Chapter 14
Acquired Neurological Disorders and Diseases of Childhood

Though relatively rare compared to neurodevelopmental disorders, acquired neurological disorders and diseases represent some of the more common disorders seen by child clinical neuropsychologists. This chapter uses a transactional neuropsychological approach to review traumatic brain injury in children; exposure to teratogenic agents, including alcohol and cocaine; childhood cancer, and infectious diseases of the CNS, including meningitis and encephalitis. Research into these various disorders and diseases suggests the need for a transactional approach to assessing and treating children with these neurological conditions. Such an approach is particularly appropriate given the complexity of these disorders as well as the long-term effects that are present during and following recovery.

Traumatic Brain Injury

Traumatic brain injury (TBI) is a relatively common occurrence in childhood. There are two types of head injury: closed and open. An open head injury involves an open type of wound and is caused by a missile or some type of object penetrating the skull and entering the brain. These types of injuries are rarer than closed head injuries and will not be discussed in this text in any great detail. For a closed head injury the child's head has struck another surface or is a result of child abuse such as in shaken baby syndrome. For a head trauma to occur it is generally the result of acceleration/deceleration forces with or without impact of the skull. The head injury may be either diffuse or focal. Focal injury is from impact and is localized to a specific region of the brain. Diffuse injury is the result of shearing white matter and gray matter due to the acceleration/deceleration of the brain and is often seen as a result of car accidents or severe falls. In diffuse injury the axons are stretched and distorted and the child often falls into a coma from the neuronal damage. The main structures involved are important for information processing and involve the transfer of information from one area of the brain including the corpus callosum, internal capsule, cerebellum, frontal and temporal lobes. Seizures are not uncommon as a result of head trauma and most children are either carefully monitored following the injury or are placed on antiepileptic medications as a precaution.

Neurobehavioral Sequelae of Head Injury

The neurobehavioral sequelae of head injury may include declines in nonverbal intelligence; visual-motor impairment; attentional and memory deficits; decreases in oral fluency, comprehension, and verbal association; achievement declines in reading, and an increase in psychiatric disorders (Coelho, 2007; Tonks, Williams, Frampton, Yates, & Slater, 2007). Obviously, deficits will vary among children depending on their age at injury and on the nature, type, and severity of injury sustained. The extent to which TBI alters brain development and functional capacity of the CNS depends on a variety of factors, including the age of injury, the etiology and severity of the injury sustained, the neurological

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complications, and the treatment protocol. These factors are reviewed in the following sections.

Incidence

The incidence of children and adolescents experiencing TBI has increased and head injury has become the leading cause of death in those under age 35, with children under 15 years of age with head injuries accounting for over half of the deaths due to trauma (Fletcher, Ewing-Cobbs, Francis, & Levin, 1995). The incidence of TBI is approximately 180 per 100,000 for children and adolescents aged 1–15 years (Langlois, Rutland-Brown, & Thomas, 2004). Of this number 475,000 are children between the ages of 0–14 years, and 160,000 are between 15 and 19 (Jantz & Coulter, 2007). This incidence is significantly higher than deaths from the second leading cause of death, childhood leukemia. For those children who do not die from their injuries, a significant proportion results in learning and behavioral difficulties.

Of those children with severe TBI, 80 percent have educational needs or require modified educational environments two years post-injury (Rao & Lyketsos, 2000). Behavioral difficulties are also relatively prevalent even among those children with mild head injuries (Stavinhoa, 2005). The majority of children admitted alive to the hospital are discharged with a prognosis for good recovery. However, good recovery does not mean full recovery and many of the children have been later found to develop temporary to permanent difficulties in cognition, memory, or physical disability (Semrud-Clikeman, 2001).

Age

The age of the child is closely related to the type of injury sustained. For the youngest children severe head trauma is generally due to child abuse or car accidents. For those between four and 11 the cause is generally due to pedestrian and bike accidents, while for teenagers it is generally due to automobile accidents that involve teenage drivers. For infants and toddlers the most common cause of head trauma is a fall with few long-term consequences. Severe head trauma at this age is generally due to child abuse or automobile injury. For young children through elementary school the most common type of head injury is pedestrian and bicycle accidents. For teenagers injury is generally due to automobile accidents particularly when the teenager is the driver.

Age is an important variable in understanding the sequelae that follow TBI. Younger children show different patterns of recovery and future learning is more impacted due to the incomplete development. An injury that occurs at an early age is generally associated with more significant deficits than one that occurs later on (Morgan, Ward, Murdoch, Kennedy, & Murision, 2003). In this case, the developing brain may be more vulnerable to damage due to the rapid growth spurts that occur during early development. In addition, the early ages are most vulnerable because the child is learning so many new things. With neuronal disruption, such learning is not present and key structures such as the hippocampus and others that are responsible for new learning may be damaged. Research has indicated that damage occurring prior to age one results in significant impairment because there is little that has been previously learned including language. From the ages of 1–5 years of age reorganization of functions and recovery of language ability appear to be more likely. Damage after the age of five is also problematic as the brain is not as plastic as it was earlier and, thus, reorganization is not as readily obtained.

Structures that do not generally develop until later in life may be compromised by early damage and this injury may not be obvious until years later (Baron, 2008). Executive functioning tasks that develop with myelination are particularly vulnerable to damage from head injury. These abilities allow the adolescent to self-monitor behavior, have insight into behavior, and integrate information from simultaneous sources.

Child abuse is the most common reason for head injury in infants with 64% of infant head injuries due to abuse (Starling, Sirotnak, Heisler, & Barnes-Eley, 2007). Injury rates decrease for females during the first 15 years, while incidence rates increase for males between five and 15 years. Children and adolescents may be at risk for TBI as a result of sports activities, including football and soccer.

Nature, Type, and Severity of Injury

The nature, type, and severity of brain injury affect the outcome and long-term sequelae associated with such injury in children. Further, injury type (e.g., falling versus being hit on the head) may produce very different cognitive, behavioral, and neuropsychological impairment, which should be carefully assessed and monitored. The mechanisms of closed head injury involve several factors, including compression of neural tissues, which are pushed together; tension as tissues are torn apart; shearing as tissues slide over other tissue, and skull deformations that change the volume of cerebrospinal fluid (Semrud-Clikeman, 2001).

Brain injury may occur in three basic ways. First, acceleration occurs when a moving object (e.g., baseball bat) makes sudden contact with the skull. This type of injury may result in bruising or contusions in the brainstem, under the corpus callosum, in the cerebellum, or in the occipital lobes. Contre coup is common in these conditions and results in more severe damage in regions opposite the point of contact. McCrea (2008) indicates that pressure waves spread out from the injury site and cause tissue tearing. The frontal lobes are particularly sensitive to this kind of injury because of the bony protrusions in the anterior skull. Second, deceleration occurs when the head is moving faster than a stationary object (e.g., the dashboard of an automobile), causing abrupt deceleration of the skull. Contusions occur at the site of injury, and contre coup may also result as the brain is thrust back against the skull. Occipital impact may cause frontal and temporal involvement. Midbrain injury also may involve temporal lobe injury to the opposite lobe, while impact to the frontal regions is less likely to result in occipital damage because the surface of the posterior skull is smooth (McCrea, 2008). Third, rotations of the neck or head may occur when there is both acceleration and deceleration, and this rotation results in shearing. Although the skull is less rigid in children than in adults, shearing may still cause significant distortions and damage.

There are basically three levels of head injury. The Glasgow Coma Scale (GCS) (Jennett & Teasdale, 1981) is frequently utilized to ascertain the level of head injury. The child's level of consciousness and response is evaluated by this scale and is predictive of recovery and disability level. It assesses nonverbal response to stimuli, motor responses, and verbal responses, and ranges from a score of 3–15. A higher score is desirable. Table 14.1 presents the behaviors assessed by the GCS.

Table 14.1 Glasgow coma scale

Behavior	Points
Eye Opening (E)	
Spontaneous	4
In response to speech	3
In response to pinprick (pain)	2
No response	1
Motor (M)	
Follows commands	6
Can localize pain	5
Withdraws from painful stimulus	4
Abnormal flexion to pain	3
Extensor response to pain	2
No response	1
Verbal (V)	
Oriented	5
Confused conversation	4
Inappropriate words	3
Incomprehensible sounds	2
No response	1

Coma Score = E + M + V
Source: Jennett & Teasdale (1981)

The duration of impaired consciousness is used to gauge severity of head trauma and involves the number of days from the injury until the child is able to follow commands. Severe injuries are generally seen when impaired consciousness exceeds 24 hours. The highest rates of fatality are for those patients with a GCS of eight or less (Donders, 2007). The time required for the child to be able to understand time and spatial orientation as well as memory for prior events [post-traumatic amnesia (PTA)] is another measure of the injury's severity.

Levels of Head Injury

Mild head injury compromises approximately 50–75 percent of all traumatic head injuries (Semrud-Clikeman, 2001). Many of these are not fully evaluated by medical personnel or are dismissed as non-remarkable. Mild head injuries are those that

result in a loss of consciousness, or PTA, for less than one hour and a GCS score of 13–15. Many children have minor blows to the head throughout development that are not considered problematic by most personnel and are not often evaluated. Research has generally not supported long-term neuropsychological deficits as present from mild injury (Anderson & Yeates, 2007). Mild head injuries may be accompanied by headache, lethargy, irritability, withdrawal, and/or lability.

For head injuries where the loss of consciousness or PTA lasts from one to 24 hours, with a GCS score of 9–12, the injury is considered moderate in nature. Headache, memory deficits, and behavioral difficulties persist over time as the child recovers from the head injury. In addition, secondary symptoms such as hematomas and edema (brain swelling) that require surgery are more frequently present than for mild head injury. Early signs include difficulty with problem solving, memory, and attention/concentration which often improve with time (Yeates et al., 2007).

Severe head injury is one with a loss of consciousness or PTA for more than 24 hours and a GCS of 3–8. Medical treatment is often immediate and intensive, and roughly half of the children brought to the emergency room with severe head injuries die (Wade et al., 2008). For those who survive, the deficits are more severe both physically and neuropsychologically. Children with severe head injury remain in the hospital and often have additional injuries.

