
Pergamon

Aggression and Violent Behavior
10 (2004) 1–30

AGGRESSION AND VIOLENT BEHAVIOR

Affective and predatory violence: A bimodal classification system of human aggression and violence ☆

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Received 29 April 2002; received in revised form 6 June 2003; accepted 19 June 2003

Abstract

The etiology of violent and aggressive behavior has been studied for several decades. Observations in the 1920s of human patients who manifested aggressive behavior after incurring neurological insults led researchers to explore a biological basis for the behavior. Animal research soon followed and provided the foundation for understanding this complex behavior. Efforts to use animal models of adaptive aggressive behavior to explain pathological aggression in a subgroup of the human population has proven to be a daunting task. The research has produced a vast database encompassing several distinct disciplines. *Predatory* and *affective* aggression garners support as a classification system from clinical, social, biopsychological and forensic databases. This article draws together this vast research and delivers an argument for a bimodal classification system of aggressive and violent behavior.

Keywords: Predatory; Affective; Violence; Aggression; Forensic

The study of aggression is complex and multifaceted. Research groups have focused on key aspects of aggression without necessarily integrating findings across disciplines. This has become problematic for the advancement and application of the research, as there is no universal language or agreement through which to communicate findings across the various databases. This article integrates information from various fields of research to form an applied understanding of aggressive and violent behavior. The author proposes that there are

☆ This research was supported by a grant from Forensis.
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1359-1789/$ – see front matter © 2003 Published by Elsevier Ltd.
doi:10.1016/j.avb.2003.06.002

comprehensive and compelling data supporting two primary modes of aggressive and violent behavior, *affective* and *predatory*, that both elucidates and subsumes the multiplicity of typologies that attempt to parse out this mode of behavior. Existing research can be generally divided into three areas: animal, clinical, and forensic. Within each of these domains, information from observational studies, neuroanatomy, and neurochemistry form what we currently know about violence and aggression. This descriptive model of aggression will also be linked to special populations encountered in the fields of forensic psychology and psychiatry.

1. Animal research on aggression and violence

Early investigations of aggression focused on animal models (Bard, 1928; Bard & Rioch, 1937; Cannon & Britton, 1925), which provided categorization of observable behavior (Bard, 1928), evidence for neuroanatomical correlates (Bard & Rioch, 1937), and evidence for neurotransmitter correlates of aggression (Cannon & Britton, 1925). The premise has been that utilizing animal models to explore and understand the biological and environmental mechanisms that underlie aggressive behavior may provide a framework for understanding the origins of aggression in humans.

1.1. Initial behavioral classifications and hypothalamic precursors of aggression

Through stimulation and ablation experiments in a number of species, researchers have systematically been able to trace the neural pathways through which the hypothalamus influences other areas of the brain involved in the production of rage and attack behavior (Anderson & McCann, 1955; Fonberg, 1967; Hess & Brugger, 1943; Hunsperger, 1956; Nakao, 1958; Roberts, Steinberg, & Means, 1967; Wasman & Flynn, 1962; Wyrwicka & Doty, 1966). The basic technique used in such analysis involves stimulation of various aspects of an animal's brain, most typically that of a cat, and the elicitation of various forms of attack on an anesthetized rat.

Early experiments focused specifically on the role of the hypothalamus in aggressive behavior (Hunsperger, 1956; Sheard & Flynn, 1967; Wyrwicka & Doty, 1966) Such experiments typically involved electrical and chemical stimulation of the hypothalamus of a cat placed in an otherwise stimulus-free environment (Hess & Brugger, 1943; Hunsperger, 1956; Masserman, 1941, 1943; Nakao, 1958). Although the role of the hypothalamus was not made completely clear in these early studies, results from these experiments led to some classification of the cat's aggressive behavior (Bard, 1928; Masserman, 1941, 1943).

Bard (1928) and Bard and Rioch (1937), who experimented with decorticated cats in 1928, were one of the first researchers to attempt to elucidate the role of the hypothalamus in aggressive behavior. After sectioning the structures rostal to the hypothalamus, Bard found that the cats would exhibit sham rage responses with little or no provocation. The heightened sympathetic and somatic arousal seen in the stimulated cats was referred to as *sham rage* or a *pseudo-affective* response (Masserman, 1941). The spurious quality suggested by these terms

indicated a marked affective display without the behavior culminating in a directed attack. Based on his research, Masserman (1941) suggested that this behavior was purely a motor phenomenon devoid of any forebrain involvement. Subsequently, the importance of perceptual elements necessary to direct and guide the attack behavior became a focus of investigation.

The first such observations of the importance of perceptual elements came from Hess and Akert (1955), who noted that Masserman's stimulated cats sometimes attacked the experimenter. This finding brought into question the role of appropriate environmental cues for eliciting the full range of attack behavior. A cat exhibiting this behavior will modify it if perceptual elements within the stimulus field require it (Flynn, Vanegas, Foote, & Edwards, 1970). This display of higher order behavior suggested forebrain involvement and later established that the sham rage was in fact indisputable if the appropriate targets for the cat were available. Studies that closely examined the importance of targets varied exposures from a dead rat to a live one, to stuffed animals, cat food, and other cats, to name a few (Wasman & Flynn, 1962). These studies indicated that cats typically prefer a target that most closely approximates their preferred animal of prey, the rat.

With appropriate targets in place, investigators observed and described two distinct patterns of attack: *affective defense* and *quiet biting attack. Affective defense behavior*, a term coined by Hess and Brugger (1943), described the behavioral and autonomic arousal seen in cats following electrical stimulation of the hypothalamus. The behavior was interpreted as defensive because there was no directed attack and the cat did not attempt to flee the situation. Masserman (1941) supported this interpretation and regarded the displays of rage as a pure motor phenomenon having little to do with affective experience and behavior. While rage responses without directed attack can be elicited through stimulation of the hypothalamus, such purely affective responses are more reliably produced in the central gray region surrounding the aqueduct of Sylvius (Bandler, 1982; Shaikh, Brutus, & Siegel, 1986). As research produced greater specificity in identifying activating sites within the hypothalamus, the notion of affective defense as a proper label was reconsidered. Wasman and Flynn (1962) questioned whether it was an appropriate characterization, noting that the cat's behavior could hardly be considered defensive given that an anesthetized rat could offer little threat. The cat's behavior was observed to be clearly aggressive, and earlier observations of the cat's attempts to flee were not reliably reproduced in later experiments (Hess & Brugger, 1943). Affective attack, whether or not it involves contact with a target, was clearly associated with avoidance of a noxious or punishing stimulus, usually associated with fear, which may cause the animal to flee or strike out. Although the term *affective defense* continues to appear in the literature, some attempts have been made to redefine the term to exemplify what it actually represents, affective attack.

