of neurodevelopmental changes and continuously changing environmental demands. Thus, in planning assessment and treatment, clinicians working with adolescents with TBI must consider existing functions that are affected by the injury as well as functions that are dependent on the development of injured regions in the future.

The research presented here also indicates that adolescence, as a period of natural change, provides a particularly important window of opportunity for intervention. As noted earlier in this paper, preadolescence is characterized by a new wave of synaptogenesis and subsequent pruning. Clinicians may be able to capitalize on this plasticity to focus on the development of complex cognitive skills. At a minimum, the research to date suggests that there are potential risks to *not* treating children who have sustained TBI during their adolescence.

Barriers to providing services in a life-span framework include current service delivery and reimbursement models, which do not readily allow for this type of approach. However, as research begins to expand in the area of adolescent brain development and clinical disorders, it is possible that the evidence would contribute to a paradigm shift in service delivery models and policy changes to support them. The adolescent brain is a work in progress, and the opportunities for intervention at this stage are likely to outweigh the challenges.

FUTURE DIRECTIONS FOR CLINICALLY BASED RESEARCH

It is a very exciting time in the area of clinical intervention for adolescents. The connection of imaging and behavioral research has allowed questions to be asked and answered that previously could not have been addressed (Munson et al., 2006). While the current research

evidence suggests only preliminary recommendations for clinical intervention with individual clients, the results provide an exciting base upon which to ask future clinical questions. Longitudinal studies are needed that combine imaging and behavioral measures over time to increase understanding of the complex interactions among brain development, brain injury, and outcome. A critical need also exists for studies that link brain recovery mechanisms to behavioral outcomes and specific interventions, as currently is being done in studies of adults with neurological disorders (Raymer et al., 2008). The results of this research will arm clinicians with powerful information that truly encompasses the complexities of treating adolescents with TBI.

CONCLUSIONS

The physical developments of adolescence are accompanied by dynamic changes in multiple cognitive, language, and social domains. These include improvements in executive functions, working memory, efficiency of information processing, social cognition, and emotion recognition. When a TBI occurs during the preadolescent and adolescent years, or even earlier, it may affect not only skills that are emerging at that time but also skills that are expected to develop in the future. The impact of injury during adolescence is exacerbated by the type of damage that is most common in TBI, including diffuse axonal injury and reduction in cortical volume and associated brain functions, particularly in the frontal lobes—the very regions that are developing during this stage.

Given the dynamic and protracted nature of both behavioral and brain changes that occur during adolescence, the interconnections of these two aspects of development, and the role that environment plays in development and rehabilitation, one can begin to understand how an injury that interrupts this intricate process can have effects at the time of injury and also many years later. Clinical intervention for adolescents with TBI requires an understanding of the typical trajectory of adolescent brain and cognitive developments and the ability to use this information to inform the “when,” “what,” and “how” of clinical assessment and intervention. In the future, the results of research combining imaging techniques with behavioral approaches have the power to change how clinical services are provided to adolescents with TBI.

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References

- Anderson SW, Bechara A, Damasio H, Tranel D, Damasio AR. Impairment of social and moral behavior related to early damage in human prefrontal cortex. *Nature Neuroscience*. 1999; 2(11): 1032–1037. [PubMed: 10526345]
- Anderson V. Assessing executive functions in children: Biological, psychological, and developmental considerations. *Neuropsychological Rehabilitation*. 1998; 8(3):319–349.
- Anderson V, Anderson P, Northam E, Jacobs R, Catroppa C. Development of executive functions through late childhood and adolescence in an Australian sample. *Developmental Neuropsychology*. 2001; 20:385–406. [PubMed: 11827095]