Recovery for children with severe head injuries is often compromised with intellectual impairment and co-occurring psychiatric disabilities. School achievement is problematic and difficulties are found in naming objects and/or pictures, verbal fluency and writing skills (Yeates et al., 2004). Additional deficits in memory, mathematics, attention, and organization have repeatedly been found with children suffering from severe head injuries (Ayr, Yeates, & Enrile, 2005). The length of the coma is associated with enduring cognitive impairment, and the ability to return to school after a longer coma is associated with poorer outcome (Yeates et al., 2005).

Adults generally regain the skills that will be obtained within 180 days of injury; the likely extent of recovery will be present within 6–9 months of the injury (Lezak, Howieson, & Loring, 2004). For children, recovery from severe TBI can span 5–6 years post-injury with most improvement seen within 2–3 years after injury (Draper, Ponsford, & Schonberger, 2007).

Unilateral Damage

The effects of lateralized damage has been investigated extensively and it has been found that functional loss following injury may be recovered by the intact hemisphere when injury occurs early (Donders, 2007). While the right hemisphere can assume language functions following damage to the left hemisphere, it does so at the expense of reduced right hemisphere (i.e., visual-spatial) functions. Thus, transfer of language occurs primarily when the speech regions of the left hemisphere are involved. In instances where the Broca's area remains intact, the left hemisphere may reorganize rather than transfer language functions to the right hemisphere.

Although the two hemispheres are functionally specialized at birth, both are relatively flexible in their capacity to pick up functions for the hemisphere that has been surgically removed (Lettori et al., 2008). The price of transfer, however, seems to be a loss of higher level abilities and generalized lower intelligence. For example, simple language functions appear intact following left hemispherectomy, but complex language skills (e.g., complex syntax) are compromised. Conversely, right hemispherectomy results in normal language functions and decreased complex visual-spatial skills (e.g., visual organization, perception of mazes) (Fournier, Calverley, Wagner, Poock, & Crossley, 2008). Thus, while both hemispheres can assume functions of the opposite hemisphere if it has been removed early, neither can mediate all of these functions. While these findings support the notion of brain plasticity, Fournier et al. (2008) cite studies indicating that surgical removal of one hemisphere following injury may actually produce fewer problems than when surgical removal is not feasible. It may be that the damaged hemisphere exerts abnormal influence as the intact hemisphere attempts to assume the functions of the damaged hemisphere, an influence that is not possible when the damaged hemisphere is removed.

Transactional Features of TBI

The neuropsychological, academic, and psychosocial sequelae of TBI depend on numerous factors (e.g., age, severity and site of injury). Further, environmental and premorbid status, including IQ level and presence of psychiatric or behavioral problems, is an important factor affecting outcome. Table 14.2 presents a summary of select research findings for children sustaining TBI. This summary suggests that a variety of domains are affected by traumatic injury. Individual children will vary in terms of the specific features and dysfunctions manifested following injury. The various domains are reviewed next.

Genetic Factors

Although traumatic brain injury is not a result of genetic factors, there is some evidence that certain children may be at higher risk for sustaining brain injury. In a discussion of risk factors associated with TBI, Goldstein and Levin (1990) indicate that children who sustain injuries may not be a random group. Preexisting conditions often include hyperactivity and antisocial behavioral problems; developmental learning problems, particularly in young males; reading difficulties, impulsivity, and overactivity (Farmer et al., 2002; Max et al., 2004; Yeates et al., 2005). Preexisting behavioral patterns may increase risk taking behaviors leading to traumatic injuries and exacerbating previous psychiatric diagnoses (Hayman-Abello, Rourke, & Fuerst, 2003; Max, Robertson, & Lansing, 2001).

Prenatal Postnatal Factors

There are no known prenatal factors that predispose a child to TBI, although brain damage can occur during the birth process. Postnatal factors generally are most important, including both child and family characteristics associated with increased rates of TBI in children. The level of violence and child-related homicides appears to be on the rise, and one can only wonder how many children and adolescents sustain TBI as a result of gunshot wounds and physical attacks.

Parental behaviors, avoidable situations such as drinking and driving or not restraining children while driving, may also place children at risk for TBI. However, family socioeconomic status and parental employment history do not appear related to increased rates of TBI, but these variables may increase problems during recovery and exacerbate neuropsychological deficits (Donders, 2007). Child abuse victims do sustain high levels of brain injury, especially among young children (Anderst, 2008).

Neuropsychological Correlates

Neuropsychological correlates usually relate to the major areas that have been damaged. Patterns of neuropsychological performance of children with head injury begin to mimic those of adults when injury is sustained in late childhood or early adolescence (Armstrong, Allen, Donohue, & Mayfield, 2008). Particular issues that arise in severe head injury for children are in the domains of attention, memory, and executive functioning. The following sections discuss neuropsychological domains involved in TBI in more detail.

Intellectual, Perceptual, Memory, and Attentional Functions

Intellectual Functions

Persistent intellectual deficits have been found in children sustaining brain injury with coma status for more than 24 hours (Wood & Rutherford, 2006). Performance IQ is lower than Verbal IQ in children suffering posttraumatic amnesia, particularly with lower Glasgow Coma scores (7 or less) and with severe head injury (Campbell, Kuehn, Richards, Venuevra, & Hutchison, 2004). Specifically, on the WISC lower scores on the processing speed (PS) and the perceptual organization (PO) factors relate to the severity of TBI (Donders, 1997; Donders & Warschausky, 1996). Further study comparing moderate and severe head injury

Table 14.2 Transactional features of traumatic brain injury in children

Genetic
– No genetic linkage
– TBI children may not be random group
  • Hyperactivity
  • Antisocial behavioral problems
  • Reading problems, impulsivity, and hyperactivity

Environmental
– No known prenatal factors
– Birth process may produce brain damage
– Level of violence in environment
– Parental behaviors place child at risk
  • Child abuse
  • Drinking while driving
  • Not restraining child in car

Neuropsychological Correlates
– Patterns depend on site and type of injury.
– Begin to mimic adult patterns in later childhood.
– Mild injury show few NP deficits

Intelligence
– Persistent deficits coma (24 hrs +)
– PIQ >VIQ with amnesia
– Low PIQ with low Glascow (7 or less)
– Less specific cognitive deficits
– Laterlized damage not always clear-cut
– Left hemisphere—language deficits
– Right hemisphere—design deficits
– Laterlization of higher level skills not always predictable
– Laterlization of sensory-perceptual and motor deficits more clear-cut

Perceptual
– Severity of injury
– Timed conditions
– Visual-spatial

Memory
– Common in TBI
– Verbal learning and memory
– Visual-spatial
– Selective reminding
– Memory improves first year
– Verbal learning of new information deficits persist with severe injury
– Even mild injury can affect

Attentional and Executive Functions
– Disinhibition
– Impulsivity
– Attentional deficits
– Excessive verbalization
– Socially inappropriate
– Insensitivity

Academic
– Not well researched
– Recognized as handicapping condition (IDEA, 1990)
– Problems persist after EEGs and neurological exams appear normal
– Language difficulties
– Writing to dictation and copying
– Verbal associations
– Left hemisphere damage—deficits across all areas (injury before 5 years of age)

Psychosocial
– Changes in personality
– Increased psychiatric disorders in severe injury (not mild)

Family
– Discruptive to relationships
– Home environment impacts

found statistically lower scores on the perceptual organization and processing speed factors of the WISC III for the severe group, but not for the mild and moderate groups (Donders & Warschausky, 1996). Moreover, selective impairment on the PO and PS indices was found to be specific to the TBI group, but not to the WISC III standardization sample (Donders, 1996; Hoffman, Donders, & Thompson, 2000).

While personality factors following injury in children are somewhat similar to patterns found in adults, the cognitive deficits appear less specific and not as clearly lateralized (Max et al., 2001). For example, there is only a mild tendency for injury to the left hemisphere to produce language-related deficits, and for right hemisphere injury to produce deficits in memory for designs. Donders and Warschausky (1996) found a distinct pattern on intelligence testing where children scoring poorly on the PO and PS indices showed a disproportionate incidence in the severe head injury group. This group, as a whole, showed diffuse lesions on CT/

MRI with lesions mostly present in the right hemisphere. Because these children experienced more difficulty on tasks that required visual-spatial reasoning (perceptual organization index) and processing speed, it was suggested from these findings that damage to the right hemisphere was related to these difficulties.

IQ abilities also change over time with decreasing IQ seen in children with focal lesions and with those experiencing damage prior to the age of three (Donders, 2007; Semrud-Clikeman, 2001). These differences are present to a lesser degree for older children and adolescents. Gains in ability have been found after recovery and are maintained into adulthood (Campbell et al., 2004). A quantitative MRI study of moderate and severe closed head injury found structural differences in patients with IQs less than 90 with enlarged ventricles and atrophy in the temporal regions (Bigler, Johnson, & Blatter, 1999). Those with higher IQs did not show compromised structures.

Academic and School Adjustment

Children with TBI have difficulties with language and reading, arithmetic calculation, writing, and spelling (Levin, Ewing-Cobbs, & Eisenberg, 1995). Studies have found significant difficulties in reading recognition, spelling and arithmetic scores in children with severe head injury compared to those with mild to moderate TBI (Arroyos-Jurado, Paulsen, Merrell, Lindgren, & Max, 2000; Ewing-Cobbs et al., 2004). Significant long-term effects were found for reading, arithmetic and spelling with the most significant weaknesses present in children with the more severe injury. Impairment was also seen for children with mild/moderate injury—although not to the degree shown in the severe TBI group. Children injured at a younger age showed a deceleration in academic growth for all groups compared to older children. Phonological processing and verbal memory were variably affected in these children. These findings suggest that all children with TBI need intensive and ongoing support for continued academic growth to occur. This support appears particularly crucial for children injured at a younger age as their skills do not show the same rate of growth as those children injured at older ages.

A longitudinal study also found these weaknesses (Ewing-Cobbs, Fletcher, Levin, Iovino, & Miner, 1998). Older children were also found to score more poorly on measures of numerical operations and reading comprehension compared to the younger children. Achievement improved during the initial six-month recovery period with improvement showing less recovery after the first six-month period. After two years, 79 percent of the severely injured children had either been retained in a grade or had received special education services.

