CHAPTER 9

The Neuropsychology of Violence

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Introduction

Neuropsychology has typically sought to assess the often subtle, yet dramatic effects of brain lesions on information processing and behavior. Following certain brain lesions, a well-adapted individual can become irritable, impulsive, incapable of sustaining concentration, and neglectful of social rules. In such cases, the individual's ability to process information in a socially adaptive way becomes severely impaired. The observation of these profound changes prompted the development of neuropsychological accounts for the deficits, seen in a broad range of behavior problems, including violent behavior.

These *deficit models* emphasize fundamental processing difficulties that are tied more closely to brain anatomy or physiology. In contrast, other accounts of violent behavior, such as social information-processing models, provide several complementary angles of analysis to a neuropsychological model. Social information-processing models (see Chapter 15, "Social-Cognitive Processes in the Development of Antisocial and Violent Behavior") can be described as *distortion models*, as they emphasize the role of biases, beliefs, attributions, appraisals, and schemas, such as those targeted by cognitive-behavioral therapies. Deficits and distortions influence behavior either through interpretive biases or limits in the capacity to process information. In fact, deficits can sometimes be manifested as distortions. Thus, cognitive therapies may prove less effective when a fundamental neuropsychological deficit is at the core of an information-processing difficulty. Conversely, individuals with hostile attributional biases but without neuropsychological deficits may be more amenable to cognitive therapies. Finally, these systems may combine additively or interact statistically with individual motivational predispositions and situations.

Chapter Overview

In this chapter, we review the neuropsychological approach to cognitive deficits associated with violent behavior. In Part I, we examine issues pertaining to the assessment of

neuropsychological function and the assessment of clinical syndromes, which include delinquency and criminality, associated with violence. In Part II, we provide an overview of developmental issues affecting brain maturation and behavioral regulation. We integrate the aforementioned issues in Part III, where we review studies that help us understand violence from a neuropsychological perspective. As we note there, because a key method in neuropsychology has been the use of lesion analyses, we examine the extent to which violence is a consequence of brain lesions. We then turn to the few neuropsychological studies of violence and examine the larger body of literature on clinical syndromes associated with violence. In that section, we examine the extent to which neuropsychological problems have been identified in violence-prone individuals. Finally, we summarize and integrate the key observations derived from this review, address limitations in the extant body of research, and offer suggestions for further research on in this important and still growing area.

Part I: Assessment

Neuropsychological Assessment

Classic neuropsychological testing involves the administration of a battery of tests. These tests are designed to assess a variety of brain functions, ranging from basic perception to more complex neocortical problem solving, and require either verbal or motor responses. The stimuli used for these tests may be visual or auditory. Visual stimuli include pictures, abstract designs, and combinations of these stimuli, such as those found in various forms of puzzles, mazes, assortments of objects, pictorial depictions of story lines, printed colors, words, and numbers. Auditory stimuli may involve spoken words, numbers, problems, or stories. Computerized batteries are usually limited to motor responses performed through the click of a mouse or by means of a touch screen interface, although voice-onset recording, eye tracking devices, electrophysiology, and functional brain imaging are also used, albeit more rarely. Many of these tests qualify as neuropsychological because they were developed to test theories of brain function and were typically validated with lesion analysis studies, brain electrophysiological studies, or, more recently, with brain imaging studies. In other words, individuals with relatively well-circumscribed brain lesions were found to perform poorly on such tests, or these tests were found to engage specific areas of the brain. Thus, these batteries provide a profile of strengths and weaknesses that presumably vary as a function of the location and extent of lesions. Because location may correspond to some aspect of function, neuropsychological tests, in addition to being used to test individuals with documented lesions, are used to infer localization of brain lesions. For example, a frontal lobe hypothesis of violence emerged because violent individuals often perform poorly on tests of frontal lobe functioning.

The brain is a highly complex organ of interrelated areas that function as networks. Functioning in one area may depend on the functioning of others. This phenomenon refers to hierarchy of function and is called single (or simple) dissociation. Functioning in two areas may also occur independently from one another, referred to as double dissociation (Shallice, 2003). These notions are essential for testing specificity of deficits. Thus, tests are typically sensitive to certain functions, but not to others. However, pure tests of specific functions are rare. The interpretation of performance on such tests must be conducted in the context of other discriminating tests, in part because "lesions" can be diffuse or circumscribed, subtle or gross. Neuropsychological testing often complements a more basic but equally important neurological examination (which involves testing of snout, suck, and grasp reflexes; abnormal smooth pursuit eye movements; reciprocal hand movement coordination; and other capacities).

Neuropsychological lesions can be the outcome of pregnancy or birth complications, various illnesses, aging, head injury, intracranial tumors, cerebrovascular

disorders, exposure to toxic substances, or corrective surgical procedures. They can also be temporary and reversible, such as those observed under the acute effects of drugs and alcohol or certain illnesses, although some of these conditions can cause irreversible damage. Finally, results on neuropsychological tests are often assumed to represent the actual competence of the individual. However, there may be a gap between competence and actual performance. Thus, interpretation of test results should take into account the individual's motivation, attention, capacity to remember the sometimes complex rules required for optimal performance, language of administration, and cultural background. We refer the reader to more specialized sources for additional information (Kolb & Wishaw, 2003; Lezak, Howieson & Loring, 2004).

Clinical Syndromes Associated With Violence

Although physical violence is relatively easy to identify because of its overt nature, there is a paucity of studies examining the neuropsychology of violence. Much of what we know about the neuropsychology of violence derives from research on conditions that are associated with violence rather than violence per se. Thus, to appreciate the strengths and limitations of this body of literature, we first discuss the clinical syndromes most commonly associated with violence.

Violence research is conducted within two broad and overlapping nomenclatures, legal/judicial and clinical. In the legal/judicial areas, researchers have studied delinquency and criminal behavior. In the clinical arena, physical violence as a symptom is found under such conditions as conduct disorder (CD; 312.xx), antisocial personality disorder (ASPD; 301.7), Personality Change due to a General Medical Condition, Aggressive Type (310.1), and the differential disorder Intermittent Explosive Disorder (IED; 312.34) in the *Diagnostic and Statistical Manual of Mental Disorders – Text Revision* (DSM-IV-TR; American Psychiatric Association, 2000). In addition, it is sometimes found in psychopathy (Hare, 1999). Although the DSM-IV-TR regards ASPD as essentially synonymous with psychopathy, research strongly suggests otherwise, as we discuss later in this chapter (Hare, 2003).

One major limitation to studying violence by examining these disorders is that violent behavior is not necessary for their diagnosis. Only 6 of 15 CD symptoms and 1 of 7 of the adult ASPD symptoms qualify as explicitly violent if we define violence as physical aggression toward other people or threats of physical force. None of the criteria for IED meets this strict definition because a diagnosis can also be made in cases of property destruction alone. Three symptoms are necessary to obtain a diagnosis of CD (at least one of them before age 10 years) and ASPD, although ASPD also requires a history of CD before age 15 years (provided that symptoms are not due primarily to schizophrenia or a bipolar episode). IED can only be diagnosed when its symptoms are not attributable to CD, ASPD, other impulse control disorders, or a medical condition. The table of contents of the DSM–IV-TR does not include the words "violence" or "aggression." However, physical abuse of children (995.54 or V61.21) or adults (995.81 or V62.83), Adult Antisocial Behavior (V71.01), and Child or Adolescent Antisocial Behavior (V71.02) are additional nondisorder categories that may be the focus of clinical attention and in which violence may be present. Finally, aggression may also be secondary to "persecutory or grandiose delusions with anger" in Schizophrenia, Paranoid Type (295.30), and child or spouse abuse, as well as violent behavior, may occur during the course of an acute manic phase of Bipolar Disorder with psychotic features (296.xx) although none of these behaviors constitute official symptoms of these conditions.