- Anokhin AP, Lutzenberger W, Nikolaev A, Birbaumer N. Complexity of electrocortical dynamics in children: Developmental aspects. *Developmental Psychobiology*. 2000; 36(1):9–22. [PubMed: 10607357]
- Arnett JJ, Taber S. Adolescence terminable and interminable: When does adolescence end? *Journal of Youth and Adolescence*. 1994; 23(5):517–537.
- Asato MR, Sweeney JA, Luna B. Cognitive processes in the development of TOL performance. *Neuropsychologia*. 2006; 44(12):2259–2269. [PubMed: 16797612]
- Barkley J, Morales D, Hayman LA, Diaz-Marchan P. Static neuroimaging in the evaluation of TBI. In: Zasler N, Katz D, Zafonte R, editors *Brain injury medicine: Principles and practice*. New York: Demos Medical Publishing; 2007. 129–148.
- Barnea-Goraly N, Menon V, Eckert M, Tamm L, Bammer R, Karchemskiy A, et al. White matter development during childhood and adolescence: A cross-sectional diffusion tensor imaging study. *Cerebral Cortex*. 2005; 15(12):1848–1854. [PubMed: 15758200]
- Baron-Cohen S, Ring HA, Wheelwright S, Bullmore EJ, Brammer MJ, Simmons A, Williams SC, et al. Social intelligence in the normal and autistic brain: An fMRI study. *European Journal of Neuroscience*. 1999; 11:1891–898. [PubMed: 10336657]
- Baron-Cohen S, Wheelwright S, Hill J, Raste Y, Plumb I. The “Reading the Mind in the Eyes” Test, Revised Version: A study with normal adults, and adults with Asperger syndrome or high-functioning autism. *Journal of Child Psychology and Psychiatry*. 2001; 42(2):241–251. [PubMed: 11280420]
- Beer JS, Ochsner KN. Social cognition: A multi level analysis. *Brain Research*. 2006; 1079(1):98–105. [PubMed: 16513097]
- Berryhill P, Lilly M, Levin H, Hillman G, Mendelsohn D, Brunder D, et al. Frontal lobe changes after severe diffuse closed head injury in children: A volumetric study of magnetic resonance imaging. *Neurosurgery*. 1995; 37(3):992–400. [PubMed: 8559350]
- Bhatnagar SC. *Neuroscience for the study of communicative disorders*. 3. Baltimore: Lippincott Williams & Wilkins; 2008.
- Bigler ED. Neuroimaging in pediatric traumatic head injury: Diagnostic considerations and relationships to neurobehavioral outcome. *Journal of Head Trauma Rehabilitation*. 1999; 14(4):406–423. [PubMed: 10407213]
- Blakemore, Choudhury S. Development of the adolescent brain: Implications for executive function and social cognition. *Journal of Child Psychology & Psychiatry*. 2006; 47(3):296–312. [PubMed: 16492261]
- Blakemore S-J. The social brain in adolescence. *Nature Reviews Neuroscience*. 2008; 9(4):267–277. [PubMed: 18354399]
- Blakemore S-J, Choudhury S. Development of the adolescent brain: Implications for executive function and social cognition. *Journal of Child Psychology and Psychiatry*. 2006; 47(3):296–312. [PubMed: 16492261]
- Blosser J, DePompei R. *Pediatric traumatic brain injury: Proactive intervention*. San Diego, CA: Singular Publishing; 1994.
- Bosacki SL. Theory of mind and self-concept in preadolescents: Links with gender and language. *Journal of Educational Psychology*. 2000; 92(4):709–717.
- Bosacki SL. Psychological pragmatics in preadolescents: Sociomoral understanding, self-worth, and school behaviour. *Journal of Youth and Adolescence*. 2003; 32(2):141–155.
- Brothers L. The social brain: A project for integrating primate behaviour and neurophysiology in a new domain. *Concepts in Neuroscience*. 1990; 1:27–51.
- Brown BB, Mounts N, Lamborn SD, Steinberg L. Parenting practices and peer group affiliation in adolescence. *Child Development*. 1993; 64(2):467–482. [PubMed: 8477629]
- Cameron JL. Effects of sex hormones on brain development. In: Nelson CA, Luciana M, editors *Handbook of developmental cognitive neuroscience*. Cambridge, MA: The MIT Press; 2001. 59–78.
- Casey BJ, Giedd JN, Thomas KM. Structural and functional brain development and its relation to cognitive development. *Biological Psychology*. 2000; 54(2000):251–257.