Although language deficits are reported to be present in many adults and children with TBI, it appears that receptive language skills are frequently spared following injury while expressive language skills are significantly impacted. For younger children, particularly those injured during the 6–8-year-old period, problems are present in the development of abstract language abilities, written language, and the ability to express complex ideas (Levin et al., 1995). These difficulties with expressive and written language likely disrupt the child's academic progress, particularly in reading and in writing. It is also important to note that when injury occurs prior to the expectation for performance on measures of inferential reading comprehension and higher level writing skills, the child may perform adequately on a usual achievement test, but show a decline in later grades that is indicative of the aforementioned problems. For a child with TBI it is particularly important to conduct serial evaluations in order to assess these skills which are not expected at younger ages, but are crucial for performance at older ages.

Perceptual Functions

Perceptual problems appear related to the severity of injury, particularly under timed conditions, and visual and visual-spatial impairment have been identified in children following injury (Lehnung et al., 2003). Difficulties are found in motor speed and in the ability to process information quickly following severe TBI (Anderson & Catroppa, 2007). Perceptual difficulties are generally studied in relation to memory and cognition rather than separately. Visual-motor deficits are dependent on whether motor areas of the brain and parietal regions are impacted. These regions are less likely

to be injured in TBI, compared to frontal and temporal regions that are related to difficulties with memory, executive functioning, and attention.

Memory Functions

Memory deficits appear to be fairly common in children following TBI (Catroppa, Anderson, Ditchfield, & Coleman, 2008). Verbal learning and verbal memory, and working memory deficits have been reported in children following TBI while visual-spatial memory and immediate memory are not as affected (Anderson & Catroppa, 2007; Lehnung et al., 2003). Visual spatial memory is when a child is asked to recall where something is on a page while immediate memory is similar to that tapped by Digit span. Working memory is generally memory that requires the child to hold something in mind while solving a problem. Working memory and complex auditory-verbal memory (learning of word lists) have consistently been found to be problematic for children with severe TBI (Anderson & Catroppa, 2007; Vakil, Blachstein, Rochberg, & Vardi, 2004). Further study has indicated that memory difficulties continue after 24 months for those children with severe TBI. In addition, pre-injury academic skills and verbal memory ability was highly predictive of academic success after injury (Catroppa & Anderson, 2007).

Attentional and Executive Control Functions

Children who experience TBI have difficulty with attention and executive functioning. In the area of executive functioning particular difficulty is found on measures of problem solving and planning, and processing speed (Brookshire, Levin, Song, & Zhang, 2004). Children with severe head injuries frequently exhibit problems with disinhibition, impulsivity, and problems with working memory compared to mild injury TBI and control children, and these difficulties continue into adulthood (Mangeot, Armstrong, Colvin, Yeates, & Taylor, 2002; Nybo & Koskiniemi, 1999). Children who experienced TBI in preschool and who were studied as adults showed continuing problems with executive functioning. Those who had made improvements in cognitive flexibility were found to show the best outcome vocationally (Nybo, Sainio, & Muller, 2004)

Attentional difficulties, as measured by continuous performance tasks, have also been documented in children sustaining brain damage, and require special education support (Schachar, Levin, Max, Purvis, & Chen, 2004; Vriezen & Pigott, 2000). Disinhibition has been frequently found in children with TBI, particularly with those children with severe head injuries (Wassenberg, Max, Lindgren, & Schatz, 2004). The prevalence of acquired ADHD is seen in 20% and 50% of children with TBI, with higher rates found in those children with severe TBI (Bloom, Levin, & Ewing-Cobbs, 2001; Max et al., 2004). Studies have also found that when a child has premorbid symptoms of ADHD, he/she is more likely to showed acquired ADHD following injury (Bloom et al., 2001; Max et al., 2004). Thus, children with ADHD or several symptoms of ADHD are at higher risk for experiencing TBI and once they have TBI are more likely to have significant problems with attention.

Yeates et al. (2005) studied long-term attentional functioning in children with TBI longitudinally over four years and compared their functioning with children with orthopedic injuries. Findings indicated that 20 percent of the severe TBI group showed ADHD: combined subtype, where 4 percent of the orthopedically injured children did. In addition, premorbid attentional difficulties predicted the level of attentional problems after injury. It was suggested by these authors that the underlying difficulties with executive functioning (working memory, disinhibition) significantly impacted the child's attentional difficulties apart from cognitive factors.

Taken together these findings indicate that attentional issues are an important consideration when evaluating children with TBI. In addition, they indicate that children with premorbid problems with attention are more likely to show significant attentional difficulties after injury. These problems are more pronounced for children with severe TBI. In addition, attentional problems do not appear to improve dramatically with recovery for these children and continue to be an area that is significantly impacted. It is also highly likely that these problems interact with learning difficulties to further complicate recovery and success in school.

Social-Psychological Adjustment

New psychiatric disorders post-injury appear significantly more often in children with severe head injury (in 50% of cases), whereas children with mild head injury have not been found to differ from a control group (Janusz, Kirkwood, Yeates, & Taylor, 2002). Disorders most frequently seen in children with TBI are in externalizing behaviors including ADHD, oppositionality, irritability, and aggression (Bloom et al., 2001; Janusz et al., 2002; Max et al., 2001). While depression and anxiety are seen in adults with TBI, these disorders are much less common in children (Robin, Temkin, & Machamer, 1999).

One of the issues that is most troubling for children with TBI is difficulty in relating to others and social competence issues (Semrud-Clikeman, 2007). The difficulties that are seen with lability and aggression certainly impair the child's ability to form and maintain friendships. In addition, these problems are exacerbated by difficulties these children have in being critical of their own abilities and having insight into their contribution to the social difficulties. This area makes treatment very difficult for the children, particularly as many of them exhibit an aggressive style toward their peers (Poggi et al., 2005). Most studies investigating head injury in children stress the importance of considering premorbid status in order to assess the full impact of injury on the child (Goldstein & Levin, 1990).

Family and Home Factors

TBI can disrupt family interactions and the home environment can have an impact on the recovery process following TBI. Families that have children with moderate and severe TBI frequently experience not only the stress from the injury and anxiety about the child's future, they also experience significant problems with financial and time obligations during the child's recovery. Continuing difficulties with the child's behavior and adjustment following TBI have been linked to familial functioning, family cohesion, and increased divorce rates (Wade, Taylor, Drotar, Stancin, & Yeates, 1996; Wade et al., 2008). Moreover, severe injury coupled with premorbid family dysfunction and social disadvantage have been linked to a poorer prognosis (Taylor et al., 2002). Children who have experienced severe TBI, had poorer performance premorbidly, and whose family was coping poorly have had the poorest recovery both for the immediate and long-term time periods (Anderson et al., 2006).

Studies that have evaluated the child's functioning within the family have found that an open communication style, flexibility, and positive coping strategies are associated with improved adjustment for the child (Benn & McColl, 2004; Hawley, 2003). Mothers and fathers cope with a disability differently. Mothers focus on the needs of the child and the family while fathers focus on the long-term financial aspects of the child's injury and recovery (Minnes, 1998). Parents often are important for determining how the child eventually adapts to his/her injury and the recovery period. Training for this role is often rudimentary and parents often seek additional information in order to cope with the child's difficulty (Semrud-Clikeman, 2001). Findings that help the parent learn helpful coping strategies and manage their own anxiety and concern about the child indicate that supporting the family in the development of coping strategies is a crucial aspect for the child's rehabilitation (Benn & McColl, 2004; Minnes, Graffi, Nolte, Carlson, & Harrick, 2000).

Implications for Assessment

Due to the various neuropsychological, cognitive, academic, memory, and psychosocial disorders accompanying TBI, a broad-based evaluation is imperative. Areas that should be included are attention, executive functioning, language, cognition, memory, visual-perception, adaptive behavior, and psychosocial functioning. Tests that are discussed in Chapter 6 need to be included in the evaluation of a child with TBI. In addition to the initial evaluation, serial evaluations need to be conducted to monitor the child's progress. Research has indicated that the largest amount of recovery occurs in the first six months following the accident, with additional recovery found 2–5 years post-injury. Not only should the neuropsychologist investigate the

standard and scaled scores that are obtained, but comparison of the raw scores is also desirable. In some cases, children with TBI may appear to decline in ability due to a lowering of standardized scores. However, if the raw scores are compared the neuropsychologist may find that the raw scores have plateaued, indicating that the child has neither progressed nor made progress. This difference is important particularly when developing treatment options.

Neuroimaging is generally part of the assessment process for the neurologist and medical team. Obtaining the reports of these results is crucial for understanding the damage that may have occurred. Scans obtained right after the injury will generally differ from those taken 6–12 months later when damage may be more readily seen (Donders & Nesbit-Greene, 2004; Wilde et al., 2005). As the child grows and develops it is helpful to obtain the follow-up neurological and neuroradiological studies to further inform the neuropsychological testing results.

Implications for Intervention

Cognitive and personality characteristics of the child, as well as family resources, marital stability, and socioeconomic status, have an impact on outcome variables measuring the child's recovery. With this in mind, developmental history and circumstances in the child's environment must be carefully considered. Further, teacher reports and a review of history help to determine the presence of preexisting disorders like hyperactivity, attentional problems, social interaction, and academic difficulties. Many teachers have had little training in TBI, and information that can be provided to the parent and the teacher can be invaluable in assisting the development of an appropriate individual educational plan. Emerging research using web-based applications that provide information as well as video conferencing for family interventions is promising and may provide the support that families need (Braga, Da Paz Junior, & Ylvisaker, 2005; Wade, Carey, & Wolfe, 2006; Wade, Wolfe, & Pestian, 2004).

Medications may be helpful for some of the symptoms seen in children with TBI, including attention and aggression. Some studies have found modest improvement with stimulant medication (Jin & Schachar, 2004). Other medications for internalizing and externalizing disorders have found small to modest improvement (Beers, Skold, Dixon, & Adelson, 2005) with few studies solely targeting TBI using selective serotonin reuptake inhibitors (SSRIs) (Donders, 2007). In summary, studies indicate the need to integrate data from various sources in order to measure the full impact of head injury on children, and support the need for an integrated paradigm for developing educational and psychosocial treatment programs for brain-injured children.

Fetal Alcohol Syndrome

Prenatal exposure to teratogenic agents, including alcohol and cocaine, has been known to produce various neuropsychological, neurocognitive, and neurobehavioral disorders in children. Fetal alcohol syndrome disorders (FASD) describe children who exhibit a growth deficiency, facial anomalies, and CNS dysfunction (Premj, Benzies, & Hayden, 2007). FASD is an umbrella term and describes the continuum of difficulties found when a fetus is exposed to alcohol. It includes Fetal Alcohol Syndrome (FAS), Fetal Alcohol Effects (FAE), Partial Fetal Alcohol Syndrome (PFAS) and miscellaneous conditions included in the spectrum (Alcohol-related Neurodevelopmental disorder, Alcohol-related birth defects, and static encephalopathy) (Streissguth & O'Malley, 2000).