The term *affective attack* is currently understood to encompass behaviors one might associate with that of a stereotypical Halloween decoration of an intimidating cat. Behavioral alerting and dilation of pupils characterize the cat's initial response. As the stimulus is increased, piloerection down the midline becomes prominent and hair along the tail becomes fluffed out. Breathing may be heavy and audible. The cat will begin to hiss and growl, and typically will unsheathe its claws. The animal may arch its back to make itself look larger and more threatening. As the cat approaches the target, typically a rat, it will be vigilant of any

movement, which may trigger an immediate attack. At other times, the cat may test the prey with a few directed blows with its paw or may pounce on the rat in a wild manner, tearing at it with its claws.

The second form of attack, quiet biting "predatory" attack or stalking attack (Wasman & Flynn, 1962) represents a predatory-like stance toward an object of prey. Like affective attack, it begins with pupil dilation and behavioral alerting. However, this is where the similarity ends, as most overt sympathetic signs are usually absent. Rather, the cat tends to be unusually quiet and lowers its profile to avoid detection. The attack comes quickly and the apprehension seen in affective attack is transformed into a swift and directed approach to the rat. Instead of using its claws, the cat executes the attack with its mouth to the neck or head of the rat, and it is more precise in delivering a series of deadly blows.

Forty years after the pioneering work of Bard, and in the midst of Flynn and colleagues' (Flynn, 1976, 1977; Flynn et al., 1970; Flynn, Edwards, & Bandler, 1971; Wasman & Flynn, 1962) burgeoning research on the anatomical substrates involved in affective and predatory aggression, Moyer (1968) described seven categories of aggressive behavior displayed by animals. Moyer defined aggressive behavior as one "which leads to, or appears to an observer to lead to, the damage or destruction of a goal entity" (p. 66). Moyer's seven categories of aggressive behavior came from observational studies of animals in the field and in the laboratory. The categories are (1) predatory, (2) intermale, (3) fear-induced, (4) irritable, (5) territorial, (6) maternal, and (7) instrumental. Moyer's predatory category most closely resembles that of the quiet biting "predatory" attack described by Wasman and Flynn (1962). Later, Reis (1971, 1974) restructured Moyer's categories, recognizing predatory attack and collapsing the six other categories into one affective aggression. Reis supported this bimodal classification with observation that except for the category of predatory attack, the other six divisions all feature sympathetic arousal resulting from similar neuropathways. These categories differentiate themselves from the discrete, mutually exclusive sites that elicit predatory aggression. The return to a bimodal classification of aggression remains the approach of contemporary research.

1.2. Neuroanatomical substrates of feline aggression

Historically, the limbic hypothalamic–midbrain axis was the focus for understanding the production of affective attack and quiet biting "predatory" attack in cats. Over time, much has been learned about the various brain regions involved in affective and predatory violence. The hypothalamus is considered the principal autonomic center in the brain concerned with crude or elemental emotions and involuntary functions. The hypothalamus also plays an important role in the organization of goal-seeking behaviors, such as feeding, drinking, mating, and aggression. The hypothalamus, contained within the limbic system, lies ventral to the thalamus and forms the floor and part of the inferior lateral walls of the third ventricle. It receives input from many parts of the limbic system and the reticular formation, as well as from other nuclei in the brainstem (Reitan & Wolfson, 1992).

The limbic system is a collection of interconnected telencephalic and diencephalic structures believed to be involved in the regulation of motivated behaviors, including fleeing,

feeding, fighting, and sexual behavior (Pinel, 1993). There are several large subcortical structures, most of which are considered part of either the limbic system or the basal ganglia motor system (Pinel, 1993). Located at the juncture of the forebrain and the brainstem, they include the hippocampal formation, amygdala, septal area, cingulate gyrus, and prefrontal cortex (Pinel, 1993). Below the forebrain and contained within the midbrain lies the periaqueductal gray matter, which appears to be a focal point for the integration and organization of both quiet biting "predatory" attack and affective attack responses (Pinel, 1993). The sites of these responses in the central gray region are relatively close to, but independent from, each other (Pinel, 1993).

The two modes of behavior share limbic structures, including the ventral and dorsal hippocampus, septal area, amygdala, and portions of the prefrontal cortex and cingulate gyrus (Berntson, 1973; Bandler, McCulloch, & Dreher, 1984, 1985; Brutus, Shaikh, Edinger, & Siegel, 1986; Brutus, Shaikh, Edinger, Siegel, & Siegel, 1984; Chi & Flynn, 1971; Edwards & Flynn, 1972; Flynn et al., 1970; Mirsky & Siegel, 1994; Proshansky, Bandler, & Flynn, 1974). In addition, both modes of aggression have been traced directly to sites in close proximity to each other in the periaqueductal gray matter (Bandler et al., 1984, 1985; Brutus et al., 1984, 1986; Berntson, 1973; Chi & Flynn, 1971; Edwards & Flynn, 1972; Flynn et al., 1970; Mirsky & Siegel, 1994; Proshansky et al., 1974).

1.2.1. Affective attack

Affective attack can be elicited over a wide extent of the medial hypothalamus, starting from the preoptic region and extending back through the posterior hypothalamus into the dorsal aspect of the midbrain periaqueductal gray matter. A cat's behavior of hissing, growling, and piloerection results from autonomic arousal. The expression of the attack involves the central tegmental fields of the midbrain and pons, locus coeruleus, and motor and sensory nuclei of the trigeminal complex (Bandler et al., 1984, 1985; Berntson, 1973; Brutus et al., 1986; Flynn et al., 1970; Mirsky & Siegel, 1994).

1.2.2. Predatory attack

Through electrical, chemical, and ablation experiments, predatory attack has been reliably elicited from areas in the forebrain and brainstem. In particular, researchers have identified the lateral and perifornical hypothalamus and have traced eliciting sites as far caudally as the ventral tegmental area and the midbrain central gray (Bandler et al., 1984, 1985; Berntson, 1973). The regions known to modulate predatory attack behaviors include the structures of the midbrain periaqueductal gray, locus coeruleus, substantia innominata, bed nucleus of the stria terminalis, and central nucleus of the amygdala (Bandler et al., 1984, 1985; Berntson, 1973; Brutus et al., 1986; Flynn et al., 1970; Mirsky & Siegel, 1994).