Psychopathy can be considered a clinical syndrome, although it is not listed officially in the DSM-IV-TR. It has been investigated most commonly in criminals. Psychopathy

is most often assessed with the Psychopathy Checklist-revised (PCL-R score > 30), which involves both a standardized interview and a thorough review of official records (Hare, Hart, & Harpur, 1991). Other methods of assessing psychopathy have been developed. For example, self-report instruments exist (Levenson, Kiehl, & Fitzpatrick, 1995; Lilienfeld & Andrews, 1996; Lynam, Whiteside, & Jones, 1999), and children with psychopathic tendencies have been studied using various behavior rating systems (Frick, O'Brien, Wootton, & McBurnett, 1994; Lynam, 1998). In prisons, the overlap between psychopathy and ASPD is substantial but asymmetrical: most incarcerated psychopaths meet criteria for ASPD but not vice versa. Approximately 70 to 80% of prisoners meet criteria for ASPD, whereas only about 15 to 25% meet PCL-R criteria for psychopathy (Hare, 2003). Higher rates of violence are found in criminal psychopaths than in other criminals (Hare,), but not all psychopaths are violent. Psychopathy is also a potent risk factor for criminal and sexual recidivism (Salekin, Rogers, & Sewell, 1996).

Physical violence has also been studied developmentally. The most relevant studies focus on physical aggression. However, in most developmental studies, aggression scales often fail to distinguish physical from other forms of aggression. For example, the Child Behavior Checklist (CBCL; Achenbach, Edelbrock, & Howell, 1987) yields an aggression scale comprising 23 items, 3 of which refer explicitly to physical aggression, but more studies are extracting physical aggression items from that scale to study its development (Bongers, Koot, van der Ende, & Verhulst, 2004; National Institute of Child Health and Human Development Early Child Care Research Network, 2004). A similar problem plagues research on proactive and reactive aggression; most items of either scale do not refer specifically to physical aggression.

Given these limitations, it is therefore possible for neuropsychological studies in this literature to include nonphysically violent forms of CD, ASPD, psychopathy, or aggression. Nevertheless, investigators have not always made this distinction, as all of these behavior problems are often subsumed under the broad banner of antisocial problems. Further, the clinical syndromes in which physical violence is present, in addition to being comorbid with each other, are often comorbid with other conditions characterized by impulsivity, drug and alcohol abuse (DSM codes 303.xx, 304.xx, or 305.xx), and gambling (312.31) and with attention-deficit hyperactivity disorder (ADHD; 314.xx) and oppositional defiant disorder (ODD; 313.81) in developmental studies with childhood externalizing disorders. As we see later, several studies find or fail to find neuropsychological impairments in these associated conditions but without having taken violence into account. Conversely, several studies of violence have not taken these comorbid conditions into account. Thus, questions of specificity remain largely unresolved, although there have been notable improvements along these lines in the recent literature.

Part II: Developmental Issues

Developmental Patterns of Behavior

Moffitt (1993a) emphasized a brain-behavior account for the *development* of antisocial behavior. Historically, evidence supporting such an approach became clearer in the mid-19th century (Damasio, Grabowski, Frank, Galaburda, & Damasio, 1994; Mataro et al., 2001; Weiger & Bear, 1988). Moffitt built on this existing literature and her own longitudinal studies to bring this model to the forefront of research on antisocial behavior.

An initial classification as a function of developmental history of behavior gave rise to a flurry of studies. Research on the etiology and trajectory of CD suggests that "early-onset/persistent" (a.k.a. as "life-course-persistent;" Moffitt, 1993a) and "adolescent-limited" CD are actually distinct types (Kivlahan, Marlatt, Fromme, Coppel, & Williams, 1990). This finding underscores

a key methodological point; namely, that comparing or contrasting groups of adolescents or adults without knowing their natural history can be fraught with problems. "Early-onset/persistent" CD is presumably a more heritable condition likely to persist into adulthood, whereas "late-onset CD" is presumably less severe and usually limited to adolescence (Moffitt, 2003). Individuals with "early-onset/persistent" CD appear to be more likely to engage in physically aggressive behavior than individuals with "late-onset" CD (Lahey et al., 1998). This finding suggests that what may be driving "late onset" CD is not physical aggression but other antisocial behaviors. The identification of a "childhood-only/recovery/childhood-limited" group that displays antisocial behavior uniquely in childhood (Lahey, Waldman, & McBurnett, 1999; Moffitt, Caspi, Harrington, & Milne, 2002; Raine et al., 2005; Raine, Yaralian, Reynolds, Venables, & Mednick, 2002) suggests an additional problem behavior group. In one study, this group remained significantly impaired in adult life, as it displayed symptoms of internalizing disorders and continued to partake in less severe antisocial behaviors (Moffitt et al., 2002).

Although the concept of age of "onset" has sparked controversy (Tremblay, 2000) it has spawned valuable research into the development of antisocial behavior. Research into developmental trajectories suggests that the "early-onset/persistent" versus "late-onset" CD distinction is less clear than proposed by the DSM-IV-TR (Bongers et al., 2004; Broidy et al., 2003; National Institute of Child Health and Human Development Early Child Care Research Network, 2004). The typical developmental trends are for declines over time (Tremblay, 2000; Tremblay et al., 1996), a few children (about 5% of community samples) maintain relatively high levels of antisocial behavior across development (Bongers et al., 2004; National Institute of Child Health and Human Development Early Child Care Research Network, 2004), with an "onset" at around 2 years of age, although children may begin hitting, pushing, and kicking as soon as their limbs have enough strength. Moreover, in a few samples, an increase from low-level childhood physical aggression can be observed until pre-adolescence (Broidy et al., 2003). But the absence of adolescence data in these samples limits their usefulness for understanding trajectories of physical aggression across childhood and adolescence. We also note that developmental patterns may differ as a function of the delinquent behavior of interest (i.e., physical aggression versus theft or vandalism; Barker et al., in press; Lacourse et al., 2002).

In sum, moderate levels of physical aggression are normative in preschoolers, and most children exhibit a decline over time. This may correspond to a "childhood-only" pattern, that is, an "early onset" (or "early starter") group that is not persistent. For a few children, initial levels of antisocial behavior remain high across development, corresponding to the "early-onset/persistent" pattern. In very few samples, we observe increases in physical aggression over time, but no evidence of sudden "late onset," and lack of adolescent data in those samples precludes a full appreciation of their trajectory. Nonetheless, the labels "late-onset/late starter/adolescent limited" may reflect less violence than "early onset/persistent" when global measures of behavior, such as CD, antisocial behavior, or delinquency, are used.

Risk Factors That May Affect Brain Development

As developmental patterns have been studied with longitudinal designs, there has been increasing interest in conditions that can contribute both to those patterns and to poor neuropsychological function, such as a history of exposure during or after pregnancy to brain-altering psychopharmacological agents (e.g., cigarette smoke, alcohol, drugs), other perinatal or birth complications, poor nutrition, traumatic experiences (e.g., abuse), chronic stress, or behavior problems that heighten the risk of head

trauma through accidents or fights (i.e., ADHD, ODD).

Cigarettes may exert early effects in the intrauterine environment and later in the home environment. For example, exposure to environmental tobacco smoke appears related to poor cognitive performance in children aged 6 to 16 years (Yolton, Dietrich, Auinger, Lanphear, & Hornung, 2005). These effects were not due to exposure during pregnancy. Cigarette use during pregnancy has been extensively studied in recent years because it appears to remain relatively widespread, affecting 20 to 25% of mothers (Huijbregts et al., 2006), and could constitute one important target for preventative measures. Although there are sustained efforts to encourage pregnant women to stop smoking, the mechanisms by which smoking during pregnancy could affect behavior or neuropsychological development remain poorly understood. These effects may vary as a function of age. For example, early cognitive problems are related to poor behavioral adjustment in the preschool years (Séguin, 2004; Séguin & Zelazo, 2005), and maternal smoking during pregnancy was related to early physical aggression (Tremblay et al., 2004) and multiple problem behavior (Huijbregts, Séguin, Zoccolillo, Boivin, & Tremblay, in press). However, maternal smoking during pregnancy was not related to early cognitive problems once parental education was taken into account (Huijbregts et al., 2006). This finding suggests that the neurotoxic effects of cigarette smoke during pregnancy may more specifically affect preschool behavior regulation, but not necessarily through a cognitive regulation pathway.