Children with FASD frequently show delayed development, overactivity, motor clumsiness, attention deficits, learning problems, cognitive retardation, and seizure disorders. The prevalence of FAS is estimated to be between 0.5 per 1,000 live births (May & Gossage, 2001) to 25 per 1,000 for children from alcoholic mothers (Canadian Pediatric Society, 2002). Differences in incidence rates of FAS depend on community, ethnic, and cultural mores, and on geographical area.

Etiology of Fetal Alcohol Syndrome

The type and severity of FAS depends upon when the mother drank during gestation, how much alcohol was consumed, how frequently it was used, and the age of the mother. During the first few weeks of prenatal development, alcohol (ethanol) is thought to cause either cell death or modify chromosomes in such a way as to precipitate a miscarriage (Nichols, 2007). Heavy alcohol consumption from weeks four to 10 of gestation has been found to cause disorganization of cells due to disruption of cell migration and severe cell loss leading to microcephaly (small brain). From eight to 10 weeks and upward during pregnancy, ethanol disorganizes cell migration and development and neural synapses are not formed, thus preventing appropriate neuronal transmission (Nichols, 2007). Finally, alcohol consumption in the third trimester interferes with the development of the cerebellum, hippocampus, and prefrontal cortex, thus setting the stage for later problems with balance, attention, and new learning (Livy, Miller, Maier, & West, 2003).

Exposure to alcohol in utero has a wide range of consequences to the child, from no problems to fetal death, and the probability of a child experiencing FASD from a mother who drank ranges from 1 to 7 percent (Clarren, Randels, Sanderson, & Fineman, 2001; May et al., 2006). The threshold for alcohol use during pregnancy appears to be between seven and 28 drinks per week in early and mid-pregnancy. This level of alcohol intake is highly related to neurobehavioral sequalae (May et al., 2008). Although the mechanism behind FAS is not fully understood, nutritional and metabolic effects of alcoholism, age of the mother, whether the mother binged or drank steadily, and maternal and fetal metabolism, along with the teratogenic effects of the alcohol itself, are believed to play a role (Jacobson, Jacobson, Sokol, Chiodo, & Corobana, 2004).

Animal models are being used in order to understand the etiology of FAS. Rats prenatally exposed to alcohol were found to be highly irritable and to have difficulty with regulating their sleep and eating adequate amounts of food (Kelly, Day, & Streissguth, 2000). Studies with animals have also found that damage to the hippocampus occurs with alcohol ingestion during pregnancy (Berman & Hannigan, 2000), as well as the frontal cortex (Mihalick, Crandall, Langois, Krienke, & Dube, 2001).

Studies of the brains of children diagnosed with FAS show decreases in total brain size, particularly in the cerebrum and the cerebellum (Riley & McGee, 2005; Spandoni, McGee, Fryer, & Riley, 2007). Moreover, smaller volume of the basal ganglia has also been found, particularly in the caudate nucleus (Archibald et al., 2001), as well as reduced metabolic activity in the caudate in children with FASD (Clark et al., 2000). The corpus callosum has been affected in individuals with FASD. Agenesis of the corpus callosum has been found as well as thinning of the regions near the genu and splenium (Sowell et al., 2001). Asymmetry of the hippocampus has also been found to differ in FASD children, showing a smaller left hippocampus than right compared to typically developing children (Riikonen, Salonen, Partanen, & Verho, 1999). This increase in asymmetry is related to poorer memory skills. Reduced volume of the frontal lobes, particularly in the left, has also been found, particularly in the ventral portions of the frontal lobes (Archibald et al., 2001; Malisza et al., 2005). These regions were found to show less activation in children with FASD when performing a working memory task compared to typically developing children (Connor & Mahurin, 2001). These regions that are compromised in FASD children are important for the development and maintenance of attention, executive function, and memory skills – all areas found to be compromised in children with FASD.

Implications for Assessment and Diagnosis

Facial features usually assist in the diagnosis and are more prominent on the left side of the face. The discriminating facial features of FAS include a shorter than expected eye opening, flattening of the midface, a short nose, indistinct ridges between nose and mouth, and a tiny upper lip. Associated features include small folded skin at the inner corner of the eye, low nasal bridge, ear anomalies, and an abnormally small jaw (Sokol, Delaney-Black, & Nordstrom, 2003). In addition, the child's growth

is generally delayed. The facial features generally become less evident after puberty, and diagnosis at that point becomes problematic (Astley & Clarren, 2001). A small head continues to be a distinguishing feature, with only 28 percent of samples showing normal head size (Hoyme et al., 2005). FAS is diagnosed when the facial characteristics are present, a growth deficiency is present, and CNS malfunctioning occurs in conjunction with a maternal history of alcohol abuse. If the child shows some of the facial characteristics of FAS and/or CNS signs along with maternal drinking, the diagnosis of fetal alcohol effect (FAE) is given (Autti-Ramo et al., 2006).

Hoyme et al. (2005) have suggested that the criteria established by the Institute of Medicine be revised to be more specific. The original criteria basically are an umbrella term that includes most of the symptoms. Hoyme et al. (2005) suggests that FAS includes the minor facial anomalies, growth retardation, and deficient brain growth while partial fetal alcohol syndrome (PFAS) includes the minor facial anomalies and either growth retardation or structure brain abnormalities/smaller brain with or without confirmed maternal alcohol use. For a diagnosis of alcohol-related birth defects (ARBD) there must be characteristic facial features, a confirmed history of prenatal alcohol exposure, and at least one organ system showing significant structural defects. Alcohol-related neurodevelopmental disorder (ARND) requires documented maternal alcohol use, deficient brain growth, and evidence for behavioral and cognitive difficulties that are not within age expectations.

Ervalahti et al. (2007) compared dysmorphic features and cognitive functioning in children with FASD. It was found that dysmorphic features and growth deficiency were significantly related to cognitive ability. The more dysmorphic the child appeared, the lower the ability level. The relation was found to be modest indicating that facial features and growth retardation alone were not highly predictive of cognitive ability. The risk, however, for significant learning disabilities, ADHD, social skills deficits, and mental retardation increased by 37–82 percent for children with FAS/PFAS (Burd, Klug, Martsolf, & Kerbeshian, 2003). In order to more fully understand why types of neuropsychological problems may be present in FASD, it is important to briefly review the basic neuropsychological domains.

Neuropsychological Aspects of FASD

Longitudinal studies of children with FAS indicate that this disorder persists throughout the life span. Difficulties are present with cognitive retardation, attention, and adaptive behaviors. Information processing skills also appear to be significantly affected and sensitive to the effects of maternal binge drinking (Nichols, 2007). Newborns of alcoholic mothers have been found to be delayed in their response to the environment and to be born with low birth weights (Jacobson et al., 2004). These difficulties continue into adulthood. It is accepted by researchers and clinicians that interventions need to begin early to assist with adaptation to problems most frequently seen cognitively and behaviorally for these children (Green, Diaz-Gonzalez de Ferris, Vasquez, Lau, & Yusim, 2001; Zevenbergen & Ferraro, 2001).

There is a high incidence of cognitive retardation and ADHD in children with FAS and ability is generally lower for children with FAS than those with PFAS, ARBD, or ARND. Streissguth and O'Malley (2000) reviewed the literature on FAS and found that IQs ranged from 29 to 120 with a mean of 70 for children with FAS, and from 42 to 142 with a mean of 90 for those with PFAS. When children with FAS, PFAS, and ARND were compared using a Finnish sample, there was no difference in Full Scale IQ (FSIQ) or Verbal IQ (VIQ) on the WISC III. However, there was a significant difference on Performance IQ (PIQ), with the FAS group scoring significantly more poorly compared to the PFAS group. For all groups, the scores were significantly poorer than the standardization sample, with the FAS group scoring more poorly on all indices (Ervalahti et al., 2007). Ability scores were fairly stable throughout the life span (Niccols, 2007).

Thus, these findings indicate that for the severely affected children with FAS, cognitive retardation is common. For those with PFAS and ARND retardation is not as clear-cut and some children function within the average to above average range in

cognitive ability. In addition, the degree of dysmorphic features appears to be related to the level of cognitive functioning, with more dysmorphic features associated with poorer performance. It is likely that the children who are exposed throughout pregnancy and whose mothers may binge drink or are older, are at highest risk for the most severe form of FAS.

Attentional and Executive Functioning

Attentional problems are frequently found in children with FASD (Riley & McGee, 2005). Comparing children with FASDs to those who do not have a history of alcohol exposure, but have ADHD using a computerized attention test, found that children with FASDs showed more inattention problems while those with a sole diagnosis of ADHD showed more impulsivity (Mattson, Calarco, & Robertson, 2006). When FAS children were compared to those without FAS on attentional measures the FAS children had significant visual attention problems. Their auditory attention was average unless they were asked to listen to long and complex material (Mattson, Lang, & Calarco, 2002).

It has been suggested that executive functioning is a core deficit in children with FASD (Kodituwakku, Kalberg, & May, 2001; Schonfeld, Mattson, Lang, Delis, & Riley, 2001). Cognitive flexibility, response inhibition, planning, and concept formation were evaluated in children with FASDs compared to typically developing children (Mattson, Goodman, Caine, Delis, & Riley, 1999). The children with FASDs had marked deficits in cognitive flexibility, response inhibition, and planning skills. Additional problems were found on measures of abstract problem solving. When children with FASD, ADHD, and typically developing controls were compared on measures of executive functioning, both clinical groups had difficulty on measures of cognitive flexibility. The FASD children had additional problems on measures of working memory, compared to those with a sole diagnosis of ADHD. In addition, both the ADHD and FASD groups had difficulty with verbal fluency, with the FASD group scoring the poorest, although actually functioning above ability level (Vaurio, Riley, & Mattson, 2008). These results were also found when children with heavy prenatal exposure to alcohol were compared to those without on measures of verbal and nonverbal fluency (Schonfeld et al., 2001). In this case the children with and without FAS, but with prenatal alcohol exposure, showed problems on measures of fluency compared to controls. The alcohol-exposed groups did not differ from each other. IQ was not a significant predictor for difficulties on these measures, while it was for the diagnostic group.