1.3. Neurotransmitters and the regulation of aggression and violence in animals

Neurotransmitters and neuromodulators play a key role in the expression or suppression of affective and predatory attack. The study of neurochemical substrates involved in the facilitation and inhibition of aggression has focused primarily on acetylcholine, serotonin,

norepinephrine, dopamine, and the amino acid neurotransmitter gamma aminobutyric acid (GABA).

Acetylcholine appears to facilitate aggressive behavior. Studies that involved injecting carbachol, a cholinomimetic, into feline amygdalas produced aggressive behavior (Grossman, 1963). Similarly, when carbachol is injected into the lateral hypothalamus of rats, the rats will kill mice, whereas atropine blocks such attacks (Smith, King, & Hoebel, 1970). Even in rats that are not mouse killers, injections of carbachol will induce them to kill mice with the same stereotyped mannerisms as those of experienced killers (Bandler, 1970; Siegel & Pott, 1988; Smith et al., 1970). This was observed even in rats that had never manifested the predatory behavior involved in such killing (Bandler, 1970; Siegel & Pott, 1988; Smith et al., 1970).

GABA, an amino acid neurotransmitter, is the most prevalent inhibitory neurotransmitter and is negatively correlated with aggression in animals. Decreased GABA levels in mouse-killing rats have been contrasted with higher GABA levels in mice that do not kill (Mack, Simler, & Mandel, 1975). Further, injections of GABA in mouse-killing rats have produced short-term inhibition of killing of rats (Paglisi-Allergra & Mandel, 1980). Both isolation-induced fighting and pain-induced fighting by rodents are reduced when the GABAergic system is activated (Mack et al., 1975; Paglisi-Allergra & Mandel, 1980). The class of drugs known as benzodiazepines is known to have anxiolytic, sedative, and anticonvulsant effects, and to have particular importance in relation to GABA (Costa, Guidotti, & Mao, 1976). Benzodiazepines bind to GABA and increase the binding of GABA molecules to the receptor and increase GABA's inhibitory effects (Costa et al., 1976; Randall, Schallek, Heise, Keith, & Bagdon, 1960). Benzodiazepine binding sites are particularly dense in the amygdala, a structure known to play a role in emotion. Use of benzodiazepines in animals has produced decreases in affective aggressive behavior in rats and other animals (Christmas & Maxwell, 1970; Eichelman, 1990; Randall et al., 1960; Valzelli, 1973).

The serotonergic system has also been shown to inhibit aggressive behavior in rodents (Eichelman, 1988). Both predatory and affective aggression can be increased in rats by restricting foods with tryptophan, a precursor of serotonin, or by inhibiting the activity of tryptophan hydroxylase, the synthesizing enzyme for serotonin (Gibbons, Barr, Bridger, & Leibowitz, 1979; Kantak, Hegstrand, Whitman, & Eichelman, 1980). Subcutaneous injections of a tryptophan hydroxylase inhibitor (*p*-chlorophenylalanine) in cats produced an increase in the occurrence of spontaneous predatory and affective behavior (DiChiara, Bamba, & Spano, 1971; Sheard & Davis, 1976). Lesions made in the serotonin-containing raphe nuclei will decrease serotonin levels and result in affective aggression (DiChiara et al., 1971; Kantak, Hegstrand, & Eichelman, 1981). This is consistent with studies whereby, on stimulation of the raphe nuclei, predatory aggression was inhibited (Eichelman, 1988, 1990). In contrast, rats made more aggressive through septal lesions or neurotoxin treatment will not show evidence of the behavior when 5HT (synthesized from the amino acid tryptophan) is increased (Eichelman, 1995; Jacobs & Cohen, 1976). These various studies demonstrate that a reduction of serotonin-induced neural excitability will result in expression of the predatory and affective response in lower animals.

In contrast to the inhibitory mechanisms of GABA, norepinephrine (a monoamine neurotransmitter) has been shown to increase affective aggression. There are conflicting

data, however, as to whether norepinephrine functions as a neuromodulator or as a primary neurotransmitter responsible for elicitation of affective attack behavior. Discharges of norepinephrine from the brainstem of cats have led to increasing incidents of affective attack (Eichelman, 1995; Reis, 1974). Tricyclic antidepressants (which block reuptake and thus increase the level of norepinephrine at the synapse) and monoamine oxidase inhibitors (which inhibit one of the enzymes that breaks down norepinephrine) both enhance aggression in rodents (Eichelman & Barchas, 1975; Eichelman, Thoa, & Perez-Cruet, 1973; Stolk, Conner, & Barchas, 1971). Lithium, which has been shown to influence norepinephrine, decreases aggressive behavior in rodents and fish, presumably because it reduces the availability of norepinephrine (Weischer, 1969). Other studies that employed intraventricular injections to decrease norepinephrine levels, weaken adrenergic nerve terminals, and disrupt uptake failed to disrupt the hypothalamically elicited affective attack behavior, thus supporting the hypothesis that norepinephrine may function as a neuromodulator (Eichelman, Elliott, & Barchas, 1981). Perhaps most important is the observation that while norepinephrine increases incidents of affective aggression, it has the opposite effect on incidents of predatory aggression, suppressing such displays (Eichelman, 1988, 1990).

The dopaminergic system has also been studied to discern its role in predatory and affective attack. In rodents and cats, stimulation of the central dopamine system intensifies affective aggression, yet the route of action is unclear (Eichelman et al., 1981; Siegel & Pott, 1988). Use of dopamine agonists has been shown to reduce the threshold for directed attack and hissing (Siegel & Pott, 1988). Similarly, dopaminergic antagonists have been shown to inhibit affective attack (Maeda, 1976). There is less evidence to support the role of dopamine in regulating predatory attack (Siegel & Pott, 1988). There is some indication, however, that dopamine plays a role in completing the attack response (Marshall, 1979). These findings are supported by lesion studies that demonstrate the role dopamine plays in sensory elements related to the attack response.

2. Clinical research on aggression and violence

Are there human equivalents to affective and predatory attack? Animal models of aggression, although not directly applicable, have helped researchers understand aggression and violence in humans. This portion of the review will be divided into four areas: (1) behavioral classifications of modes of aggression, (2) bridges from ethological models to human models, (3) neuroanatomical substrates of aggression, and (4) neurochemical correlates of aggression.