Low birthweight is one consequence of maternal smoking during pregnancy (Huijbregts et al., 2006), but other conditions, such as perinatal and birth complications, may also affect neuropsychological and behavioral development (Raine, 2002a). For example, one study found that obstetrical complications interacted statistically with early family adversity in predicting later violent delinquency (Arseneault, Tremblay, Boulerice, & Saucier, 2002). As a complication, hypoxia at birth is associated with poor cognitive development (Hopkins-Golightly, Raz, & Sandler, 2003). In another study, minor physical anomalies of the mouth, which are thought to correspond to in utero neural development, were markers of later violent delinquency even after statistically controlling for familial adversity (Arseneault, Tremblay, Boulerice, Séguin, & Saucier, 2000).

Several chronically stressful developmental conditions have been associated with the development of smaller hippocampal volumes, at least in animals (Meaney, Aitken, van Berkell, Bhatnagar, & Sapolsky, 1988), which in turn may increase vulnerability to stress (Gilbertson et al., 2002). Although hypotheses positing a neurotoxic effect of cortisol have yet to be fully tested, early traumatic experiences have been implicated in the poor development of executive function (Mezzacappa, Kindlon, & Earls, 2001), even though the potential causal role of third variables (e.g., parental aggression or impulsivity) is difficult to exclude. In other studies, duration and quality of early care were associated with cognitive development (Castle et al., 2000). Malnutrition at age 3 years, one aspect of the early environment, was found to be related to later externalizing problems, a link that was mediated by IQ (Liu, Raine, Venables, & Mednick, 2004). Conversely, the duration of breastfeeding was found to be related to higher IQ even after statistical control for key confounds (Mortensen, Fleischer Michaelsen, Sanders, & Machover Reinish, 2002). Another risk factor that may be even more direct is family history of antisocial behavior, \vspace*{pt} although the effect of this risk factor may be genetic, shared environmental, or both. We now know that maternal smoking during pregnancy is correlated with parental antisocial behavior (Huijbregts et al., in press). Finally, early head injury is sometimes thought to be an important risk factor. One might expect early lesions to increase the risk of physical aggression. However, there is little support for that hypothesis as follow-up studies of children with early documented lesions rarely report physical aggression (Eslinger, Flaherty-Craig, & Benton, 2004).

Part III

Given the background considerations reviewed thus far, we now address two complementary questions: (1) Do some brain lesions dependably increase the risk for violent behavior? and (2) Do violence-prone individuals exhibit specific neuropsychological deficits?

The Effects of Brain Lesions on the Risk for Violence

Brain lesions in various areas can affect social behavior. However, interest in the cognitive aspects of the brain-violence relation has centered largely on the role of the frontal lobe because of its centrality to the regulation of antisocial behavior. Anatomically, the frontal lobe represents 20% of the neocortex and is located above the eyes and behind the forehead. Nomenclatures for describing the frontal lobe and its functions vary. Areas are typically distinguished by cytoarchitectonic analysis; that is, the architecture of the cells and their connections. Briefly, three major areas are typically designated: motor (area 4), premotor (areas 6 and 8, although area 8 is also referred to as posterior dorsolateral; Petrides, Alivisatos, Evans, & Meyer, 1993a), and prefrontal. The prefrontal cortex is further divided into dorsolateral (areas 9 & 46; both may be referred to as mid-dorsolateral; Petrides et al., 1993a), inferior (or ventral or orbitofrontal; areas 10 through 14), and medial (areas 25 and 32) regions.

One of the major cognitive functions of the prefrontal cortex is the temporal organization of behavior in memory (Milner, Petrides, & Smith, 1985), which is also one of the core aspects of executive functioning. In contrast, the premotor cortex is involved in movement selection and the motor cortex in movement execution. Frontal patients have difficulty regulating their behavior in response to external stimuli, as well as organizing it. One set of functions relates to the concept of working memory, which involves not only the online maintenance of information and control of interference but also the active processing of that information as an individual engages in action. Poor working memory affects all stages of executive function (Séguin & Zelazo, 2005); hostile biases in appraising a problem could be difficult to reconsider, plans could be difficult to carry out, rules (even if they are otherwise well-known) could be difficult to apply in real time, and monitoring of a plan (detection and error correction) could be difficult to achieve.

In terms of emotional regulation, individuals with lesions involving the orbitofrontal lobe have been shown to be disinhibited, socially inappropriate, susceptible to misinterpreting others' moods, impulsive, unconcerned with the consequences of their actions, irresponsible in everyday life, lacking in insight into the seriousness of their condition, and prone to weak initiative (Damasio et al., 1994; Rolls, Hornak, Wade, & McGrath, 1994). Primate studies also show that individuals with orbitofrontal lesions may be insensitive to social dominance hierarchies. The main consequence of such lesions has also been described as impairments in self-reflective awareness (Stuss, Gow, & Hetherington, 1992), perspective taking (Stuss, Gallup, & Alexander, 2001), social schema knowledge (Grafman et al., 1996), the ability to respond appropriately to social reinforcement (Rolls et al., 1994), the ability to make inferences about the mental states of others (or a Theory of Mind; Stone, Baron-Cohen, & Knight, 1998), and processing of social cues (Brothers, 2001). Some of these problems may also reflect malfunction of the amygdala (Bechara, Damasio, & Damasio, 2003), which is involved in processing of cues of fear (Whalen et al., 2004) and sadness (Blair, Morris, Frith, Perrett, & Dolan, 2000). In this way, all of these functions are less cognitive in nature and require more than straightforward neuropsychological testing to be evaluated. Individuals with such acquired conditions may lead a relatively stable life when provided with good post-trauma care, support, and considerable external structure. Nonetheless, their behavior may remain difficult to manage, and they may not recover any sense of autonomy (Mataro et al., 2001).

The ensuing syndrome has often been labeled as either acquired sociopathy (Damasio, 2000; Damasio, Tranel, & Damasio, 1991), acquired ASPD (Meyers, Berman, Scheibel, & Hayman, 1992), or pseudopsychopathy. Because these lesions appear to lead to psychopathic-like behavior, they were often thought to underlie physical violence. However, there is limited support for this hypothesis. Studies of war veterans, for example, support the hypothesis that orbitofrontal and mediofrontal damage increase the risk for aggressive and violent attitudes (Grafman et al., 1996), but not necessarily physical violence. Thus, physical violence is rare in acquired forms of antisocial behavior, which may in part be due to the fact that individuals with acquired brain lesions do not necessarily possess the many premorbid risk factors associated with physical violence. In fact, aggression (including physical aggression) appears to be associated with acquired frontal lesions only when there is a premorbid history of major depression, poor social functioning, and alcohol and drug abuse (Tateno, Jorge, & Robinson, 2004).

Other cortical abnormalities associated with aggressive outbursts include acute episodes of temporal lobe (temporolimbic) epilepsy (also known as interictal violence), which are similar in manifestation to IED (for a critical review, see Filley et al., 2001). Epilepsy is often an exclusion criterion in brain studies of violence (Critchley et al., 2000). Otherwise, violence in epilepsy is very rare, and a temporal lobe hypothesis of violence must be treated with caution (Teichner & Golden, 2000). Similarly, a limbic psychotic trigger reaction is a second type of seizure thought to underlie certain forms of sudden and unplanned violence. Consciousness may be severely clouded, resulting in amnesia, in the epileptic forms of seizures. By contrast, in the psychotic trigger reaction the individual typically remembers violent and bizarre acts, which are committed with flat affect and are totally – uncharacteristic (Pontius & Lemay, 2003). Such a psychotic reaction also appears to be distinct from violence related to schizophrenia (Pontius, 2003).