- Chiu Wong S, Chapman S, Cook L, Anand R, Gamino J, Devous M Sr. A SPECT study of language and brain reorganization three years after pediatric brain injury. *Progress in Brain Research*. 2006; 157:173–185. [PubMed: 17167907]
- Ciccia AH, Turkstra L. Cohesion, communication burden, & response adequacy in adolescent conversations. *Advances in Speech-Language Pathology*. 2002; 4(1):1–8.
- Crone EA, Wendelken C, Donohue S, van Leijenhorst L, Bunge SA, Smith EE. Neurocognitive development of the ability to manipulate information in working memory. *Proceedings of the National Academy of Sciences of the United States of America*. 2006; 103(24):9315–9320. [PubMed: 16738055]
- Crone EA, Zanolie K, van Leijenhorst L, Westenberg PM, Rombouts SA. Neural mechanisms supporting flexible performance adjustment during development. *Cognitive, Affective & Behavioral Neuroscience*. 2008; 8(2):165–177.
- DeBellis M, Keshavan M, Beers S, Hall J, Frustaci K, Masalehdan A, et al. Sex differences in brain maturation during childhood and adolescence. *Cerebral Cortex*. 2001; 11:552–557. [PubMed: 11375916]
- De Luca CR, Wood SJ, Anderson V, Buchanan J-A, Proffitt TM, Mahony K, et al. Normative data from the Cantab. I: Development of executive function over the lifespan. *Journal of Clinical and Experimental Neuropsychology*. 2003; 25(2):242–254. [PubMed: 12754681]
- Di Stefano G, Bachevalier J, Levin H, Song J, Scheibel R, Fletcher J. Volume of focal brain lesions and hippocampal formation in relation to memory function after closed head injury in children. *Journal of Neurology, Neurosurgery, and Psychiatry*. 2000; 69:210–216.
- Donders J, Strom D. The effect of traumatic brain injury on children with learning disability. *Pediatric Rehabilitation*. 1997; 1(3):179–184. [PubMed: 9689253]
- Durston S, Hulshoff HE, Casey BJ, Giedd JN, Buitelaar JK, van Engeland H. Anatomical MRI of the developing human brain: What have we learned? *American Journal of Psychiatry*. 2001; 40(9): 1012–1020.
- Eyre JA, Miller S, Ramesh V. Constancy of central conduction delays during development in man: Investigation of motor and somatosensory pathways. *Journal of Physiology*. 1991; 434:441. [PubMed: 2023125]
- Felser C, Marinis T, Clahsen H. Children's processing of ambiguous sentences: A study of relative clause attachment. *Language Acquisition*. 2003; 11(3):127–163.
- Giedd JN, Blumenthal J, Jeffries NO, Castellanos FX, Liu H, Zijdenbos A, et al. Brain development during childhood and adolescence: A longitudinal MRI study. *Nature Neuroscience*. 1999; 2(10): 861–863. [PubMed: 10491603]
- Grumbach MM. The neuroendocrinology of puberty revisited. *Hormone Research*. 2002; 57(Suppl. 2): 2–14. [PubMed: 12065920]
- Hanten G, Dennis M, Zhang L, Barnes M, Roberson G, Archibald J, et al. Childhood head injury and metacognitive processes in language and memory. *Developmental Neuropsychology*. 2004; 25:85–106. [PubMed: 14984330]
- Hanten G, Bartha M, Levin H. Metacognition following pediatric traumatic brain injury: A preliminary study. *Developmental Neuropsychology*. 2000; 18(3):383–398. [PubMed: 11385831]
- Hanten G, Wilde E, Menefee D, Li X, Vasquez C, Swank P, et al. Correlates of social problem solving during the first year after traumatic brain injury in children. *Neuropsychology*. 2008; 22(3):357–370. [PubMed: 18444714]
- Holland SK, Vannest J, Mecoli M, Jacola LM, Tillema JM, Karunanayaka PR, et al. Functional MRI of language lateralization during development in children. *International Journal of Audiology*. 2007; 46(9):533–551. [PubMed: 17828669]
- Jacobs R, Harvey A, Anderson VA. Executive function following focal frontal lobe lesions: Impact of timing of lesion on outcome. *Cortex*. 2007; 43(6):13.
- Jennett B, Snoek J, Bond MR, Brooks N. Disability after severe head injury: observations on the use of the Glasgow Outcome Scale. *Journal of Neurology, Neurosurgery, and Psychiatry*. 1981; 44:285–293.
- Kail RV, Ferrer E. Processing speed in childhood and adolescence: Longitudinal models for examining developmental change. *Child Development*. 2007; 78(6):1760–1770. [PubMed: 17988319]