2.1. Behavioral classifications of aggression

Elegant and systematic research paradigms have produced an abundance of animal models of aggression. However, clinical literature describing human aggression and violence has often been speculative, attempting to draw theoretical parallels from the ethological literature to human behavior. Unfortunately, human studies have not adhered to the same scientific

rigor, and no standardized lexis has emerged by which researchers across disciplines can communicate about observed behavior. Part of the problem has been the tendency to intermingle value-laden notions of "good" and "evil" with the study of human violence, or to approach the study as a nature (inherited) versus nurture (acquired trauma or poor parenting) polarization. A vast literature addresses aggressive behavior from diverse theoretical perspectives, including psychoanalytic, ethological, sociological, and learning theory, as well as from a clinical database that includes subgroups of patients with psychiatric disorders. The following sections will address some of these aforementioned early notions of human aggression leading to modern-day classification systems.

In 1920, Freud (1958) postulated that all humans possess an aggressive drive from birth, which, together with the sexual drive, contributes to personality development. Freud also theorized that these drives are expressed through a variety of behaviors. The Austrian ethologist Lorenz (1966) suggested that aggression is innate, an inherited fighting instinct, as significant in humans as it is in other animals. He contended that the suppression of aggressive instincts common among human societies allows these instincts the chance to build up, occasionally to the point where they are released during instances of explosive violence. In the late 1930s, Dollard, Miller, Mowrer, Sears, and Sears argued that any sort of frustration inevitably leads to an aggressive response.

More recently, Bandura (1966) performed studies with children indicating that aggression is a learned behavior. He demonstrated that, by watching another person act aggressively and obtain desirable rewards or by learning through personal experience that such behavior yields rewards, children can be taught aggression.

The theoretical and experimental literature that attempts to define and delineate aggressive behavior involves primarily psychiatric populations. Kingsbury, Lambert, and Hendrickse (1997) recently organized aggression into instrumental and hostile categories, a bimodal classification based on a social–psychological perspective for application in clinical settings.

Instrumental aggression is promoted through the principles of operant conditioning. That is to say, the behavior (aggression) is learned and learning takes place as the individual acts on the environment. Whereas classical conditioning involves innate reflexes, operant conditioning requires voluntary behavior. Described as a means to an end, instrumental aggression is driven by the expectation of desired reinforcement following the behavior. Therefore, from this perspective, instrumental aggression requires an environment that will reinforce the behavior. As with predatory aggression, to which instrumental aggression has been linked, the behavior is purposeful and goal directed (Aronson, 1992; Kingsbury et al., 1997).

Hostile aggression denotes the specific intention of harming another individual (Aronson, 1992; Kingsbury et al., 1997). Environmental and interpersonal cues that lead to anger, fear, or frustration may elicit an aggressive response. Unlike instrumental aggression, whereby harm is inflicted on another person in the pursuit of some type of reward, hostile aggression is an end unto itself. Similar to descriptions of affective aggression, hostile aggression involves an associated state of heightened arousal that is facilitated by intermediary affective states such as anger, rage, or frustration. These intermediary affective states (anger being the most reliable elicitor) seek discharge through the vehicle of aggression. There are some individual

differences or conditions that must be met for the expression of hostile aggression. For example, people have different thresholds for enacting aggression, and some people are particularly sensitive to unique environmental stimuli that will produce the behavior.

Berkowitz (1994) contended that all animals learn the most effective response to an aversive occurrence (one where the expected reward is denied), whether it is attack or flight. He made the distinction between *reactive* and *instrumental* aggression. Reactive aggression comes in response to frustration, similar to the frustration–aggression model. Like affective attack, reactive aggression is typically in response to a provocation or perceived threat. Instrumental aggression, as previously discussed, is goal oriented.

Barratt, Kent, Bryant, and Felthous (1991) classified aggression into three broad categories: (1) premeditated or learned aggression, (2) medically related aggression, and (3) impulsive aggression. In this classification, premeditated aggression is a learned behavior influenced by the social context and culture of the individual. Medically related aggression involves some type of medical disorder, such as a psychopathology. Broad in its scope, medically related aggression involves neurological abnormalities caused by traumatic brain injury, psychosis, and panic attacks. Impulsive aggression is typically defined as a spontaneous "hair-trigger" display of aggression that is neither planned nor the result of a medical disorder. Remorse often follows the act but does not necessarily reduce future occurrences given the individual's poor behavioral controls. Terms, such as *neuropsychotic aggression* or *episodic dyscontrol* (Monroe, 1975), have also been used to describe processes similar to impulsive aggression.

In the child aggression literature, two consistent subtypes have also emerged, that of a controlled–proactive–instrumental–predatory category and that of an impulsive–reactive–hostile–affective category (Cornell et al., 1996). They are qualitatively different from each other in neurobiology and psychology. In an effort to differentiate children according to the predatory–affective dichotomy, Vitiello, Behar, Hunt, Stoff, and Ricciuti (1990) studied 73 children and adolescents with a history of aggressive behavior. These researchers used a 10-item questionnaire in which 5 items measured affective rage and 5 items measured predatory attack. A bimodal distribution occurred, indicating one group with a mixed predatory–affective pattern of behavior and a second group with a predominately affective pattern of behavior. Youth who exhibited primarily affective violence were more likely to receive lithium or neuroleptics, to be diagnosed with schizophrenia, and to have a lower IQ. Among the predatory group, a history of drug abuse was more common.

Crick and Dodge (1996) examined 624 children ages 9–12 and classified them as proactive–aggressive, reactive–aggressive, nonaggressive, or a mixed type. Making a theoretical distinction between "reactive" and "proactive," Crick and Dodge described reactive as "an angry, defensive response to frustration or provocation" and proactive as "a deliberate behavior that is controlled by external reinforcements ... a means for obtaining a desired goal" (p. 993). They found that children with a history of reactive aggression interpreted their peer's behavior as intentionally harmful to them, and that their violent response served as retaliation. Children with proactive aggression possessed greater confidence in their ability to perform violent acts than did those evidencing primarily reactive aggression. These authors considered their definitions somewhat incomplete, yet they

maintained that there was a significant overlap between their term *reactive aggression* and the common term *affective aggression* in the adult research. Likewise, *proactive aggression* seemed consistent with behavior commonly termed predatory in the adult research.