The above findings indicate that executive function deficits and attentional problems are key difficulties in FASDs. These disorders are relatively independent of IQ and are present even when the full diagnosis of FAS is not. The finding of problems with cognitive flexibility and response inhibition are important for the development of appropriate interventions; a topic discussed later in this section.

Adaptive Behavior

Adaptive behavior skills appear to be most problematic for FAS children and appear related to the problems cited above with executive functioning. The finding that adaptive behavior difficulties are present is true for those children who were and those who were not cognitively retarded (Riley & McGee, 2005). Areas that were particularly difficult for children with FASDs were acting without considering the consequences, problems with initiative, inappropriateness of behaviors due to an inability to read social cues, and inability to establish social relationships (Whaley, O'Connor, & Gunderson, 2001). These findings were present for those children with mental retardation and FASDs as well as those without these disorders. In addition, these difficulties tend to increase in severity with age so that the FASD child's adaptive behavior does not progress at the same rate as those without FASDs.

Academic Achievement

Academic achievement also poses difficulty for children with FASDs. A large study of children with FASDs found that approximately 50 percent of the sample were retained at least once, 40 percent were receiving special education services at some point in

school, and 65 percent were receiving additional support in reading and mathematics (Streissguth, Barr, Kogan, & Bookstein, 1996; Streissguth & O'Malley, 2000). Children with FASDs have difficulties particularly in new learning and in arithmetic (Kodituwakku et al., 2006). A relation between how much alcohol has been imbibed during gestation in the second trimester of pregnancy and mathematics ability with IQ controlled has been found (Goldschmidt, Richardson, Stoffer, Geva, & Day, 1996). Mathematics appears to be the most vulnerable as reading and spelling skills were not as directly affected. In addition, children with pronounced dysmorphic features also had more difficulties in mathematics calculation and reasoning compared to those with fewer dysmorphic features (Howell, Lynch, Platzman, Smith, & Coles, 2006).

Language Skills

Language skills in FAS children have not been found to be generally deficient. Language development seems more closely related to the quality of caretaking, independent of SES and/or alcohol exposure. As discussed in the executive function section, verbal fluency appears to be the most affected in the language area (Schonfeld et al., 2001). Language comprehension difficulties are problematic for children with FAS (May et al., 2006). In addition both verbal and nonverbal IQ scores were significantly below average for children with FASDs who also showed significant dysmorphic facial features. These findings indicate that verbal skills are compromised in the more significantly affected children. These challenges likely interact with learning difficulties to further complicate the establishment of intervention strategies. It is important to evaluate children with suspected or confirmed FASD for possible language difficulties, particularly those involved in language comprehension.

Motor

Difficulties in motor functioning have been found in children with FASD particularly in the areas of balance, visual-motor integration, fine motor dexterity and gross motor skills (Adnams et al., 2001; Connor, Sampson, Streissguth, Bookstein, & Barr, 2006; Roebuck, Mattson, Marion, Brown, & Riley, 2004). These difficulties continue into adulthood with FASD adults exhibiting significant problems with balance and fine motor control (Connor et al., 2006). The difficulties that continue to be present in motor skills are likely to compromise the individual's ability to readily complete activities of daily living such as buttoning, writing, and walking smoothly. Continued evaluation and support for motor skills would appear to be appropriate for the more severely affected individuals with FASD.

Psychosocial Considerations

Many FAS children come from chaotic home environments where alcohol and other drugs are used. In addition, there is a relation between poor executive functioning and social skills attainment for children with FAS (Schonfeld, Paley, Frankel, & O'Connor, 2006). Attachment difficulties have been confirmed in many children with FASDs, as well as in animals exposed to prenatal alcohol (Kelly et al., 2000). Children with FASDs have been found to be very outgoing, affectionate, socially engaging, and relate to both familiar and unfamiliar people similarly. It has been suggested that children with FASDs do not understand the perspective of another person and, thus, act on their own impulses without a clear understanding of boundaries between themselves and others (Coggins, Olswang, Olson, & Timler, 2003).

Individuals with FASDs appear at higher risk for psychiatric disorders throughout adolescence and adulthood (Niccols, 2007). In addition, many experience problems with substance abuse, criminal behavior, and disruptive behavior (Streissguth et al., 2004). These difficulties relate to problems with learning from previous mistakes, disinhibition, and likely poor school achievement, yielding fewer vocational options.

Many of these deficits appear to be related to a form of disinhibition and executive control problems, particularly where higher level functions are required (e.g., in social relations). It is also difficult to ascertain the degree to which psychopathology is a function of abnormal neuropsychological and cognitive development, and where it reflects the

troubled family environment. At any rate, the interaction of these factors should be considered when designing intervention programs.

Implications for Interventions

Studies evaluating interventions with FAS children are just beginning to establish treatment paradigms. For preschool children, early identification is crucial. Many children are cognitively retarded, show language delays, have problems with attention and memory, and have delayed social skills (Streissguth et al., 2004). Interventions such as early referral by physicians to the school or educational multidisciplinary team is improving, but continues to be below the incidence levels (Clarren et al., 2001; Eriksson, 2007). Medical education in this regard is sorely needed.

Early childhood special education services are invaluable for these children and their families. It is particularly important to involve families in any intervention program. Federal regulations provide for Individual Family Service Plans (IFSPs) as part of special education for young children (see P.L. 94–357). For elementary-aged children, continued special education services are needed for academic and social support. Medication for distractibility and overactivity may be considered for ADHD symptoms. Social skills training can be helpful to assist with development. Skills training needs to focus on foundational skills of learning social cues and gestures, and should be conducted in a school setting or in another setting where natural, ecologically valid social situations serve as the training ground. Minimizing sensory overload, recognizing sleep and eating disorders, and establishing a specialized curriculum are helpful (Riley et al., 2003). Emphasis should be placed on appropriate vocational training for adolescents. In addition, structured behavioral and vocational training is crucial for these students because social judgment, consequential thinking, and risk taking behaviors are problematic for FAS adolescents (Zevenbergen & Ferraro, 2001).

There is no research currently published that evaluates intervention programs with families. Interventions would differ depending on whether the child remains in a chaotic home or is placed in a more stable environment. Some success has been found in utilizing cognitive-behavioral techniques to prevent alcohol use in women who are drinking during pregnancy. Additional research is sorely needed in both intervention efficacy and family interventions. Prevention seems to be a more effective way to reduce the very serious deleterious effects to unborn infants, and should be a priority. A critical review of FASD interventions found limited scientific evidence for efficacy (Premj et al., 2007) and recommended a comprehensive research agenda developed by the main researchers in the area of FASD.

Conclusions

FASD is a preventable cause of cognitive retardation in children. Research has pinpointed difficulties for FAS children, specifically in terms of attention, self-regulation, problem solving, and social awareness. These difficulties continue into adulthood and create significant adjustment problems. Intervention programs have been developed for academic skills, but progress in adaptive behavior skills and basic living skills have been sorely lacking. Research investigating appropriate vocational training is needed, and the need for earlier training in this area is probably necessary. Moreover, using a transactional approach to the neuropsychology of FAS necessitates that interventions be developed for both the child and the family. Community-based interventions that recognize the particular values and culture of the community are also needed. Efforts to alert the medical profession to the need for early educational interventions is also advised.

Cocaine-Exposed Infants

The incidence of infants born exposed to cocaine, either during gestation or passively after birth, has risen in the past decade particularly, in the inner city where this is estimated to occur in one out of 3–6 infants (Lustbader, Mayes, McGee, Jatlow, & Roberts, 1998). Estimates indicate that more than 100,000 babies are born with exposure to cocaine and/or other drugs annually. Research investigating

the effects of cocaine exposure to the developing fetus has produced mixed results. Early studies indicated that there were statistically significant abnormalities across many measures of behavior, temperament, and cognition in early development (Singer, Farkas, & Kliegman, 1991). These abnormalities were well publicized, and many believed that the school systems would be flooded with "crack cocaine children" with severe developmental and behavioral disabilities.

Children with prenatal exposure to cocaine were initially characterized as listless, without affect, difficult to soothe, unmotivated, unable to establish attachments to caregivers, and hyperactive or aggressive (Coles, Platzman, Smith, James, & Falek, 1992). More recent findings have indicated that the effects of prenatal exposure to cocaine include intrauterine growth retardation, low birth weight, and problems with attention, arousal, and reactivity to stimuli (Kliegman, Madura, Kiwi, Eisenberg, & Yamashita, 1994; Mayes & Bornstein, 1995). Exposure to cocaine also increases the risk for sudden infant death syndrome either through active or passive exposure as an infant (Mirchandani, Mirchandani, & Hellman, 1991).

Effects of Cocaine

Cocaine is a powerful stimulant that can be ingested by snorting, "freebasing," or smoking. Crack is a form of cocaine that is increasingly popular because of its lower cost. Cocaine or crack produces an intense feeling of euphoria, with increased energy and self-esteem, and decreased anxiety. The rebound effects, which include increases in anxiety, exhaustion, and depression, are so emotionally painful that the addicted person will continually smoke cocaine or crack to avoid them. Chronic use is associated with paranoid and affective disorder, weight loss, and poor judgment and insight (Ahmadi, Kampman, Dackis, Sparkman, & Pettinati, 2008). Thus, cocaine's effect on the CNS is evident in adults and is related to the chemical properties of the drug.

Cocaine is water and lipid soluble and passes easily across the placenta (Behnke, Eyler, Garvan, Wobie, & Hou, 2002). Moreover, the fetus is exposed for a longer period of time than adults because of a deficiency in the mother's ability to chemically deactivate the drug action (Buck & Gurwitch, 2003). The cocaine also causes uterine vessel vasoconstriction, which results in reduced blood and oxygen flow to the fetus.