- Kamhi H, Musterson J, Apel K. Clinical Decision Making in Developmental Language Disorders. Baltimore, MD: Brooks Publishing; 2007.
- Karunanayaka P, Holland S, Yuan W, Altaye M, Jones B, Michaud L, et al. Neural substrate differences in language networks and associated language-related behavioral impairments in children with TBI: A preliminary investigation. *NeuroRehabilitation*. 2007; 22(5):355–369. [PubMed: 18162699]
- Kennard MA. Relation of age to motor impairment in man and in subhuman primates. *Archives of Neurology and Psychiatry*. 1940; 44:377–397.
- Kennedy MRT, Wozniak JR, Muetzel RL, Mueller BA, Chiou HH, Pantekoek K, et al. White matter and neurocognitive changes in adults with chronic traumatic brain injury. *Journal of the International Neuropsychological Society*. 2009; 15(1):130–136. [PubMed: 19128536]
- Klahr D, McClelland JL, Siegler RS. Time matters in cognitive development. Mahwah, NJ: Erlbaum; 2001.
- Kolb B, Gibb R, Gorny G. Cortical plasticity and the development of behavior after early frontal cortical injury. *Developmental Neuropsychology*. 2000; 18(3):423–444. [PubMed: 11385833]
- Kolb B, Wilson B, Taylor L. Developmental changes in the recognition and comprehension of facial expression: Implications for frontal lobe function. *Brain Cognition*. 1992; 20(1):74–84. [PubMed: 1389123]
- Kraus MF, Susmaras T, Caughlin BP, Walker CJ, Sweeney JA, Little DM. White matter integrity and cognition in chronic traumatic brain injury: A diffusion tensor imaging study. *Brain: A Journal of Neurology*. 2007; 130(10):2508–2519. [PubMed: 17872928]
- Levin H, Benavidez A, Verger-Maestre K, Perachia N, Song J, Mendelsohn D, et al. Reduction of corpus callosum growth after severe traumatic brain injury in children. *Neurology*. 2000; 54:647–653. [PubMed: 10680798]
- Levin HS, Hanten G. Executive functions after traumatic brain injury in children. *Pediatric Neurology*. 2005; 33(2):79–93. [PubMed: 15876523]
- Lezak MD. The problem of assessing executive functions. *International Journal of Psychology*. 1982; 17:281–297.
- Mabbott DJ, Noseworthy M, Bouffet E, Laughlin S, Rockel C. White matter growth as a mechanism of cognitive development in children. *Neuroimage*. 2006; 33(3):936–946. [PubMed: 16978884]
- Matthews P, Johansen-Berg H, Reddy H. Non-invasive mapping of brain functions and brain recovery: Applying lessons from cognitive neurosciences. *Restorative Neurology and Neurosciences*. 2004; 22:245–260.
- McEwen BS. Estrogen effects on the brain: Multiple sites and molecular mechanisms [Invited review]. *Journal of Applied Physiology*. 2001; 91(6):2785–2801. [PubMed: 11717247]
- Moran CA, Nippold MA, Gillon GT. Working memory and proverb comprehension in adolescents with traumatic brain injury: A preliminary investigation. *Brain Injury*. 2006; 20(4):417–423. [PubMed: 16716987]
- Moran C, cillon G. Intence comprehension of adolescents with traumatic brain injury: A working memory hypothesis. *Brain Injury*. 2005; 19(10):743–751. [PubMed: 16175835]
- Muneoka KT, Shirayama Y, Minabe Y, Takigawa M. Effects of a neurosteroid, progesterone, during the neonatal period on adenosine A1 receptor, dopamine metabolites in the front-parietal cortex and behavioral response in the open field. *Brain Research*. 2002; 956(2):332–338. [PubMed: 12445703]
- Munson S, Schroth E, Ernst M. The role of functional neuroimaging in pediatric brain injury. *Pediatrics*. 2006; 117(4):1372–1381. [PubMed: 16585335]
- Nippold M. Later language development: The School-age and adolescent years. 2. Austin, TX: Pro-Ed; 1998.
- Nippold M. Language development during the adolescent years: Aspects of pragmatics, syntax, and semantics. *Topics in Language Disorders*. 2000; 20(2):15–28.
- Nippold MA. Later language development: Ages nine through nineteen. Boston: College-Hill Press; 1988.