The most comprehensive approach to creating a nosology was conducted by Eichelman and Hartwig (1990), who developed the Carolina Nosology of Destructive Behavior (CNDB). The authors proposed a standard classification system to delineate potential treatment-responsive groups of individuals who display destructive behavior. It allows researchers and clinicians a structure from which to assess, reassess, or use as a comparison between patients who exhibit destructive behavior. The most comprehensive nosology of its kind, the CNDB provides a multiaxial, empirically based system for categorizing patients who clinically manifest destructive behavior. This system involves two major components. The first establishes a database of destructive behaviors by defining and codifying such behaviors. It is comprised of 10 elements: (1) event characteristics, (2) type of destructive behavior, (3) stimulus characteristics, (4) degree of premeditation, (5) focus of behavior, (6) target for the behavior, (7) weapons, (8) sexual correlate, (9) severity, and (10) frequency. The second component makes use of four axes: (1) medical diagnosis (Axis A), (2) psychological correlates (Axis B), (3) biological correlates (Axis C), and (4) moral/cultural correlates (Axis D). The multiaxial system is intended to provide a standardized classification system by which clinicians and researchers can communicate. Using this system, the authors believe a substantial clinical database will emerge that can then be used across patient populations by clinicians and researchers.

This multiplicity of labeling similar behavior creates at least three problems. First, the labels are used interchangeably, although subtle differences have evolved over time. Second, the terms can lead to confusion. For example, all modes of violence are instrumental in the sense that they have a goal. Affective aggression has the goal of threat removal, so in a sense it is instrumental. Third, implying a connection between labels (e.g., instrumental) and predatory aggression confuses the explicit etiology of the terms. Instrumental aggression, largely defined through models of social learning, ignores the well-established biological basis of the aggressive behavior.

2.2. Bridges from ethological models to human models

Attempts to apply animal models of violent and aggressive behavior to that of human behavior have be fraught with methodological problems. First, affective and predatory aggression seen in feline research is adaptive. That is to say, both forms of aggression are part of a larger survival mechanism seen in normally functioning cats. Much of the research on human aggression typically focuses on pathological aggression, which is maladaptive and is restricted to a subgroup of the human population.

Second, the neuroanatomical structures and functions are far more complex in the human brain. Similar anatomical structures have been identified in both humans and animals that provide the same function. For example, all brains are constructed of neurons, and the neural structures that compose the brains of one species can usually be found in the brains of related species. Over 80% of the human cerebral cortex is a neocortex of relatively recent evolu-

tionary origin that is responsible for the regulation of a broad range of higher cognitive, emotional, and motor functions. As primates evolved, the innervations between sensory and motor systems became more multifaceted. The more primitive structures were not replaced but rather enhanced as new levels added more complexity and control over earlier structures.

Therefore, when considering animal models, we must keep in mind the role of these additional structures, particularly the presence of the frontal neocortex, which modulates the expression and suppression of aggressive behavior. Consequently, when examining studies that address the effects of damage to the hypothalamus in humans, we cannot simply conclude that resultant behavior is a result of the damage. The changes in behavior seen after ablation or damage to an area may not be due to the loss of those particular neural structures but instead may be due to an alteration in functioning of distal brain regions that no longer receive the accustomed innervation from the ablated region.

2.3. Anatomical basis of aggression in humans

Animal studies cannot be replicated in human subjects due to legal and ethical constraints. Therefore, investigators have turned to disease processes, head injuries, and mental illnesses to study the effects of damage to human anatomical structures. Prior to published reports of the role of the hypothalamus in felines, human case studies hinted at possible links between subcortical structures in the brain and the production of aggression. Papez (1937) proposed that the limbic system served a function in emotional expression. After their findings were published, reports of lesions in the limbic system and resulting psychiatric symptoms began to surface. The research on aggression as it relates to the limbic system typically involves disease processes such as rabies, tumors, and cysts.

One of the earliest connections between aggressive behavior and diffuse damage to the limbic system in humans came from patients who contracted rabies (Viets, 1926)). Studies of these patients revealed that Negri bodies in the hippocampus had a role in aggressive behavior. Tumors located in the ventromedial hypothalamus of humans have resulted in unpredictable and unprovoked attacks and are typically directed at those in proximity to the affected individual rather than at a logically identifiable target (Viets, 1926). The location of the tumor and the resulting necrosis to surrounding tissue is related to brain areas in cats that, when stimulated, led to affective displays of aggression (Viets, 1926).

Leslie (1940) reported the case of a man who, in 1922, complained of chronic, severe splitting headaches. Over the course of 15 years, a slow growing cyst of the cavum vergae caused defects in the septum pellucidum, producing definite personality changes. The patient was described as irritable, irresponsible, and prone to displays of unmodulated temper tantrums.

Reeves and Plum (1969) detailed the case of a 20-year-old Puerto Rican woman who developed hyperphagia and obesity, as well as overt aggression. At times, she would hit, scratch, and attempt to bite the medical examiner involved in her care. After her death, an autopsy revealed that a small neoplasm had destroyed her ventromedial hypothalamus. This is the area that, when destroyed in felines, produces affective attack behavior. This woman's behavior appeared consistent with the manner of affective attack.

Alpers (1937) studied tumors that grew on the hypothalamus and the resultant behavioral presentation, which included psychosis. He described the case of an attorney who had a teratoma that damaged the hypothalamus. As a result, the attorney displayed aggressive behavior, including rage when under the influence of alcohol and fights with guests in his home.

Malamud (1967) reviewed 18 autopsied cases of patients with intracranial tumors located in various regions of the limbic system. In seven of the cases, aggressive behavior was noted. His findings supported the role of the limbic system in emotional expression. There was less conclusive evidence that limbic lobe lesions produced aggressive behavior. All the patients had seizures, but most did not manifest aggressive behavior or schizophrenia; mood disorders were the primary diagnosis.

Narabayashi, Nagao, Saito, Yoshida, and Nagahata (1963) investigated the value of amygdalotomy in three groups of patients. Initially, the researchers targeted patients with temporal lobe epilepsy who evidenced marked behavioral disturbances, including hyperexcitability, assaultive behavior, and violent aggressiveness. Later, Narabayashi and his colleagues extended their study to those with seizures other than temporal lobe epilepsy who nonetheless displayed aggressive behavior. Finally, they studied a group of patients considered feeble-minded or with less than average intelligence. The total sample size was 60, with patients ranging in age from 5 to 35 years. Eighty-five percent of the sample showed marked improvement after the stereotaxic amygdalotomy, as measured by a marked reduction in emotional excitability and normalization of social behavior and adaptation. Subsequent studies, although not as large or as comprehensive, for the most part have supported the findings of the Narabayashi et al. study (Hitchcock & Cairns, 1973; Memple, 1971). Not all studies of amygdalotomy, however, found reductions in aggressive behavior (Kim & Umbach, 1973; Nadornick & Sramka, 1973).