Variables that are just beginning to be studied include individual differences in one's ability to metabolize cocaine, the difference in frequency of cocaine use, and variations in placental perfusion. Koren (1993) suggests that these variables may account for the variation in severity of symptoms in children prenatally exposed to cocaine. Use of cocaine at high levels and at frequent intervals appears to have more adverse effects on the fetus than low level use. Some pregnant women using cocaine appear to have a lower activity level of a primary enzyme (cholinesterase) that metabolizes cocaine. For these women's babies, there is an increased risk of high exposure. Additionally, the human placenta varies in its ability to metabolize cocaine (Hoffman et al., 1992). Some fetuses have had higher exposures to cocaine compared to others when mothers from both groups consumed equivalent doses. Moreover, some fetal placental vessels appear to restrict blood and oxygen flow more than others when cocaine is ingested by the mother (Simone, Derewlany, Knie, & Koren, 1992). This variable may have significant effects on the level of exposure to cocaine, regardless of the amount of cocaine taken. To understand the effect of cocaine on the developing nervous system, it is important to evaluate a number of related variables before describing existing research on behavioral and cognitive outcome for these children. The following sections will discuss environmental variables, pre- and postnatal complications, animal models examining cocaine exposure on a fetus, previous studies on the result of cocaine exposure on the fetus, and current knowledge of the behavioral and cognitive status of these children.

Environmental Variables

Socioeconomic status has been frequently cited as an important variable in the evaluation of newborns for cocaine exposure. Many studies used nonrandom subject selection from large urban hospitals that primarily have indigent and minority women as patients. Results from these studies indicated that minority and poor women were more likely to have drug-exposed children

(Minnes, Singer, Humphrey-Wall, & Satayathum, 2008). The use of cocaine in pregnant minority women has been found to interact with such variables as polydrug use, less prenatal care, lower weight at time of delivery, and less weight gain during pregnancy (Arendt et al., 2004a). These women are more likely to provide a poorer caregiving environment for the infant, possibly due to poor maternal mental status (Beeghly et al., 2006).

Mothers who used cocaine during and after pregnancy were studied to evaluate environmental aspects present in these homes (Minnes et al., 2008). Mothers who lost custody of their child were compared to those who maintained custody. The mothers who had their child removed from the home showed more childhood neglect and physical abuse in their history. In addition, these mothers were more likely to not have had prenatal care, used more cocaine during pregnancy, and showed greater psychological distress compared to the mothers that maintained custody. The use of crack cocaine was detrimental to the mother's functioning, a problem that increases in severity over time. Thus, understanding the dynamics of cocaine use during pregnancy depends on a systems model that interrelates polydrug use with environmental and/or lifestyle issues. It is not clear what difficulties may be found between the children removed from these homes and those who remained with the mother. This type of study would be interesting, particularly given the higher rate of cocaine use in the mothers who lost custody.

One issue that is important to note is the preponderance of studies of non-white women who use cocaine. Population demographics indicate that addiction is color-blind. However, babies born to non-white women addicted to cocaine are more likely to be removed from the home than those of white women also addicted to cocaine (Neuspiel, 1996). One issue that needs to be further evaluated are the environmental and societal reasons for such addiction as well as the paucity of services for poor, ethnic women with addictions.

Animal Models

Animal models help us understand the mechanisms underlying the effects of various neurotoxic agents. Applying the results of animal studies to human behavior has been found to be helpful with these agents. A study of offspring from Sprague-Dawley CD rats given multiple daily doses of cocaine found that exposure to cocaine produced poorer performance on more complicated cognitive tasks when the rats were fully mature (Smith & Morrell, 2008). Problem solving and socialization behaviors were particularly susceptible to cocaine exposure in utero (Seymour & Wagner, 2008).

Alterations in neural function have also been found, lending support to the hypothesis that cocaine, used frequently and in large doses, acts as a neurobehavioral teratogen (Thomas, Kalivas, & Shaham, 2008). Controlled studies utilizing animal models have been conducted only in the past six to eight years, and data are continuing to emerge. These findings can inform clinicians about the possible affects cocaine has on the developing nervous system.

Pre- and Postnatal Medical Effects

Prenatal exposure to cocaine is believed to disrupt blood flow in the fetus, which in turns interferes with organ development (Rizk, Atterbury, & Groome, 1996). Cocaine also causes vasoconstriction in the placenta, thus reducing blood flow to the fetus and likely contributing to increased birth defects in cocaine-exposed fetuses (Minnes et al., 2006). Cocaine use in the early stage of pregnancy places the fetus at high risk for changes in brain growth, synaptic formation, and cell migration (Bandstra, Morrow, Anthony, Churchill et al., 2001).

Increased risk of spontaneous abortions, abrupted placentas, and meconium stained amniotic fluid is associated with maternal cocaine use (Volpe, 1992). Increased rates of prematurity have been confirmed, and may be related to the early neurodevelopmental effects found in these children (Bada et al., 2005). Premature birth has been associated with a history of heavier drug abuse and/or significant socioeconomic disadvantage. Intrauterine growth retardation has been present in cocaine-exposed infants, when compared to non-cocaine-exposed infants, as well as small head size and slower brain growth (Singer et al., 2002). When very low birth weight babies were studied on follow-up who had been diagnosed with chronic

lung disease, 25 percent of these babies had been cocaine-exposed; double the rate of cocaine exposure generally seen (Singer et al., 1991).

Neurological abnormalities found in neonates prenatally exposed to cocaine include cerebral infarcts and EEG abnormalities (Chiriboga, 1998; Jones, Field, Davalos, & Hart, 2004). Cocaine-exposed children show greater right frontal EEG asymmetry when confronted with stimuli requiring an empathic response. In addition quantitative EEG (QEEG) studies have found that children with utero cocaine exposure show differences in response to stimuli that is similar to that of adults who are addicted to cocaine (Prichep, Kowalik, Alper, & de Jesus, 1995). These findings indicate that neuronal transmission differs in children with exposure to cocaine. No longitudinal study was found that evaluated whether these differences continue in childhood or whether there is a relation between EEG abnormalities and neuropsychological deficits.

Neuroimaging Findings

Rivkin et al. (2008) studied children with in utero exposure to cocaine, alcohol, tobacco, and marijuana using volumetric MRI. In the sample of 14 children, nine had exposure to cocaine and alcohol, 12 to cocaine and tobacco, and eight to cocaine and marijuana. For the control group 13 had no exposure to substances while two had exposure to alcohol, six to tobacco, and three to marijuana. The mean age of the children is 12.3 years, with a range from 10 to 14. As a whole, children with intrauterine exposure to cocaine showed smaller gray matter volumes, smaller head circumferences and smaller deep gray matter volumes compared to those without cocaine exposure. In addition, for children with more types of substance exposure cortical gray matter volume and deep gray matter volume, as well as head circumference, declined significantly with the smallest volume present for children who were exposed to all four substances. While this study has very small numbers, the findings suggest that polydrug abuse during pregnancy is related to significant declines in gray matter, particularly that in the subcortical regions that are responsible for response inhibition, gating of stimuli, and reactivity. These findings, while suggestive, need to be replicated with larger samples and possibly by age.

A large scale study of children exposed to cocaine in utero utilized diffusion tensor imaging (DTI) when the children were between the ages of 10 and 12 (Warner et al., 2006). Findings indicated that DTI of children exposed to cocaine found more diffusion in the left frontal callosal and right frontal projection fibers. When the sample was analyzed including additional substances of marijuana, alcohol, and tobacco use, it was found that prenatal exposure to cocaine, alcohol and marijuana affected the left frontal callosal fibers. In addition, for children exposed to both cocaine and marijuana, the connectivity was much poorer than for those children solely exposed to cocaine. In addition, the children completed executive functioning measures and these measures were correlated with the DTI measures. It was found that, for the sample as a whole, children who performed better on the executive functioning tasks had greater diffusion in the left frontal callosal fibers. In addition, faster performance on the Trail Making test was associated with better diffusion in the right frontal regions. These findings were taken to suggest that hemispheric connectivity is not as well developed in children with cocaine exposure, and that their brains may be neurologically immature for their age.

In sum the neuroimaging studies have just begun to evaluate the neurological integrity of the brains in children with cocaine exposure. These findings are intriguing and suggest that the trajectory for developing white matter tracts through DTI analysis may be different and is particularly compromised for children with exposure to both marijuana and cocaine. Moreover, there appears to be a compromise in the gray matter both cortically and subcortically for these children indicating, again, that those exposed to several substances exhibit the most effect. Given that many individuals who use substances during pregnancy are often polysubstance abusers, these findings are important for our understanding of the child's development and difficulties (Minnes, Singer, Arendt, & Satayathum, 2005). These aspects of neurological development are likely related to neuropsychological functioning in children with cocaine exposure. The following section provides a brief overview of the literature

describing what has been found in the major domains in children exposed to cocaine.

Neuropsychological Functioning

Cognitive Development

The early development in utero of cocaine-exposed babies has been studied more extensively than later development. Infant development has been evaluated to determine whether cognitive delays are present early on and whether they continue over time. These studies have been difficult to fully conduct in order to determine cognitive challenges due to confound factors such as low socioeconomic status, poor prenatal care, low maternal education and IQ, and negative environmental factors that have all been linked to lower ability without the additional substance abuse issue (Singer, 1999). Studies that seek to isolate these additional environmental causes of cognitive difficulty apart from the cocaine exposure are difficult to conduct. Singer et al. (2008) sought to control these additional variables and studied children with cocaine exposure during the first two years of life. Results found that children with cocaine exposure showed significant delays on the Bayley Mental Scales of Development, with scores generally below 80 that continued through the first two years of life. There was a 13.7 percent rate of mental retardation in the group, which is significantly higher than the 2 percent that would be expected in the population. In addition, 38 percent of the children had mild to moderate delays significant enough to warrant special education. These findings were true even when the variables of the home environment were controlled. These results were related to the amount of cocaine the mother used during pregnancy and the frequency of the use suggested that the fetal brain was directly affected by the cocaine, possibly due to hypoxemia (lack of oxygen in the blood). No motor difficulties were found.

Children at five years of age have had difficulties on measures of language, school readiness, impulse control and visual attention (Pulsifer, Butz, O'Reilly, & Belcher, 2008; Richardson, Goldschmidt, & Willford, 2008). Global ability, visual-motor skills, fine motor skills and sustained attention were found to be within normal limits. Early elementary school-aged children exposed to cocaine have not exhibited significant intellectual difficulties, but did have an increased risk for learning disabilities, particularly in the area of reading (Morrow et al., 2006).