- Paus T, Collins DL, Evans AC, Leonard G, Pike B, Zijdenbos A. Maturation of white matter in the human brain: A review of magnetic resonance imaging studies. *Brain Research Bulletin*. 2001; 54(3):255–266. [PubMed: 11287130]
- Paus T, Zijdenbos A, Worsley K, Collins DL, Blumenthal J, Giedd JN, et al. Structural maturation of neural pathways in children and adolescents: In vivo study. *Science*. 1999; 283:1908–1911. [PubMed: 10082463]
- Peters M, Jancke L, Staiger JF, Schlaug G, Huang Y, Steinmetz H. Unsolved problems in comparing brain sizes in *Homo sapiens*. *Brain and Cognition*. 1998; 37(2):254–285. [PubMed: 9665746]
- Premack D, Woodruff G. Does the chimpanzee have a theory of mind? *Behavioral and Brain Sciences*. 1978; 1(4):515–526.
- Proctor A, Wilson B, Sanchez C, Wesley E. Executive function and verbal working memory in adolescents with closed head injury (CHI). *Brain Injury*. 2000; 14(7):633–647. [PubMed: 10914645]
- Rabinowicz T. The differentiate maturation of the human cerebral cortex. In: Falkner F, Tanner JM, editors *Human growth*. Vol. 3. New York: Plenum; 1986. 97–123.
- Raymer AM, Beeson PM, Holland AL, Maher LM, Martin N, Murray L, et al. Translational research in aphasia: From neuroscience to neurorehabilitation. *Journal of Speech, Language, and Hearing Research*. 2008; 59:S259–S275.
- Ricker J, Arenth P. Functional neuroimaging of TBI. In: Zasler N, Katz D, Zafonte R, editors *Brain injury medicine: Principles and practice*. New York: Demos Medical Publishing; 2007. 149–156.
- Schulkin J. *Roots of social sensibility and neural function*. Cambridge, MA: The MIT Press; 2000.
- Scott CM. Spoken and written syntax. In: Nippold MA, editor *Later language development: Ages nine through nineteen*. Austin, TX: Pro-Ed; 1988. 49–95.
- Shaw P, Greenstein D, Lerch J, Clasen L, Lenroot R, Gogtay N, et al. Intellectual ability and cortical development in children and adolescents. *Nature*. 2006; 440(7084):676–679. [PubMed: 16572172]
- Singer BD, Kamhi AG, Masterson JJ, Apel K. *Clinical decision making in developmental language disorders*. Baltimore: Paul H. Brookes; 2007. Assessment of reading comprehension and written expression in adolescents and adults; 77–98.
- Snow PC, Douglas JM. Conceptual and methodological challenges in discourse assessment with TBI speakers: Towards an understanding [Subject review]. *Brain Injury*. 2000; 14(5):397–415. [PubMed: 10834336]
- Sohlberg M, Mateer C. *Cognitive rehabilitation: An integrative neuropsychological approach*. New York: Guilford Press; 2001.
- Sowell ER, Delis D, Stiles J, Jernigan TL. Improved memory functioning and frontal lobe maturation between childhood and adolescence: A structural MRI study. *Journal of the International Neuropsychological Society*. 2001; 7(3):312–322. [PubMed: 11311032]
- Sowell ER, Thompson PM, Holmes CJ, Jernigan TL, Toga AW. In vivo evidence for post-adolescent brain maturation in frontal and striatal regions. *Nature Neuroscience*. 1999; 2(10):859–861. [PubMed: 10491602]
- Sowell ER, Trauner DA, Gamst A, Jernigan TL. Development of cortical and subcortical brain structures in childhood and adolescence: A structural MRI study. *Developmental Medicine & Child Neurology*. 2002; 44(1):4–16. [PubMed: 11811649]
- Spanos GK, Wilde EA, Bigler ED, Cleavinger HB, Fearing MA, Levin HS, et al. Cerebellar atrophy after moderate-to-severe pediatric traumatic brain injury. *AJNR American Journal of Neuroradiology*. 2007; 28(3):537–542. [PubMed: 17353332]
- Spear LP. The adolescent brain and age-related behavioral manifestations. *Neuroscience & Biobehavioral Reviews*. 2000; 24(4):417–463. [PubMed: 10817843]
- Steinberg L. *Adolescence*. 5. Boston: McGraw-Hill College; 1999.
- Steinberg L. Risk taking in adolescence: What changes, and why? *Annals of the New York Academy of Sciences*. 2004; 1021(1):51–58. [PubMed: 15251873]
- Strangman G, O'Neil-Pirozzi T, Burke D, Cristina D, Goldstein R, Rauch S, et al. Functional neuroimaging and cognitive rehabilitation for people with traumatic brain injury. *American Journal of Physical Medicine and Rehabilitation*. 2005; 84(1):62–75. [PubMed: 15632490]