Sano, Mayanagi, Sekino, Ogashiwa, and Ishyima (1970) reported that 27% of their population of 44 patients with surgically produced lesions in the posterior hypothalamus showed "excellent" improvement, as measured by decreases in violent, aggressive, and restless behavior. Twenty-eight patients continued to show occasional irritability, indicating a "good" response to treatment. Two patients showed no changes in behavior and two died from postoperative complications.

2.4. Neurotransmitters

Research on the role of neurotransmitters in animal aggression has prompted researchers to explore possible links to human neurophysiology. This has led not only to an understanding of the role of neurotransmitter systems in the regulation of human aggression but also to practical suggestions for pharmacological interventions. Extrapolating from the neurotransmitter functions in lower animals to those of human neurotransmitter systems however should be done cautiously. Manipulating one neurochemical in a complex system and observing the behavioral changes overlooks the possible effects on the system as a whole. Information gained from animal research has led to successful treatments of assaultive patients involving the use of beta-blockers, serotonin, and anticonvulsants. The bulk of the data suggests that the

GABAergic and serotonergic systems function as inhibitors to affective and predatory aggression, whereas the noradrenergic and dopaminergic systems facilitate affective aggression (Eichelman, 1987, 1988).

Animal studies revealed that the GABAergic system can inhibit aggression and that activation of the benzodiazepine receptor system can further facilitate the functionality of the GABA system (Costa et al., 1976). Uses of benzodiazepines in humans have also been shown effective in reducing aggressive behavior (Boyle & Tobin, 1961; Lion, 1979; Monroe, 1975). This class of drugs, prescribed for their tranquilizing, anti-anxiety, sedative, and muscle-relaxing effects, has shown promising results in hostile outpatients (Lion, 1979), aggressive psychotic patients (Boyle & Tobin, 1961), and patients with episodic dyscontrol (Brown, Ballenger, Minichiello, & Goodwin, 1979; Brown, Goodwin, Ballenger, Goyer, & Major, 1979).

Research on the role of the serotonergic system in humans has produced evidence for a serotonergic influence on aggressive behavior (Brown, Ballenger, et al., 1979; Brown, Goodwin, et al., 1979). In general, when serotonin is elevated in the system, decreases in aggression have been observed. Some studies have not been able to replicate these findings (Volavka et al., 1990; Whitehead & Clark, 1970); however, most data support the finding that lower levels of serotonergic activity correlate with higher rates of aggression across the life span. This finding has been replicated in populations of impulsive offenders, adults, and children (Brown, Ballenger, et al., 1979; Brown, Goodwin, et al., 1979; Kruesi et al., 1990; Linnoila et al., 1983).

Lithium is believed to impose its effects through the serotonergic system by increasing the uptake of tryptophan, a serotonin precursor, into the brain and subsequently increasing the synthesis and release of serotonin. It has been used in a variety of aggression studies. A study of prison inmates found that lithium, a natural salt, decreased aggressive episodes (Sheard, 1975; Sheard, Marini, Bridges, & Wagner, 1976). Lithium produced similar results in samples of patients with mental retardation (Sheard et al., 1976) and in those with attention-deficit disorder and conduct disorder (Siassi, 1982). Morand, Young, and Ervin (1983) found that tryptophan attenuated aggressive episodes in a population of schizophrenic patients with poor impulse control and high lifetime aggression. Serotonergic drugs in general show promise in the treatment of aggressive behavior.

Similar to research on animals, research on the noradrenergic system in humans has shown increases in affective aggression when norepinephrine is elevated and decreases when it is reduced. Drugs that increase norepinephrine levels (e.g., tricyclic antidepressants and monoamine oxidase inhibitors) have been shown to exacerbate aggressive behavior in a population of patients with an agitated depression (Rampling, 1978). Conversely, drugs that reduce norepinephrine levels have been shown to inhibit aggression (Eichelman, 1986). Beta-adrenergic antagonists has been tried with groups of patients with schizophrenia, organically impaired adults, and children, with resultant positive effects in reducing aggressive behavior (Eichelman, 1986).

The research is mixed concerning the effects of dopamine on human aggression. Dopamine antagonists, such as haloperidol, have long been used to treat aggressive behavior in psychotic patients. They have also been shown to decrease fighting in psychiatric patients

(Morand et al., 1983), patients with dementia (Sugarman, Williams, & Adlerstein, 1964), and individuals with borderline personality disorder (Brinkley, Beitman, & Friedel, 1979). Their use in children has also been shown to decrease aggressive behavior in conduct-disordered children (Campbell et al., 1984), aggressive delinquent children (Campbell et al., 1984), and retarded children (Dale, 1980). Although some studies have not demonstrated improvement with dopamine antagonists (Yudofsky, Stevens, Silver, & Barsa, 1984), most data show decreased aggression following decreases in dopamine levels.

Multiple pharmacological agents that were developed as a result of the research on neurotransmitters have been used to target aggressive behavior. In addition to these agents, phenytoin (Dilantin), an anticonvulsant, has shown considerable promise over the years, although its effectiveness has not been unequivocal. Its usefulness in lessening aggression was discovered through anecdotal observations of epileptic patients whose sense of well-being and social behavior both improved while on the drug (Barratt, Stanford, Felthous, & Kent, 1997). It is speculated that impulse control problems have, at root, similar anatomical and chemical bases as seizure disorders. The variability seen in the effectiveness of phenytoin may in part be related to experimental issues, such as (1) lack of common objective criteria, (2) failure to establish inclusion and exclusion criteria, and (3) lack of an agreed-upon categorization of aggressive behavior across studies. Barratt et al. (1997) confronted these issues in their carefully controlled study of the effects of phenytoin on impulsive and premeditated aggression in a sample of prison inmates. They hypothesized and confirmed that phenytoin significantly reduced both the intensity and frequency of impulsive aggression, but was not effective with premeditated aggression.

3. Forensic applications

The topic of aggression and violence has become a burgeoning area of interest in forensic research and practice. Forensic psychology and psychiatry attempt to understand and explain aggressive behavior in a psychobiological and legal framework. The study of forensic samples affords a potential means for differentiating between constellations of aggression by providing a pool of individuals known to have engaged in violent acts. This section will address three areas of research to which the forensic field contributes (1) behavioral observations of individuals known to have engaged in violent acts, (2) physiological substrates of these offenders, and (3) data from special populations of criminals that exhibit distinct expressions of violent behavior.