Neuropsychological Studies of Physically Violent Behavior

The other main approach to the neuropsychology of violence is to study physically violent individuals. This approach is probably the one most familiar to readers of this book. We begin with the most severe form, murder, and move on to less severe forms of physical aggression. We then follow with disorders and antisocial behavior problems in which physical violence is sometimes present. One of the first issues in research on such individuals involves determining whether they suffered head trauma at any point in their lives. Because of the limitations inherent in retrospective recall, most of the studies reviewed are not able to control for this variable. Moreover, defining the extent and types of injuries that qualify as head traumas is difficult. Prospective studies are better equipped to handle this issue. Nonetheless, retrospective studies of physically violent individuals have been informative.

Murderers

Several studies of murderers appear to support a dysfunctional frontal lobe hypothesis. For example, "frontal dysfunctions" were found in 65% of murderers (Blake, Pincus, & Buckner, 1995). This study used a multimethod approach including EEG and brain imaging, both complementing neuropsychological test results (although results for all tests were not available for all participants). However, far from deriving from a random sample, these individuals had been assessed at the request of their defense attorney. Further, the sample included individuals with poor general intellectual abilities, and it was not compared with a nonviolent offender group. Moreover, the use of multiple methods may have increased the risk of erroneously inferring the presence of frontal dysfunction. As a consequence of these

methodological limitations, these findings are difficult to interpret.

In another study, violent adolescent and adult psychiatric patients, compared with nonviolent controls, were shown to exhibit reduced overall prefrontal activity and increased anterior medial prefrontal activity (Amen, Stubblefield, Carmichael, & Thisted, 1996). However, neuropsychological testing does not complement many such brain imaging studies. It is therefore unclear whether these functional findings correspond to neuropsychological performance. Moreover, the correspondence between brain imaging and neuropsychology may also be elusive. For example, a series of brain imaging studies carried out by Raine and colleagues support a frontal deficit hypothesis (see also Chapter 7 in this volume). However, some of these studies used the Continuous Performance Task, a measure of sustained attention, and found brain processing differences in the absence of test performance differences between individuals who had committed impulsive murders as opposed to planned murders (Raine et al., 1994). The authors speculated that the visual cortex might have compensated for the frontal impairments. This study illustrates a key methodological point for interpreting neuropsychological test results; namely, that even under the best performance conditions, different strategies or brain networks may be employed by different individuals to achieve the same level of performance.

Although the frontal lobe has been implicated in these and other studies, they may account for only one portion of the explanation. Models that consider frontal dysfunction in murderers are broader and include developmental psychosocial factors (Blake et al., 1995; Raine, 2002b) and other factors reviewed in this book.

Physical Aggression

Although murder is the most severe form of violence, investigators have also examined whether neuropsychological problems are evident in milder forms of violence, such as in physical aggression. Physical aggression can be defined as hitting, kicking, biting, use of a weapon, and getting into fights. In some cases, this definition is broadened to include bullying and threats of violence. There are surprisingly few neuropsychological studies of physical aggression per se.

As a consequence, Séguin, Pihl, Harden, Tremblay, and Boulerice (1995) sought to develop a neuropsychological test battery on the basis of the frontal lobe and memory work of Petrides and Milner (1985). We complemented the battery on the basis of reviews of the neuropsychology of delinquency and conduct disorder. In her review of this literature, Moffitt (1990b) identified deficits in three areas: language abilities, executive function, and cerebral dominance. However, most studies did not assess these dimensions simultaneously. In a series of studies that contrasted executive function, verbal and spatial abilities, and tests of cerebral dominance, we first found that working memory, a basic ability involved in executive functions, was poorest in boys from a community sample with a history of physical aggression even after controlling for nonexecutive abilities relevant to executive function (Séguin et al., 1995).

To assess working memory we used the Self-Ordered Pointing (SOP) test, a number of randomization test (mid-dorsolateral frontal lobe), and conditional association tests (posterior dorsolateral frontal lobe; Petrides, Alivisatos, Meyer, & Evans, 1993b; Petrides et al., 1993a). We used the abstract and concrete versions of the SOP. Briefly, one trial consists of the selection of one of 12 different images in a 3×4 array by pointing at it. The images are repeated in varying positions on 12 such arrays. All 12 different images need to be chosen to obtain a perfect score. To succeed, the individual must monitor these self-ordered selections. To increase interference, two additional trials with the same set of images follow the first one. Errors can be computed within trials (and summed across trials). For the conditional association tasks, the individual must identify the underlying rule that associates each of six

pairs of stimuli. Feedback is provided, which also makes this an inductive learning task. At the behavioral level, a concurrent history of early hyperactivity is thought to increase the risk for "early-onset persistent" antisocial behavior (Lynam, 1998; Moffitt, 1990a). However, several studies fail to support this hypothesis (see, Farrington & Loeber, 2000; Lahey, Loeber, Burke, & Rathouz, 2002; Lilienfeld & Waldman, 1990; Loeber, Burke, & Lahey, 2002; Nagin & Tremblay, 1999; Stouthamer-Loeber, Loeber, Wei, Farrington, & Wikström, 2002).

Nonetheless, hyperactivity should be concurrently assessed in studies of violence because it co-occurs frequently with antisocial behavior problems, and there is considerable evidence linking executive function problems to hyperactivity in children (Nigg, 2005; Nigg et al., 2004). Studies examining the neuropsychology of hyperactivity, inattention, impulsivity, or conduct problems typically measure one behavioral dimension while controlling statistically for the others. In a follow-up to our first study, we controlled statistically for ADHD or teacher-rated hyperactivity and still found working memory impairments after also controlling for IQ (Séguin, Boulerice, Harden, Tremblay, & Pihl, 1999). However, we learned little about the neuropsychology of hyperactivity, and it remained possible that physical aggression and hyperactivity combined in additive or synergistic ways (Waschbusch, 2002). Moreover, by controlling for "comorbid" psychopathology, one may inadvertently be controlling statistically for important variance relevant to the disorder in question (Meehl, 1971).

Therefore, in a third study, we selected young adult males on the basis not only of having a physically aggressive history (teacher-rated from kindergarten to age 15), but also of having a history of hyperactivity (without a focus on inattention or impulsivity; Séguin, Nagin, Assaad, & Tremblay, 2004). We found no statistical interaction, but we did find clear additive effects, even after controlling for test motivation. Although the impairments observed included IQ and short-term memory, working memory remained significantly impaired even after statistical control for these other cognitive abilities. In other words, both physical aggression and hyperactivity exhibited significant independent associations with neuropsychological function and working memory. However, more of the tests in this battery were associated significantly and independently with physical aggression than they were with hyperactivity. Other studies of girls have also found a negative association between executive control and physical aggression after controlling for ADHD (Giancola, Mezzich, & Tarter, 1998). Studies of bullying (which includes threats of physical aggression) have revealed similar results, although they have not always controlled statistically for comorbid externalizing problems (Coolidge, DenBoer, & Segal, 2004).

In summary poor neuropsychological function is often found in adolescents and young adults from the community with a history of physical aggression. These deficits appear to be independent from other externalizing behavior problems. Developmentally, these associations can be detected as early as the preschool years (Séguin, 2003). Work on physical aggression is consistent with that position: the children who showed the greatest neuropsychological impairment had already been identified as physically aggressive and hyperactive in kindergarten (Séguin et al., 2004), and preschool physical aggression and hyperactivity trajectories were associated synergistically with cognitive performance at age $3\frac{1}{2}$ years (Séguin, Zelazo, & Tremblay, 2005). Séguin and Zelazo (2005) recently reviewed the literature on preschool cognitive function in early physical aggression. They noted that this literature was characterized by similar problems to those we are reviewing here (i.e., use of global measures of behavior problems without specific focus on physical violence, history of problem behavior not necessarily taken into account, and use of global measures of cognitive function instead of specific measures sensitive to frontal function). Nonetheless, they also noted that poor

cognitive function was often but not always associated with problem behavior in several studies of preschoolers.

Neuropsychological Studies of Antisocial Disorders in Which Physical Violence May Be Present

The bulk of research on the neuropsychology of violence derives from clinically oriented studies of disorders and antisocial behavior problems that may include physical violence.