- Tasker RC. Changes in white matter late after severe traumatic brain injury in childhood. *Developmental Neuroscience*. 2006; 28(4):302–308. [PubMed: 16943653]
- Thatcher RW. Human frontal lobe development: A theory of cyclical cortical reorganization. In: Krasnegor NA, Lyon GR, Goldman-Rakic PS, editors *Development of the prefrontal cortex: Evolution, neurobiology, and behavior*. Toronto, Ontario, Canada: Paul H. Brookes; 1997. 85–113.
- Tonks J, Williams H, Frampton I, Yates P, Slater A. Assessing emotion recognition in 9- to 15-years olds: Preliminary analysis of abilities in reading emotion from faces, voices and eyes. *Brain Injury*. 2007; 21(6):623–629. [PubMed: 17577713]
- Tranel D, Eslinger PJ. Effects of early onset brain injury on the development of cognition and behavior: Introduction to the special issue. *Developmental Neuropsychology*. 2000; 18(3):273–280. [PubMed: 11385827]
- Turkstra LS. Language testing in adolescents with brain injury: A consideration of the CELF-3. *Language, Speech, and Hearing Services in Schools*. 1999; 30:132–140.
- Turkstra LS. Should my shirt be tucked in or left out? The communication context of adolescence. *Aphasiology*. 2000; 14(4):349–346.
- Turkstra LS, McDonald S, Kaufmann PM. Assessment of pragmatic communication skills in adolescents after traumatic brain injury. *Brain Injury*. 1996; 10(5):329–345. [PubMed: 8735664]
- Wilde EA, Chu Z, Bigler ED, Hunter JV, Fearing MA, Hanten G, et al. Diffusion tensor imaging in the corpus callosum in children after moderate to severe traumatic brain injury. *Journal of Neurotrauma*. 2006; 23(10):1412–1426. [PubMed: 17020479]
- Wilde EA, Hunter JV, Newsome MR, Scheibel RS, Bigler ED, Johnson JL, et al. Frontal and temporal morphometric findings on MRI in children after moderate to severe traumatic brain injury. *Journal of Neurotrauma*. 2005; 22(3):333–344. [PubMed: 15785229]
- Williams S, Rivera S, Reiss A. Functional MRI of working memory in paediatric head injury. *Brain Injury*. 2005; 19(7):549–553. [PubMed: 16134743]