3.1. Behavioral observations in forensic samples

Descriptions of aggressive behavior in the forensic research have been adapted from clinical classification schemes and follow a bimodal classification. The first category falls under behaviors similarly described as "impulsive," "defensive," "hot blooded," "reactive," or "affective." In general, these designations reflect acts of aggression in response to a perceived threat, and typically are emotionally charged and relatively uncontrolled. The

second category uses terms such as "predatory," "proactive," "attack," "instrumental," and "cold blooded." These terms describe acts of violence that are more purposeful and controlled, and that lack an emotional display. Whereas the first category reflects an attempt at threat reduction, the second category reflects aggression in the service of premeditated goals. The first category has been more widely researched, perhaps because it is inherently easier to observe and measure. In the past, the second category has eluded researchers, perhaps because individuals engaging in more extreme forms of violence are less likely to present themselves to mental health centers for treatment or research purposes. Recently, more has been learned about this second type of aggression.

A group of researchers has noted differences between aggression committed for goal-directed purposes and aggression in response to a provocation or perceived threat (Baron, 1977; Dodge, 1991; Parke & Slaby, 1983; Zillman, 1979). Berkowitz (1993) compiled extensive data that also support this division between two classes of aggression. He used the term *instrumental* to describe goal-directed violence and the terms *reactive* or *emotional* to describe violence that is a response to frustration. In a study of juvenile offenders, Pulkinnen (1987) found that "offensive" aggression was a more stable trait and better at predicting future criminality than was "defensive" aggression. Cornell, Benedek, and Benedek (1987) reported that juvenile offenders who committed homicides while committing other crimes (e.g., robbery) could be reliably discriminated from juvenile offenders convicted of conflict-related violence (e.g., murder committed in an interpersonal context). *Episodic dyscontrol* is also a term used by various investigators to describe violence that indicates a lack of impulse control, is possibly paranoid and instigated in an altered perceptual state, and is usually elicited in response to stimuli provoking fear, anger, or rage (Monroe, 1978). Monroe believed these behaviors are seen in those with "explosive personality." The fourth edition of the *Diagnostic and Statistical Manual (DSM) of Mental Disorders* (1994) currently categorizes this type of behavior as "intermittent explosive disorder." Monroe (1978) believed this violence erupts from an imbalance of "urge control" mechanisms. The normal urge control mechanism is either overwhelmed by an extreme drive or it is underdeveloped and is overwhelmed by a relatively normal urge. This theory assumes the presence of disproportionate neuronal discharges from the limbic structures to subcortical regions like the hypothalamus and brainstem.

Meloy (1988, 1992, 1997) has compiled a body of research supporting the distinctions between affective and predatory violence, the terms that he thinks best reflect actual behavior patterns. He describes affective violence as the "garden-variety" violence seen in our society. It is preceded by intense autonomic arousal and a subjective experience of conscious emotion, usually anger or fear. It is typically a response to a provocation and is an immediate reaction. There is always an internal or external perceived threat, and the goal of the violence is to reduce the threat and return to biological homeostasis. Affective aggression is typically time limited and may be preceded by public rituals and instinctual behaviors (e.g., vocalizations) to reduce the threat. A heightened and "diffuse" awareness is present.

Meloy (2000) makes an important distinction between predatory and affective violence. In the later, sympathetic signs such as increased heart rate are evident, but in predatory violence, they are usually absent. When these signs are absent, response patterns are difficult to detect,

which could be used to anticipate the onset of predatory aggression. In predatory violence, there is minimal emotion. Any emotions that are present tend to be positive because they lead to exhilaration and increased self-esteem by providing pleasure and self-confidence in aggressive abilities. In both predatory and affective violence, the violence is purposeful. However, in predatory aggression, the perpetrator chooses the target, plans the form of attack, and tailors the strength of the aggressive response. Meloy (1988) suggests that predatory behavior "may be used to gratify certain vengeful or retributive fantasies. It may be subjectively experienced as a necessary behavior that would be clinically assessed as compulsive" (p. 215). For example, the goals of predatory violence may include sexual gratification of sadistic fantasies, securing of money or turf, or relief from compulsive drives. Rituals are often involved and can include certain items of clothing, weapons or masks, and nationalistic or religious symbols. These objects may help to fantasize control over the victim. In predatory aggression, the individual is able to maintain heightened and focused awareness and can often filter out other sensory information (e.g., as a cat does when focused on its prey) (Meloy, 1988).

The forensic field has increasingly relied on a bimodal classification of violence. Although there is research support for this classification scheme, these modes of violence are not activated at the same time but do coexist as a potential in any one subject (Cornell et al., 1996). Berkowitz (1993) noted that criminals often display both reactive and instrumental aggression at different times, but he hypothesized that there may be more than two categories of aggressive behavior in criminals that have yet to be differentiated.

3.2. Physiological substrates in forensic samples

There is a venerable literature indicating a physiological basis to criminality. Certain forms of criminality are associated with lower levels of physiological reactivity, and hyporeactivity is related to sensation seeking (Schalling, Asberg, Edman, & Oreland, 1987). This hypothesis asserts that chronic underarousal is aversive; therefore, increased stimulation is sought through risk taking and criminality.

Forensic investigators have attempted to determine the biological contributions to human aggression, criminality, and antisocial behavior. Studies of twins and criminality show a higher concordance rate among monozygotic pairs than among dizygotic pairs, suggesting a genetic contribution (Coccaro, Bergeman, & McClearn, 1993; Mednick, Gabrielli, & Hutchings, 1984, 1996; Schalling et al., 1987). Prior to 1963, no published studies showed a concordance rate below 60% with monozygotic twins (Rosenthal, 1971). However, subsequent studies relying on a more representative and systematic sampling produced concordance rates of about 33% (Christiansen, 1968). Siblings have genetic and environmental influences in common; thus, psychosocial and biological influences may both lead to aggressive behavior.

Similarly, higher rates of criminal behavior have been observed among individuals born to criminal parents (Mednick et al., 1984). Researchers found that both genetic and environmental influences contribute to criminality (Mednick et al., 1984). In their study of criminals who had been adopted as children, biological fathers were more likely to evidence criminal

behavior than adoptive fathers. The highest rates of criminality occurred when both the biological and adoptive fathers were criminals.