ANTISOCIAL BEHAVIORS

Several of the behavioral problems listed here have been grouped under the global label of "antisocial behaviors." These behaviors, among others, were examined in a meta-analysis of studies of executive function by Morgan and Lilienfeld (2000). Although executive function is one narrow type of neuropsychological deficit, this study provides a global view of that literature. We then complement this global view with a more specific focus on each key antisocial behavior problem. This meta-analysis comprised 39 studies, yielding 4,589 subjects total. To be included in the meta-analysis, tests of executive functioning must have attempted to measure volition, planning, purposive action, or effective performance and either differentiated patients with frontal lesions from other patients or preferentially activated the frontal cortex in previous studies. To investigate whether antisocial behavior was related to executive functioning deficits per se rather than neuropsychological deficits in general, three neuropsychological tests that do not rely heavily on executive functioning were analyzed as "control" measures. The antisocial behavior groups used in the meta-analysis included individuals meeting criteria for one or more of the following antisocial behavior problems: ASPD, CD, psychopathic personality disorder, criminality, or delinquency.

The results of the meta-analysis indicated that the antisocial behavior groups performed significantly worse than comparison groups, with a combined and weighted effect size Cohen's d (Cohen, 1992) of 0.62 standard deviations. Two of the three neuropsychological tests that did not measure executive functioning also produced significant, albeit weaker, differences between groups, with the antisocial behavior groups performing significantly worse (effect sizes of $d = 0.34$ and $d = 0.39$ standard deviations). However, tests of the homogeneity of variance across samples yielded significant results, indicating that the effect sizes were heterogeneous. When considering the type of antisocial grouping used in the studies, the heterogeneity of effect sizes was reduced within each group, and all group effect sizes remained significantly different than zero. Moreover, criminality ($d = 1.09$, weighted $d = 0.94$) and delinquency ($d = 0.86$, weighted $d = 0.78$) were found to be associated most strongly with executive functioning deficits. Potential moderators, including age, sex, ethnicity, and IQ, were not associated with the magnitude of the observed effect sizes, although scores on some of these moderators (e.g., sex, ethnicity) were not reported in all studies.

Morgan and Lilienfeld (2000) identified several limitations to this research. First, they were not able to examine the potential influence of substance abuse or ADHD on the results because these potential confounds were not assessed systematically in most studies. Second, the executive functioning measures were not subdivided by the frontal brain regions (e.g., dorsolateral, orbitofrontal) with which they are believed to be primarily associated. Third, the finding that criminality and delinquency were associated with more pronounced executive functioning deficits than other conditions is difficult to interpret given the differences in comparison groups used across studies. For example, executive functioning studies frequently compared criminals or delinquents with normal or unselected samples, whereas psychopaths were frequently compared with nonpsychopathic criminals. The latter studies may have yielded lower effect sizes because they used more stringent comparison samples.

This meta-analysis provides an interesting backdrop against which to compare studies

with a focus on history of physical aggression. With a focus on physical aggression trajectories between ages 6 and 15 years as originally defined by Nagin and Tremblay (1999), we found that the standardized mean difference for a contrast between extreme groups (chronic versus never) was 0.66 for a number randomization task, 1.52 for Self-Ordered Pointing, and 1.12 for conditional association tasks (Séguin et al., 2004). For the current review we examined the two most severe groups (chronic physical aggression versus high desistance physical aggression) and found our standardized mean differences for those three tests to be, respectively, 0.33, 0.65, and 0.53. This finding supports the notion that clear neuropsychological impairments can be observed when we take into account the history and type of behavior (physical aggression).

At a developmental level, one prospective longitudinal study using behavioral assessments at ages 8 and 17 years suggests that poor spatial abilities at age 3 may be characteristic of "early-onset/persistent" antisocial behavior even after controlling statistically for hyperactivity, an effect that had largely disappeared by age 11 once social adversity had been controlled statistically (Raine et al., 2002). Although frontal tasks had not been administered and physical aggression was not specifically used to classify children, this finding is consistent with others (Séguin & Zelazo, 2005) and brings an additional prospective longitudinal component to this research. However, in another study based on ages 7–17 years antisocial behavior ratings, "child-limited" antisocial children were as impaired neuropsychologically as an "early-onset/persistent" group when tested in late adolescence (Raine et al., 2005).

PSYCHOPATHY

Psychopaths are more violent than other criminals (Hare, 1999) and consequently have received considerable research attention in the past 25 years. Initial reports of neuropsychological, especially frontal lobe, impairments in psychopaths (Gorenstein, 1982) have not been consistently replicated (Hare, 1984; Hart, Forth, & Hare, 1990; Hoffman, Hall, & Bartsch, 1987; Sutker & Allain, 1987). Possible exceptions are studies in which psychopaths' anxiety levels were taken into account (Smith, Arnett, & Newman, 1992) or when life adjustment was taken into account. In the latter case, the more successful psychopaths (not defined by the PCL-R) showed better dorsolateral frontal lobe function than less successful psychopaths (Ishikawa, Raine, Lencz, Bihrle, & LaCasse, 2001). Smith et al. (1992) found two significant effects (out of six expected) for the Block Design subtest of the Wechsler Adult Intelligence Scale (Wechsler, 1981) and for the Trail Making Test-B (TMT-B) after controlling for IQ and substance abuse, but only in a contrast between low-anxious psychopaths and low-anxious nonpsychopaths. Marginally significant effects of psychopathy were found in two studies conducted on inmates, one using the TMT-B ($p < 0.06$; Hart et al., 1990), the other using the Wisconsin Card Sorting Task (WCST; Lapierre, Braun, & Hodgins, 1995). The latter test is a relatively global and nonspecific measure of executive function (Heaton, 1981), in which the perseverative error score was significant at $p < 0.08$, and categories achieved (out of six) at $p < 0.07$. However, all effect sizes were in the moderate range. For Hart et al. (1990) we computed weighted effect sizes of $d = 0.40$ for comparing high versus moderate psychopaths, and of $d = 0.64$ for comparing high versus low psychopaths. And in Lapierre et al. (1995) we computed weighted effect sizes of $d = 0.48$ and $d = -0.49$ for errors and categories achieved, respectively. In that latter study, psychopaths performed significantly poorly on tasks purported to measure orbitofrontal lobe function. Because the evidence remained unclear, we agree with Lynam (1998) that it is still reasonable to consider neuropsychological factors in studies of psychopathy.

In support of that possibility, Morgan and Lilienfeld's (2000) table examining PCL-R and non-PCL-R defined psychopathy indicates that the average effect size of neuropsychological (executive functioning) deficits was $d = 0.29$ ($d = 0.25$

weighted) in both cases (see Table 2 in Morgan & Lilienfeld, 2000). These analyses included a wide variety of tests showing heterogeneous effect sizes ranging up to $d = 1.41$. Because these studies frequently compared psychopaths with nonpsychopathic criminals, as in the two studies with results approaching significance and for which we calculated effect sizes, any effects of neuropsychological function that are correlates of criminality could be attenuated when criminals are subdivided as a function of psychopathy. Finally, one complementary account for inconsistent findings suggests that psychopaths may experience greater difficulty with left- than right-hemisphere tasks, especially when heavy information processing demands are placed on the left hemisphere (Suchy & Kosson, 2005).

In addition to studies using more classic neuropsychological tests, psychopaths appear to experience greater difficulty in shifting a dominant behavior when contingencies are changed and reversed. Newman and colleagues have proposed two pathways to account for that impairment: (1) a difficulty in regulation of affect and (2) a more fundamental information-processing deficit related to attention; that is, a difficulty in shifting attention to peripheral but potentially meaningful information from the environment (Newman & Lorenz, 2002). We found that physically aggressive boys perseverate on such "emotion regulation" tasks even after controlling for neuropsychological function and that stability/instability of physical aggression may vary as a function of both pathways proposed by Newman and colleagues (Séguin, Arseneault, Boulerice, Harden, & Tremblay, 2002). Psychopaths typically perform better than nonpsychopaths on some Stroop interference type tasks when stimuli are not totally embedded into one another, such as for standard color-word Stroop on which they show the expected interference effects (Hiatt, Schmitt, & Newman, 2004). According to the authors of that study psychopaths can focus (if not perseverate) better on a dominant rule and ignore potentially interfering information. Interference from such peripheral information is often adaptive for most individuals and helps them adjust their course of action or modulate their response.