When the cognitive ability of prenatally cocaine-exposed preschool children was evaluated, their ability measures did not differ from non-exposed children within the same socioeconomic status. However, children exposed prenatally to cocaine were found to be less likely to have average ability and more likely to show ability measures in the below average range. Differences were found on measures of visual-spatial ability, general knowledge, and arithmetic skills. Those children exposed to cocaine prenatally who were removed from their home were found to show better vocabulary scores and higher ability levels compared to those who remained with the biological parent. These findings are consistent with a study that found the biological mother's vocabulary ability and home environment to be stronger predictors of developmental outcome than prenatal drug exposure (Arendt et al., 2004b).

Language. When the confounding effects of environmental contributions to language skill development was controlled, the more heavily exposed infants had poorer auditory comprehension skills compared to control children (Singer et al., 2001). In addition, more heavily exposed infants showed poorer total language skills compared to lighter exposed infants and control children. Auditory comprehension is an important aspect for development of receptive language; a skill that generally develops prior to expressive language. Other studies have not found an effect on language, but these studies generally did not control for parental cocaine use and caregiving factors, nor did they evaluate severity of cocaine use and the effect on language abilities (Bland-Steward, Seymour, Beeghly, & Frank, 1998; Hawley, Halle, Drasin, & Thomas, 1993; Nulman et al., 1994).

For older children aged 6–9 years studies of prenatal cocaine exposure have found lower receptive language abilities for six-year-olds, but not for nine-year-olds (Beeghly et al., 2006). In addition, children with prenatal exposure had poorer scores on measures of expressive language if they also had a

low birth weight. Females also showed poorer expressive and total language scores compared to males with prenatal exposure to cocaine. Those children who had been exposed to violence in the home scored more poorly across the groups, while children who had preschool experiences scored better across the groups.

Similarly, when preschoolers were studied it was found those exposed to cocaine prenatally scored more poorly on all aspects of language skills over development. However, environmental influences were also found to be important, particularly as the children grew. Both groups of children were mainly from an urban setting, low income, and African-American. These aspects were found to negatively influence language development for all children beyond the influence of cocaine exposure.

These findings indicate that while there are language difficulties, particularly for complex language at later years, these deficits may be more due to caregiving, economic status, and exposure to learning than to cocaine exposure, particularly for those children with light to moderate exposure. In this case, widespread intervention for children in lower socioeconomic situations would likely benefit all of these children with regard to language development.

The additional finding that girls may show more detrimental effects on language than boys is also interesting. It may well be that language is less lateralized in girls and, thus, the diffuseness of language skills is more affected by hypoxia seen in children with cocaine exposure than for boys whose language skills are more lateralized. Boys had more externalizing behaviors than girls, thus possibly affected differentially by cocaine exposure.

Attention/Executive Functioning

Children with prenatal exposure to cocaine have exhibited difficulty in the domains of inhibitory control and attention. It is thought that prenatal cocaine exposure changes the level of specific neurotransmitters important for these abilities during gestation, thereby altering brain organization (Lewis et al., 2004; Stanwood & Levitt, 2004). Studies have found difficulty with disinhibition in children with cocaine exposure at five years of age (Bendersky, Gambini, Lastella, Bennett, & Lewis, 2003), and with sustained attention in children aged 6–10 years (Bandstra, Morrow, Anthony, Accornero, & Fried, 2001; Richardson, Conroy, & Day, 1996).

Selective attentional difficulties have been found in preschoolers with polydrug prenatal exposure to marijuana and cocaine. Children whose mothers had utilized these substances had more difficulty with selective attention while those who had utilized marijuana showed significant problems with sustained attention (Accornero et al., 2007; Noland et al., 2005). These findings were independent of confounding variables such as maternal psychological distress, maternal IQ, and caregiver level of functioning.

Executive functioning has also been found to be problematic for children with prenatal cocaine exposure. Children with such exposure process information much more slowly and show more difficulties with working memory, particularly with problem solving skills (Mayes, Snyder, Langlois, & Hunter, 2007), and with reaction time and motor/response speed (Mayes, Molfese, Key, & Hunter, 2005; Noland et al., 2003). These skills are particularly important as the child develops and is required to respond quickly both socially and academically. Further study is needed to more fully understand what aspects of executive functioning are particularly compromised for these children.

Social/Emotional/Behavioral Development

Studies of the neonatal behavior of cocaine-exposed infants have found that these children may show no to mild withdrawal behaviors (Singer et al., 2008). Sensory and behavioral deficits consistently have been found with irritability and difficulty with self-soothing was found more often for these children by parent report. In particular, these children had difficulty screening out upsetting stimuli and decreased habituation to environmental stimuli. (Mayes, Cicchetti, Acharyya, & Zhang, 2003). Children at six months of age who were exposed to cocaine in utero had temperamental differences, primarily in their difficulties with cooperation, manageability, and responsiveness to routine. These children were also less responsive and showed less interest in

communication and participation in activity, while their cognitive development did not differ from that of non-exposed six-month-olds (Edmondson & Smith, 1994). Reactivity differences have been found using physiological response measures that persist into middle childhood (Kable, Coles, Lynch, & Platzman, 2008). The children with behavioral disturbance showed more extreme behavior problems compared to those with cocaine exposure. These differences were in contrast to children with behavioral disturbance in that children with prenatal cocaine exposure did not show a general level of hyperarousal, but did have increased skin conductance levels to stressors. As a result of these findings, children with cocaine exposure may well respond differently to stress and may overreact to these types of experiences. In comparison to the control group, the cocaine-exposed group had more difficulty controlling their arousal, but not nearly as much difficulty as the behavioral disturbance group had. These findings suggest that children with cocaine exposure may well show more fearfulness and/or anxiety to stressful situations and may need additional assistance in calming themselves down.

In controlled testing sessions others have not found these behaviors when trained observers rated the child's behavior (Frank et al., 2002; Messinger et al., 2004). It may well be that the parent ratings are reflecting the stress the mother is under, coupled with environmental variables, thus lowering the scores. It may also be that the child's behaviors are more difficult in an unstructured setting than when in a structured setting with trained professionals.

When infants have been studied and environmental factors carefully controlled, cocaine use during the second and third trimesters was associated with fussier babies that had a difficult temperament. In addition, these behaviors were most evident at a very young age, and seemed to improve as the infants got older. Parental aspects such as lower educational levels in the home, more hospitalizations, maternal depression, and third trimester marijuana exposure with current substance use placed the child at higher risk for behavioral difficulties (Richardson et al., 2008).

It is likely that methodological differences in these studies may account for these equivocal findings. It may be that controlling for home environment as well as caregiver current use of substances may help explain these differences. In addition, it may be important to evaluate when the mother used cocaine (throughout the pregnancy, variably, etc.) and what effect this had on the subsequent behaviors of the child. Further study is needed, particularly in identifying those children most at risk, but also to assist with appropriate interventions.

Summary

Contrary to the concerns of the 1980s and early 1990s, these children are not showing the long-term global cognitive deficits that were predicted. It is too early to say that these children will have no deficits. Current evidence suggests that cocaine exposure during pregnancy may predispose the child to later difficulties in attention, social development, and emotional regulation and development. Some children appear to have subtle language deficits in the preschool years. Abilities that are unable to be measured in the first three years of life may emerge poorly at later ages. The subtle deficits in organization, regulation of behavior, and problem solving may later translate into difficulty with abstract thinking skills. Moreover, one of the best predictors of later social and emotional adjustment is attachment to the caregiver (Stroufe, Fox, & Pancake, 1983). As we have seen from the foregoing review, cocaine-exposed children not only have difficulty with attachment, but their caretakers are frequently unavailable to them because of the addiction.

A transactional approach to understanding these children is paramount. It appears from the emerging research that the environment the child is in may be just as important as whether he or she was exposed to cocaine in utero. Children from impoverished, chaotic, abusive homes will do poorly, regardless of pregnancy history. To understand the functioning of these children, the clinician must take these variables into consideration in interpreting the data. Moreover, these children should be monitored for progress, as early subtle deficits may translate into later difficulties with more complex learning. Although further study is needed on

the long-term effects of cocaine exposure, control of these moderator variables (maternal addiction, poverty, nutrition, etc.) is necessary in order to isolate the effects of cocaine on the fetus.

CNS Infectious Diseases: Meningitis and Encephalitis

Infections of the brain at an early age may result in a variety of outcomes ranging from mental retardation to normal development, and from schizophrenia to affective disorders depending on the type of infection (Dalman et al., 2008). Central nervous system infections can be the result of bacterial, viral, and/or fungal invasions of the brain and spinal cord through the sinuses, ears, nose, and mouth.

In addition to the medical conditions, social and environmental factors have been found to be predictive of later sequelae from early infections (Kopp & Kralow, 1983). Thus, the infections interface with environmental factors in the resulting deficits, if any, for these children. The social factors cannot, of course, fully account for the deficits, as children with early insults to the brain have constraints placed on their development, and such constraints are tempered by how the environment handles them (Sameroff & Chandler, 1975). Thus, a transactional model for understanding these infectious processes is important. Both meningitis and encephalitis will be discussed briefly.

Meningitis

The meninges, as mentioned in Chapter 2, protect the brain from infections, cushion it from injury, and serve as a barrier to foreign objects. However, they are not impervious to damage or disease, and meningitis results when the meninges become inflamed, particularly in the arachnoid and pia mater layers. Meningitis refers to an inflammation of the meninges or protective layers of the brain and spinal cord, whereas encephalitis is a generalized inflammation of the brain. Viral or bacterial infections are the main cause of meningitis. Children older than two years of age generally show initial symptoms of a headache, stiff neck, vomiting, high fever, and joint pain as well as a sensitivity to light. For infants and neonates symptoms may include lethargy, failure to eat and vomiting (Hilliker & Whitt, 2003).

The incidence of bacterial meningitis is 2.5–3.5 per 100,000. Prior to the introduction of the haemophilus influenzae type b (Hib) immunization, Hib was the most common cause of bacterial meningitis. For neonates Group B streptococcus (GBS) is the most common cause of meningitis and is passed from the mother to the infant at delivery. Most cases of meningitis are currently caused by Streptococcus pneumoniae or Neisseria meningitides. Viral meningitis is more common, but is generally less severe than bacterial meningitis. This type of meningitis is generally spread through direct contact with saliva and mucus and results in fever, headache, and fatigue for about 7–14 days.