Table 1

Stages of adolescence

Stage	Approximate age	Benchmarks	Characteristics
Preadolescence	9–12 years	<ul style="list-style-type: none"> • Age of puberty 	<ul style="list-style-type: none"> • Rapid physical growth; hands and feet growing faster than trunk • Development of independent values and opinions • Inconsistent abstract reasoning • Need for privacy and to be an “insider”
Early adolescence	13–16 years	<ul style="list-style-type: none"> • Typically ends with end of puberty; defined by cessation of bone growth 	<ul style="list-style-type: none"> • Development of secondary sexual characteristics and sexual behavior; associated with focus on physical appearance • Developing self-reliance • Development of metacognitive skills of self-appraisal and consideration of the future
Late adolescence	Traditionally 17–21 years, although end point continues to be debated	<ul style="list-style-type: none"> • End of adolescence determined by a combination of cultural, social, cognitive, and biological attributes; in Western cultures, it may include achievement of financial and emotional independence and independent living, and employment 	<ul style="list-style-type: none"> • Continued development of personal identity, morals, and values, with inconsistent execution • Mastery of independent living skills • High expectations for self-regulation, including responsibility for self-directed learning

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Table 2

Review of neuroanatomical terms

Term	Location/function
Gray matter	<ul style="list-style-type: none"> Regions of the nervous system that contain primarily cell bodies of neurons Examples include the cortex and subcortical structures such as the thalamus or caudate Comprises nuclei and cortical regions responsible for processing and relaying neural signals
White matter	<ul style="list-style-type: none"> Regions of the nervous system that contain primarily the axons of neurons that project to different areas Examples include the internal capsule and the corpus callosum Form fiber tracts (also called bundles, fasciuli, or collosums) that convey information to and from gray matter (nuclei)
Frontal lobe	<ul style="list-style-type: none"> Portion of the cortex anterior to the central sulcus and superior to the lateral fissure Contains regions associated with motor planning, executive functions, regulation of behavior, and syntactic components of language
Prefrontal cortex	<ul style="list-style-type: none"> Portion of the cortex anterior to the primary and association motor cortices Contains the most evolutionarily recent portions of the brain Contains regions associated with emotional processing, rational decision making, working memory, and attentional regulation
Parietal lobe	<ul style="list-style-type: none"> Portion of the cortex that lies posterior to the frontal lobe and anterior to the occipital lobe Contains regions associated with integrating somatosensory and visual information
Corpus callosum	<ul style="list-style-type: none"> White matter tract that connects the two hemispheres at midline Important for relaying information between the two hemispheres
Cingulate gyrus	<ul style="list-style-type: none"> Prominent portion of cortex seen medially of each hemisphere superior to the corpus callosum Portion of the limbic system; associated with emotional processing
Temporal lobe	<ul style="list-style-type: none"> Portion of the cortex inferior to the parietal lobe and anterior to the occipital lobe Contains regions associated with auditory processing, attention to auditory and visual information, language comprehension, and long-term memory
Occipital lobe	<ul style="list-style-type: none"> Portion of the cortex posterior to the parietal and temporal lobes Contains regions associated with visual processing
Internal capsule	<ul style="list-style-type: none"> Large white matter tract that primarily relays motor and sensory information Located between the basal ganglia and the thalamus Large diameter axons pass through this region from the primary motor cortex to the spinal cord
Arcuate fasciculus	<ul style="list-style-type: none"> White matter tract that relays information between Broca's area and Wernike's area Associated with verbal repetition and semantic tasks in the dominant hemisphere
Thalamus	<ul style="list-style-type: none"> Collection of nuclei located subcortically and lateral to the lateral ventricles Important region associated with the relay of sensory information to the cortex
Caudate nucleus	<ul style="list-style-type: none"> A major nucleus of the basal ganglia

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