Psychopathy has also received attention from neuroscientists who observed the psychopathic-like behavior that we described earlier following brain lesions involving the orbitofrontal or ventromedial frontal lobes. Studies of the effects of these lesions on behavior offer particularly interesting results, because lesions are often relatively circumscribed with an absence of typical neuropsychological impairment. Such lesions were thought to be associated with problems on the Iowa Gambling Task (Bechara, Damasio, Damasio, & Anderson, 1994) and the Intradimensional/Extradimensional Shift Task from the Cambridge Neuropsychological Test Automated Battery (CANTAB; Downes et al., 1989). The Intradimensional/Extradimensional Shift Task breaks down confounded stages of the WCST, is also essentially inductive, and consists of up to 10 progressive stages that involve visual discrimination within and between dimensions (using the dimensions of color, shapes-lines, and number), as well as shifts of reward contingencies (or underlying correct rule). These tasks are clearly described by Mitchell, Colledge, Leonard, and Blair (2002).

The Iowa Gambling Task requires participants to choose a card from one of four decks. After choosing, the participant receives either a financial reward or punishment. There are two "good" decks that offer small to moderate rewards and are associated with little chance of punishment, and two "bad" decks that offer large rewards but are much more likely to result in punishment. Healthy controls often choose the "good" decks, whereas lesioned patients more often choose the "bad" decks. Patients with such lesions are said to be insensitive to future consequences of their decisions. The "somatic marker hypothesis," which is consistent with these observations, suggests that ventromedial frontal lobe lesions impair the capacity to consider emotions when making decisions (Damasio, 1996; Damasio et al., 1991). Despite an initial failing to

extend the Iowa Gambling Task to adult psychopaths (Schmitt, Brinkley, & Newman, 1999), PCL-R psychopaths (Mitchell et al., 2002) and boys with psychopathic tendencies (Blair, Colledge, & Mitchell, 2001) did show poor performance on that task when the original methodology was applied. Nonetheless, there remain profound differences between psychopaths and individuals with these acquired conditions. For example, the aggression of brain-injured patients is typically reactive/hostile as opposed to the aggression of psychopaths, which is likely to be proactive/instrumental (Blair & Cipolotti, 2000; Cornell et al., 1996). Moreover, poor performance on the Iowa Gambling Task may be attributable to mechanisms other than insufficient somatic marking. For example, individuals may perform poorly on this task as a consequence of inadequate impulse control or reward dominance, or of poor working memory.

Recent reviews have also questioned the orbitofrontal account of psychopathy (Blair, 2004; Séguin, 2004). Although impairments on the Iowa Gambling Task (Bechara et al., 1994) and the Intradimensional/Extradimensional Shift Task (Mitchell et al., 2002) may be similar in psychopathy and in acquired psychopathy, it now appears that neither task is sensitive to *focal* orbitofrontal lesions (Manes et al., 2002). Earlier conclusions about the relevance of the OFC on these tasks relied on cases with lesions extending to the orbitofrontal cortex, but specific orbitofrontal lesions actually appear to increase deliberation and reduce impulsivity, quite the opposite of what was initially expected. Thus, the specific role of the orbitofrontal cortex in antisocial behavior may need to be re-examined.

Much research now focuses on the amygdala, which assigns motivational value to stimuli. Both psychopaths and individuals with amygdala lesions appear to exhibit poor recognition of fear and sadness (Blair et al., 2002), although their own experience of fear and sadness may be unimpaired (Anderson & Phelps, 2002). This finding constitutes an interesting dissociation between perception and subjective experience. The amygdala may be involved in the reactive aggression of psychopaths, because it is considered to be a key component in the regulation of such aggression in a complex circuit that involves the orbital frontal lobe and the anterior cingulate cortex (Davidson, Putnam, & Larson, 2000). It is important to reiterate, however, that instrumental aggression appears to be more central to psychopathy (Cornell et al., 1996). This line of research gave rise to the violence inhibition mechanism model of psychopathy, which posits that psychopaths exhibit deficiencies in a system that preferentially responds to sad, and more specifically fearful, emotional displays (Blair et al., 2004). From a developmental perspective, Blair and colleagues suggested that amygdala dysfunction may predate OFC impairments, as only amygdala function seems impaired in children with psychopathic tendencies, whereas both amygdala and OFC functions appear impaired in adult psychopaths (Mitchell et al., 2002).

Criminality and Delinquency

Physical violence is considered to be a violent offense from a legal/judicial point of view. It is rarely officially sanctioned in children, but is part of official delinquency in adolescence and criminal behavior in adulthood. Thus, violence present in any of the disorders reviewed here is also likely to be legally sanctioned. However, there are cases in which violence occurs that are not necessarily in the context of a mental disorder. Such is the case when we study physical aggression and violent criminals in childhood and adolescence (reviewed above). Morgan and Lilienfeld (2000) found their strongest effect sizes on executive function for criminals and delinquents. One recent study of incarcerated youth found them to perform more poorly than nonoffenders attending public schools on spatial span (a spatial analogue to the digit span task), a measure of short-term memory from the CANTAB tests, but not on such frontal tasks as the Intradimensional/Extradimensional

Shift Task, Tower of London, or spatial working memory task (Cauffman, Steinberg, & Piquero, 2005).

Some of the important theoretical work developed by Moffitt centered around delinquency initially (Moffitt, 1990b; Moffitt & Henry, 1989, 1991; Moffitt, Lynam, & Silva, 1994; Moffitt & Silva, 1988a,b). In one study, "early-onset/persistent" delinquents performed more poorly than "late-onset" delinquents on IQ and other neuropsychological tests (Taylor, Iacono, & McGue, 2000), as predicted by Moffitt's (1993a) developmental theory of antisocial behavior. In this study, however, IQ was not used as a co-variate, rendering it difficult to make conclusions regarding the specificity of cognitive deficits.

The use of global scales of delinquency or criminality may obscure key relations between specific behaviors and neuropsychological function. For example, one study found that among juvenile delinquents, IQ was positively related to theft, but negatively related to violence (Walsh, 1987). Presumably, this finding reflects the requirement for planning for theft, but an impulsive problem-solving style for violence. Intrigued by this isolated report, we recently analyzed data from the Rutgers longitudinal study (White, Bates, & Buyske, 2001). In that study, theft and violence were initially combined within a global index of delinquency. Neuropsychological function had failed to separate "persistent" from "adolescence-limited" delinquents. Following the lead from Lacourse et al. (2002) and our own work with physical aggression and hyperactivity (Séguin et al., 2004), we identified trajectories for theft and for physical violence. Using the same tests as the original study, we replicated Walsh's (1987) finding and found poorest neuropsychological function in highly violent individuals who were low on theft (Barker et al., in press). This replication requires further study, but suggests the need for a finer parsing of the relation between neuropsychological function and global indices of antisocial behavior.

Much research in the neuropsychology of violence may also bear implications for the justice system. Decisions to try adolescents who committed violent crimes in adult courts hinge on research on brain development and maturation. Beckman (2004; see also Sommers & Satel, 2005) recently reviewed that issue. However, the bulk of the literature reviewed by Beckman ignored the brain/violence literature, including that reviewed here. From a brain maturation perspective, some claim that adolescent brains are not sufficiently mature to justify trying violent adolescents in adult courts. However, research shows that the overwhelming majority of adolescents are not violent. Further, impaired brain function has been found in many violent (and nonviolent) adults. These data raise the question of why legal decision making should use age per se as a criterion. Moreover, neuropsychological function (and possibly brain maturation) may account only for a relatively small amount of variance in physical violence, although it could be an important influence in some cases. Thus the sensitive issue of trying violent adolescents in adult courts should probably rest on broader grounds, and evidence reviewed herein to inform the legal decision-making system should be used with caution.