Bacterial meningitis disrupts the cerebrovascular and CSF relation. The most frequent site for meningitis is in the pia mater and the arachnoid layer and space. The diagnosis is confirmed by a sample of cerebrospinal fluid (CSF) taken through a lumbar puncture; bacterium is assayed in the sample. In meningitis the CSF is generally cloudy, and pressure is elevated. Neurological indicators are present through CT scans, including hydrocephalus, edema, or cortical atrophy with abnormal EEG results (Anderson & Taylor, 2000).

Treatment generally consists of high doses of antibiotics, frequently ampicillin, for 10 days. Chloramphenicol is also often prescribed in the event that the bacteria are resistant to ampicillin. Fluids are carefully monitored, and CT, MRI, and EEG studies are ordered as needed. The sequelae from meningitis depend on the age of onset, how long before the disorder is diagnosed, the infectious agent and severity of infection, and the treatment used.

Among children with meningitis, 40 percent will experience seizures, hearing loss, and/or hemiparesis with neurological complications seen in approximately half of these children. The mortality rate for bacterial meningitis is 5–10 percent (Anderson & Taylor, 2000).

Neonates are at highest risk for mortality from meningitis. Children who experienced coma and

subdural infections have had the most severe neurological and neuropsychological sequelae (V. Anderson et al., 1997). Moreover, those children who had seizures prior to the onset of meningitis, had a longer duration of illness and higher fevers, and those who were younger at onset had the poorest cognitive result following treatment (P. Anderson et al., 1997). Studies looking at long-term effects of meningitis found that 50 percent of the children showed significant cognitive and physical difficulty, with language difficulty, hearing problems, cognitive delays, motor delays, and visual impairments being the most frequent complications (P. Anderson et al., 1997).

Children who have recovered from the disease process need a comprehensive neuropsychological battery to monitor their progress. Such an assessment should be accomplished serially in order to detect any difficulties. The child should be screened repeatedly, and parent and school personnel need to be well versed in attending to possible difficulties in these areas. Moreover, given the importance of parental support and social development, these areas need to be attended to not only in any evaluation, but also in any proposed treatment paradigm.

Encephalitis

Encephalitis refers to a generalized inflammatory state of the brain. This disorder is frequently associated with an inflammation of the meninges as well. The incidence of encephalitis is reported to be approximately 1,400–4,300 cases in the United States annually (Anderson, Northam, Hendy, & Wrennall, 2001). Viruses are frequently the culprit in this disease, which can occur perinatally or postnatally. Encephalitis can be caused by viral diseases such as Herpes simplex or through insect bites. For the majority of cases, however, no cause can be pinpointed (Hooper, Williams, Sarah, & Chua, 2007).

There are two forms of the disease: acute and chronic. Acute forms are evidenced within days or weeks of infection, whereas chronic forms can take months to become symptomatic. Fever, headache, vomiting, loss of energy, lassitude, irritability, and depressive-like symptoms are frequently seen, with increasing confusion and disorientation as the disease progresses. At times speech processes are affected, paralysis or muscle weakness is seen, and gait problems occur (Hooper et al., 2007). Encephalitis is related to later problems with respiratory ailments, particularly when it co-occurred with influenza (Armin et al., 2008). In addition, the children affected prior to age two had brain development abnormalities, while these abnormalities were not as present in children above the age of two.

Diagnosis is through examination of the CSF for viral agents, CT scans and EEG analysis. Treatment includes antiviral agents if a viral cause has been discovered, or through the monitoring of the disease process, antibiotics, and fluids if no virus has been identified. Sequelae are generally related to the type of infection and the duration of the infectious process (Arvin & Whitley, 2001; Kimberlin, 2007). Generally, mental retardation, irritability and lability, seizure disorder, hypertonia, and cranial nerve involvement can be seen with the more severe disease process, while in mild to moderate cases there are few, if any, sequelae (Engman et al., 2008). A review of the literature found very few studies of encephalitis in children and the neuropsychological effects of this disorder. Further investigation is needed to more fully understand the results of these disorders as well as appropriate interventions. Encephalitis associated with herpes virus results in mortality 4–14 percent of the time, and further results in neuropsychological difficulty in 56–69 percent of cases, suggesting that these children require further study.

Chapter Summary

Children with various acquired neurological disorders and diseases have become a focus of study, and researchers have investigated the links between psychological, behavioral, and neuropsychological functioning in traumatic injury, infectious diseases, and prenatal exposure to teratogenic agents, including alcohol and cocaine. Children who experience congenital brain dysfunction tend to have problems with neuropsychological development. These difficulties are frequently subtle and appear related to difficulty in learning new material. Attentional and organizational skills are also sensitive to these

disorders and may emerge at older ages when these skills normally develop. These deficits have a negative impact on the adolescent and eventually the adult, and interfere with adjustment and overall adaptation.

In children and adolescents exposed to toxins, traumatic brain injury, and other CNS infectious diseases, it is recommended that frequent neuropsychological evaluations be conducted to monitor progress and to evaluate possible regression. The use of ability and achievement tests needs to be suspended because the performance of children with neurological problems can *not* be predicted as it can be for typically developing children. Parents and teachers play crucial roles in the adjustment and recovery of children with various neurological disorders and diseases. Too often support for the home and school environments are not present, although research indicates the need for this type of service in the treatment plan. If the educational needs of the child are not sufficiently impaired to qualify for special education services, modifications of the regular education program are mandated under Section 504 of the Americans with Disabilities Act of 1973. Children with TBI may qualify for services under P.L. 94–142 or Section 504, while children with other neurological diseases/disorders may be considered as "Other Health Impaired" under the same legislation. In any case, regular education teachers need in-service training to help them recognize the needs of neurologically-impaired children and also during the development of effective intervention strategies for the classroom.

Parents need support not only for the stress the disorder places on the family, but also to plan for the child's long-term development. A transactional approach to neuropsychological assessment can provide the needed support for the parent, school, and child by assisting not only during the diagnostic phase, but also during the planning and implementation phase. Serial assessments can be helpful in this process and can assist in planning appropriate interventions. These evaluations can also be sensitive to the not-always-anticipated changes in the child's development. The following case presentation illustrates the need for comprehensive evaluations as well as parental support.

Case Illustration

Stan's car was hit head-on by a drunken driver. He was 17-years-old at the time of the accident, and he had been a straight A student in his junior year of high school. He was comatose at the accident site, but was moving and breathing spontaneously on the trip to the ER. Intracranial pressure was initially a problem, but improved during his hospitalization. An intracerebral hematoma in the left basal ganglia and internal capsule was diagnosed. Stan had right-sided hemiparesis with a mild right facial droop. Oral motor skills were observed to be below average and speech production was difficult. Stan's hearing and vision were normal.

Stan was hospitalized for five weeks after the accident. He underwent neuropsychological assessment during his stay at the hospital. Findings at that time included difficulties with attention, memory and verbal learning problems, and difficulty with verbal concept formation, with abstract reasoning and visual-spatial skills intact. Stan was reevaluated three months later by a private neuropsychologist using the WMS III and the Woodcock-Johnson Achievement Battery III-Reading Subtests. Scores showed average skills in all areas of memory, with relative strengths in visual memory. Achievement testing showed average performance in reading with slight difficulties in writing. Attentional difficulties continued to be present.

Stan was referred for reevaluation 11 months after his discharge from the hospital. He was succeeding in a limited educational program and was slated to graduate from high school that spring. Continued weakness was present in his right arm and leg, and writing was extremely painstaking and difficult for him. Stan's purpose in obtaining this evaluation was for additional information to aid in his selection of a college as well as assistance in obtaining help for his college program.

Stan presented as a well-groomed young man with a ready wit and smile. He was cooperative throughout the assessment and was well motivated. Stan's attention was good, and he was not easily distracted by extraneous noises. Stan achieved a Full Scale IQ of 106 on the WAIS-III, which placed him in the average range of ability for his age. His verbal IQ of 99 was also in the average range, with more well

developed skills found on the performance subtests, resulting in an IQ of 116. His working memory skills and processing speed were in the average range. The following subtest scores were obtained:

Information	9	Picture Completion	15
Digit Span	9	Matrix Reasoning	11
Vocabulary	13	Block Design	14
Arithmetic	14	Letter-No. Sequencing	11
Comprehension	7	Digit Symbol	11
Similarities	11	Symbol Search	10

Language assessment found Stan's receptive language to be in the above average range on the PPVT-III (standard score = 115). His ability to name objects was also well within the average range on the Boston Naming Test (raw score = 55). Performance on the Boston Aphasia Screening Test was well within normal levels and showed good ability to compose paragraphs and stories.

Verbal fluency scores as measured by the Controlled Word Association test were in the low average range and below what would be expected given his previous history. Stan experienced difficulty naming words beginning with the letters F, A, and S. This finding was consistent with a below average performance on the Rey Auditory Verbal Learning Test. Stan was able to remember 11 of 15 words over five trials. His performance did not improve with a second trial. When asked to recall these words after a 20-minute delay, Stan was able to recall 10 correctly which placed him in the average range of functioning.

Motor testing indicated significant right-sided weakness. On the Grooved Pegboard, Stan's right hand performance was over two standard deviations below expectations for his age. He had been right-handed prior to the accident. His performance with his left hand was within normal limits. He used his left hand to complete the VMI as he was continuing to experience right-sided hemiparesis. His performance on the VMI with his left hand was well within the normal range (standard score = 115).

Executive function assessment indicated above average performance on the Wisconsin Card Sorting Test. This finding was consistent with the results of testing during his hospitalization. He showed average performance on measures of problem solving as well as in response inhibition. Moreover, Stan's attentional skills were also within average limits and no difficulties were found in this area.

Social-emotional assessment found that Stan shows some expected emotional distress over his difficulties, but that he has coped well with these challenges. He is currently working with a therapist to assist him in adjusting to changes both in his social and academic life. He did not qualify for any psychiatric diagnoses based on interview, behavior rating scales, or the MMPI.

Impressions were of a young man with at least average ability who has shown improvement in all areas. Continued difficulty was present in word retrieval and motor skills. These motor difficulties were hampering Stan's progress in school, and it was recommended that a peer note taker be secured for him and that alternative methods be provided for him to demonstrate his knowledge. Writing could be accomplished on a specially designed computer for ease of writing. It was further recommended that Stan attend a college with support present for students with disabilities. It was also suggested that he take no more than two reading courses per quarter. Follow-up of Stan's progress indicated that he has made the transition to college, that he has a peer note taker, and that his professors have adapted their requirements to fit. He continues to be well motivated and has decided to major in special education.

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