Intermittent Explosive Disorder

The DSM-IV (American Psychiatric Association, 2000) places intermittent explosive disorder (IED) under the category of impulse control disorders not elsewhere classified. The primary feature of IED is the experience of discrete episodes of aggressive behavior resulting in personal injuries or property damage. The course, onset, and prevalence of IED are poorly understood, although this condition appears to be more common in males than females. Not surprisingly, there is a dearth of studies on the neuropsychological correlates of IED. In the only published controlled study, Best, Williams, and Coccaro (2002) show that the IED participants performed poorly on the Iowa Gambling Task compared with controls. However, there were no group differences on the Self-Ordered Pointing test, a

working memory test most sensitive to physical aggression (Séguin et al., 2004).

Conduct Disorder

As mentioned earlier, Moffitt (1993b) has reviewed the literature on the neuropsychology of CD and identified deficits in three major areas: language, executive function and cerebral dominance. Several studies supported that distinction (Lueger & Gill, 1990). However, except for our early work on physical aggression (see Séguin et al., 1995), the evidence for the specific involvement of executive function in CD per se was weak, partly because ADHD was rarely controlled (Pennington & Ozonoff, 1996). It seems that not much has changed since Pennington and Ozonoff's (1996) review (Nigg, Willcutt, Doyle, & Sonuga-Barke, 2005). Several studies find neuropsychological impairment in ADHD even after statistical control for CD. However, authors of these studies often recognize that CD is not an object of study in itself (see, e.g., Nigg, Hinshaw, Carte, & Treuting, 1998). In other words, it is possible that there might have been unique variance related to CD as well after controlling for ADHD. Indeed, a few additional studies have examined CD and contrasted it with ADHD, also in the hopes of addressing the issues of specificity and comorbidity. When Morgan and Lilienfeld (2000) reviewed the CD literature they found an effect size of $d = 0.4$ (weighted $d = 0.36$) for poor performance on executive functioning tasks. Not included in that review was one study of explicitly violent CD females that found poor executive function even after controlling statistically for ADHD (Giancola et al., 1998). Otherwise, when physical violence and ADHD have not been examined, mixed results have been typical, even from the same investigative team. For example, one group of researchers failed to find executive function problems but found verbal problems in CD adolescents (mean age 15.4 years) after controlling statistically for ADHD (Déry, Toupin, Pauzé, Mercier, & Fortin, 1999). However, this research group found an executive functioning deficit after controlling statistically for ADHD in a subsequent study of children (age range of 7 to 12 years; Toupin, Déry, Pauzé, Mercier, & Fortin, 2000). We note that the level of participants' physical violence was not clear in these two studies. The authors also speculated that differences between studies may be explained by (a) a lack of sensitivity of verbal measures in the child study; (b) the possibility that the CD adolescent group could have contained a mixture of "life-course persistent" and "adolescent-limited" delinquents (theoretically less physically aggressive); and (c) the possibility that the discrepancy between groups may increase with development (J. Toupin, personal communication, February 14, 2006). With regards to this last proposition, Blair and colleagues formulated a similar hypothesis in their developmental model of psychopathy to account for discrepancies between their child and adult data (Mitchell et al., 2002). The most relevant demonstration of a need to account for physical aggression are the findings that within CD, violence and theft are related in an opposite direction (violence positively, and theft negatively) with neurocognitive dysfunction (Barker et al. in press).

We provide an example to illustrate further the potential importance of accounting for physical aggression in studies of the neuropsychology of antisocial behavior. In one study that focused on physical aggression and hyperactivity, 67% of boys classified as CD and 72% of boys classified as ADHD between the ages of 14 and 16 years were in the high physical aggression and high hyperactivity trajectories, respectively, on the basis of teacher ratings from ages 6 to 15 years (Séguin et al., 2004). High trajectories in this case were a combination of the two highest ("chronic" and "high desistor") versus the two lowest ("low desistor" and "never") trajectories identified by Nagin and Tremblay (1999) for both physical aggression and hyperactivity. However, across the entire sample only 5% and 6.7% of boys met criteria for CD or ADHD, respectively. This finding suggests that if one is high in physical aggression (or

hyperactivity) one is more likely to meet criteria for CD (or ADHD). But that study was different from the typical studies selecting for CD and ADHD in that it selected children on the basis of physical aggression and hyperactivity instead. A selection based on CD and ADHD may not necessarily have included either physical aggression or hyperactivity, or these specific behaviors may have contributed only a low weight to the diagnoses. Thus, a focus on physical aggression and hyperactivity yielded a greater number of study participants, most of whom did not meet criteria for CD or ADHD, and those more specific behaviors were sensitive to neuropsychological function and executive function in particular (Séguin et al., 2004).

A Note on CD/ODD

There has been considerable literature contrasting ODD/CD with ADHD. A problem arises mainly when this literature is used to make claims that there are no neuropsychological impairments in CD when ADHD is taken into account. ODD/CD is a loose combination of either ODD or CD symptoms, not CD per se. As we mentioned earlier, the diagnosis of ODD does not contain physical aggression symptoms and CD children may or may not show such symptoms. Neuropsychological impairments might be found in CD when physical aggression is present, as some studies have shown. It is not surprising to find that most of these studies report that poor neuropsychological function is found mainly in ADHD (or when ADHD is combined with ODD/CD; see Clark, Prior, & Kinsella, 2000; Geurts, Verté, Oosterlaan, Roeyers, & Sergeant, 2004; Kalff et al., 2002; Nigg et al., 1998; Oosterlaan, Scheres, & Sergeant, 2005) although issues of statistical power plague these studies (Clark et al., 2002). Conclusions from these studies are also consistent with those that compared ADHD with ODD (Speltz, DeKlyen, Calderon, Greenberg, & Fisher, 1999) when it is not necessarily combined with CD. Further, these studies, as well as studies contrasting conduct problems (CP) and hyperactivity/impulsivity/attention (HIA), typically compare groups using one-factor designs (e.g., lowCP-lowHIA, lowCP-highHIA, highCP-lowHIA, highCP-highHIA) instead of considering the two behavioral dimensions (e.g., CP high & low and HIA high and low) as independent factors. The latter designs are required to determine if the effects are additive or synergistic (Waschbusch, 2002).

CD Summary

In summary, results from CD research have been mixed. Several reasons may account for this unclear picture. At the behavioral level, there is often no specific inclusion of physical aggression and no control for ADHD or hyperactivity. Moreover, many of these studies combine CD with ODD, equate delinquency or antisocial behavior with CD, derive estimates of CD from behavior scales not necessarily designed for such a purpose, and fail to take the history of problem behavior into account. At the neuropsychological level, there is often no statistical control for IQ or verbal ability in studies examining constructs related to executive function. Moreover, many studies are possibly overinclusive in describing some tasks as assessing executive function, as such studies sometimes make the questionable assumption that one task of executive function represents the entire construct.

Antisocial Personality Disorder

ASPD is diagnosed only in individuals over the age of 18 with a history of CD. As we mentioned above, psychopathy is often found among individuals with ASPD. We also note that PCL-R scores >20 but <30 (the PCL-R cut-off point for psychopathy) may reflect ASPD also and that many ASPD individuals have a criminal record. There are few neuropsychological studies of ASPD. Morgan and Lilienfeld (2000) had noted two studies that examined executive function (Deckel, Hesselbrock, & Bauer, 1996; Malloy, Noel, Rogers, Longabaugh, & Beattie, 1989). Overall, they found that

ASPD associations with executive function are weak (effect size $d = 0.10$, weighted $d = 0.08$; Morgan & Lilienfeld, 2000), as was also found in subsequent studies of neuropsychological function (Crowell, Kieffer, Kugeares, & Vanderploeg, 2003; Dinn & Harris, 2000; Stevens, Kaplan, & Hesselbrock, 2003). For example, in one study that controlled for concurrent alcohol and substance abuse, ASPD symptoms were negatively and significantly correlated with WAIS similarities scores but no other intelligence nor executive functioning variables (Stevens et al., 2003). In that study, the strongest correlate of executive function was family history of alcoholism. Finally, one brain imaging study had noted an 11% reduction in frontal gray matter in ASPD even after control for psychosocial factors, but with unclear neuropsychological significance (Raine, Lencz, Bihrle, LaCasse, & Coletti, 2000).

Other Disorders in Which Violence May be Present

Although violence is likely to be found in the more explicitly antisocial disorders that have already been reviewed, violence is often associated as well with alcohol and substance use disorders, schizophrenia, and bipolar disorder. Hence, we briefly review their relation to neuropsychological function.

ALCOHOL AND SUBSTANCE USE

A considerable limitation to the literature investigating neuropsychological function in violent problem behaviors and associated disorders is a potential confound with alcohol and substance abuse. In fact, it is possible that most violent acts occur under the influence of substances. This is relatively well documented in the case of alcohol (Murdoch, Pihl, & Ross, 1990), where there is a clear relation between per-capita alcohol consumption and homicide (studies effect size $d = 0.22$; Rossow, 2001), although the relation may not be causal (Room, Babor, & Rehm, 2005). The culture of illegal drugs is also associated with violence, but that is not dispositive evidence for the violence potential of the drugs themselves. As part of the mechanisms, we note that alcohol increases the heart rate of nonalcoholic young men with a family history of alcoholism and who also show a history of aggressive behavior (Assaad et al., 2003). These individuals tend to consume more alcohol and make more commission errors (i.e., pressing a button when a "No-go" signal is presented) on a Go/No-go task when intoxicated as opposed to sober, and as opposed to low heart rate responders (whether they were intoxicated or sober, Assaad et al., 2006). Although performance on Go/No-go tasks may not be entirely under frontal lobe control, a family history of alcoholism may be associated more strongly with poor neuropsychological function than ASPD (Stevens et al., 2003).

Our central question, however, concerns the role of neuropsychological factors in this equation. This issue may be best summarized by the work of Giancola (Giancola, 2000) and Pihl (Hoaken, Giancola, & Pihl, 1998). Giancola (2004) showed that aggression in reaction to provocation (using a shock paradigm) was possibly more a function of executive function than of alcohol. However, synergistic mechanisms may be at play, as alcohol preferentially increased aggression in men with lower levels of executive function. In this way, executive function could act both as a mediator and moderator of the alcohol-aggression relationship. However, alcohol could also moderate the quality of executive function on a state (temporary) basis. Indeed, alcohol impairs executive function, mostly on the descending limb of the blood alcohol curve (Pihl, Paylan, Gentes-Hawn, & Hoaken, 2004). Finally, a similar literature exists for substance abuse (Fishbein, 2000).

Schizophrenia

In a recent review, Pontius (2003)noted that less than 10% of violence can be attributed to psychosis, but that the odds of violence in schizophrenia as opposed to no mental disorder is about 4:1. She also pointed out that violence in schizophrenia is often intentional and planned, but that it derives from seriously distorted thinking, delusions,

or hallucinations. Such violence occurs typically in paranoid schizophrenia, and neuropsychological function may be otherwise intact. One study of paranoid schizophrenia noted that violence was associated with poor theory of mind (Abu-Akel & Abushua'leh, 2004). Theory of mind is a key social ability to attribute mental states to self and others and requires a broad network including the frontal cortex and cingulate (Calarge, Andreasen, & O'Leary, 2003). A separate neural system underlying belief attribution would complement systems underlying inhibitory control (Saxe, Carey, & Kanwisher, 2004). Although violence was also associated with a history of alcohol and drug abuse (Abu-Akel & Abushua'leh, 2004), another study of murderers showed increased risk for schizophrenia and delusional disorder even after controlling for a history of alcoholism (Schanda et al., 2004). Nevertheless, neuropsychological problems in schizophrenia can be broad and manifold and include attention, memory, executive control, language, and reasoning (Barch, 2005; Heinrichs, 2005), but none of these has yet been specifically related to violence.

Bipolar Disorder

Bipolar disorder is associated with several reckless behaviors characterized by impulsivity, poor judgment, and poor planning (Moeller, Barratt, Dougherty, Schmitz, & Swann, 2001). Bipolar disorder may be associated with violence when it is comorbid with alcohol disorders (Schanda et al., 2004) or substance use disorders (Quanbeck et al., 2005). It is sometimes comorbid with CD. However, preliminary studies of this comorbidity suggest that bipolar disorder does not appear to add to the neuropsychological impairment found in CD (Olvera, Semrud-Clikeman, Pliszka, & O'Donnell, 2005). This finding is consistent with other studies of acute mania in which neuropsychological deficits were stronger in sustained attention and verbal learning than on typical neuropsychological tests associated with CD, such as tests of executive function and the Iowa Gambling Task (Clark, Iversen, & Goodwin, 2001). However, other studies report executive function impairments even in the nonacute phase, although bipolar patients in either manic or euthymic phase did not differ much from depressed patients (Martinez-Arān et al., 2004).

Chapter Summary and Concluding Comments

The neuropsychology of antisocial behavior has a rich history dating back at least to the mid-19th century. Unfortunately, as our review shows, not as much can be said about the neuropsychology of physical violence. The main problem is that the specificity of neuropsychological deficits to physically violent behavior has been difficult to establish. The bulk of our knowledge regarding the neuropsychology of violence derives from studies of clinical syndromes in which the presence of violence is plausible, but rarely confirmed. Global measures of antisocial, disruptive, externalizing, delinquent, or criminal behavior are also often used. This state of affairs reflects the heterogeneity of processes underlying those conditions, although some factors may be common to all of these conditions. For example, we recently found that the relation between IQ and externalizing behavior problems was moderated by a variation in the dopamine D4 receptor (DeYoung et al., 2006); there was no relation between IQ and externalizing behavior in those who had the 7 repeat allele. We replicated this finding in one community sample and two clinical samples. This moderating effect held across and, in some cases within, externalizing behavior problems. Thus, global measures do have their utility.

Nonetheless, as this book shows, violence is a common outcome in a wide variety of heterogeneous conditions. Violence is present in many disorders for which there may be a neuropsychological basis, although that neuropsychological basis may not necessarily be for violence per se. To advance a research agenda in the study of the neuropsychology of violence, we recommend

testing for the specificity of behavior problems, such as identifying physical aggression and isolating it from other co-occurring behavior problems. More explicit assessment of violence or physical aggression is needed with key contrasts to such disorders as ADHD, alcoholism, and nonviolent forms of antisocial behavior. Although this approach should reduce the heterogeneity of behavior of interest, it may not reduce the heterogeneity of underlying processes as much as one hopes; subtypes of physical violence also need to be addressed. The few studies that included a focus on physical aggression or violence did find clear and large effects. But more of these studies are required to investigate the value of this research strategy. A well-documented history of behavior problems should also help reduce heterogeneity. The study of the process of desistance from violence would be informative in this regard.

Our review shows that neuropsychological impairments, even in executive function, are not necessarily specific to physical aggression. We first observed that lesions among frontal lobe patients, despite their poor executive function, rarely lead to physical violence even when acquired at very young ages. Moreover, there is considerable literature on other problems, such as ADHD, and their relation to executive function. However, we note that studies in the ADHD literature rarely control for co-occurring physical aggression. Further, neuropsychological factors tend to explain at most 10% of the variance in measures of violence. Therefore, future studies need to examine potential moderators that may increase our ability to predict violence risk from neuropsychological dysfunction. Such factors could include a history of abuse or neglect, malnutrition, abilities to process and regulate emotions (including autonomic arousal), capacities to cope with stress and perceived provocation, perinatal factors, and genetic factors such as those we referred to earlier (see, e.g., DeYoung et al., 2006). Although many of these variables have been widely studied, few have been examined in conjunction with neuropsychological function. In this context, poor neuropsychological function will probably comprise only one element of an exceedingly complex model of violent behavior. As this chapter illustrates, however, it may provide one essential piece of a still unsolved puzzle.

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