Cleckley (1941) described psychopaths as having a "peculiar form of semantic dementia" (p. 378), in that they do not understand the emotional significance of words. He believed that this characteristic suggested limbic dysfunction. Recent research seems to support the notion that psychopaths are fearless and that they have low levels of anxiety (Meloy, 1995), which suggests various biological substrates.

Biochemical, neuropsychological, and neural imaging studies of murderers, psychopaths, and habitual criminals have focused on frontal lobe and left hemisphere dysfunction, in addition to left frontal–temporal–limbic damage (Raine, 1993). Although not definitive, evidence suggests deficits in the function, but not the structure, of the orbital–frontal portion of the prefrontal cortex. Empirical findings indicate that psychopaths have moderate-to-pervasive formal thought disorders (Gacono & Meloy, 1994) that may relate to executive functioning, such as long-term planning. This function likely operates from the prefrontal cortex and may be underdeveloped in psychopaths (Raine, 1993). Barratt et al. (1997) found that impulsive violent offenders differed from controls on neuropsychological measures of information-processing deficits and event-related potential. Likewise, impulsive violent offenders have been shown to have lower serotonin (Coccaro, Kavoussi, Sheline, & Lish, 1996; Linnoila et al., 1983; Virkkunen & Linnoila, 1993) and monoamine oxidase A deficiency (Brunner, Nelen, Breakefield, & Ropers, 1993) compared to nonimpulsive offenders.

Raine, Venables, and Williams (1995) conducted a prospective study relying on autonomic and cortical measures of persons at age 15 to predict criminality at age 24. They found that criminals had lower skin conductance activity, lower resting heart rate, and slower frequency electroencephalographic (EEG) activity than noncriminals. In addition, these discrepancies were not mediated by demographic, academic, or social influences.

The psychophysiology of the psychopath has been studied by several researchers. In a review of research on skin conductance, Hare and Cox (1978) found that in aversive events, as well as in conditioning and quasi-conditioning learning paradigms, psychopaths had lower skin conductance responses and longer recovery phases after aversive stimuli. Hare and Cox concluded that these findings support the notion of the peripheral autonomic hyporeactivity hypothesis in relation to psychopaths. In recent years, debates have ensued regarding these findings. Yet, Raine's (1993) review of the literature, 15 years after Hare's review, yielded sufficient evidence to maintain the underarousal theory of autonomic responsivity in habitual criminals. More recently, Raine et al. (1998) expanded the hyporeactivity concept and suggested that chronic cortical underarousal in most criminals is significant, particularly when social risk factors, such as lower socioeconomic status, are absent. In other research, Brennan et al. (1997) and Raine et al. (1995) found lower skin conductance, slow-wave (theta) EEG activity, and lower resting heart rate to be robust predictors of habitual criminality. When these signs are absent, delinquent adolescents are less likely to adopt adult criminality.

Little information is available regarding sensory systems involved in aggressive behavior. In one study of prisoners serving time for "nonaggressive" and "aggressive" crimes, Kinzel (1970) observed a "body buffer zone." He described it as the psychological space

surrounding a person in which encroachment is felt as intrusive. This body buffer zone was larger in prisoners convicted of aggressive crimes.

Gottman et al. (1995) focused on domestic violence and identified a physiological marker that discriminates among violent men on several realms. They studied physiological responses during marital conflict in 61 battering couples. As an index of physiological response, the male batterer's heart rate reactivity was measured as the change from an eyes-closed baseline up to the first 5 minutes of the marital conflict interaction. Six physiological dependent measures were obtained from three kinds of recording devices placed on the surface of the participant's skin and from a fourth device attached to the participant's chair: cardiac interbeat interval, pulse transmission time to the finger, finger pulse amplitude and skin conductance level, and general somatic activity level. Their results indicated two significant groups of batterers, those who initially lowered their heart rates during marital conflict (type 1) and those who raised their heart rates (type 2). Differences between the two groups were also found in the amount of peripheral vasoconstriction, with type 2 men experiencing increased rates. Type 2 men not only initially raised their heart rates, but also continued to become more aggressive as interactions unfolded. While type 1 men lowered their heart rates, they were more belligerent, contemptuous, and angry than were type 2 men. Type 1 men were not more violent toward their partners but were more violent outside the marriage toward strangers, friends, bosses, and coworkers. As children, type 1 men were more likely to have witnessed physical violence between their parents. On the MCMI-II, they had higher rates of antisocial and aggressive–sadistic characteristics and were more likely to be drug dependent.

Gottman et al.'s (1995) research appears to correlate with the research suggesting a link among lower reactivity, psychopathy, and habitual criminality. The authors proposed that type 1 men may initially strike out with high levels of emotional aggression at the beginning of marital conflict in an "orienting response." Thus, the lowering of heart rate may increase focused attention and aid in the task of manipulating the wife's response, preparation more akin to predatory rather than affective violence.

Raine et al. (1998) also examined neurophysiological and neuroanatomical mechanisms that correlate with predatory and affective aggression in humans. Raine's research team hypothesized that affective violent offenders would have decreased prefrontal functioning, higher subcortical functioning, and decreased prefrontal-to-subcortical functioning. Conversely, the researchers hypothesized that predatory violent offenders would show moderately normal brain functioning relative to a comparison group. Positron emission tomography assessed glucose metabolism in left and right hemisphere prefrontal (lateral and medial) and subcortical (amygdala, hippocampus, midbrain, and thalamus) areas. These four structures are believed to play critical roles in aggression.

Affective murderers had lower prefrontal activity than the comparison group. In contrast, the predatory group had prefrontal functioning equivalent to the comparison group. Contributors to the forensic literature have debated whether psychopathic criminals have lower prefrontal dysfunction measured by neuropsychological tests (Hare, 1984; Gorenstein, 1982; Lapierre, Braun, & Hodgins, 1995). In most cases, psychopaths evidence more predatory aggression and affective aggression (Meloy 1988; Serin, 1991; Williamson, Hare, & Wong,

1987). Therefore, these findings of moderately normal prefrontal metabolism in the predatory group suggest that in a general population of psychopaths there would be abnormal prefrontal dysfunction.

Both groups had higher subcortical activity in the right hemisphere, with predatory murderers having only slightly higher right subcortical activation than the comparison group, and the affective offenders having significantly higher right subcortical activation. Activation of this area has been implicated in initiation of negative emotion (Davidson & Fox, 1989). The authors surmise that predatory and affective murderers vary in regulatory control over aggressive impulses, with predatory offenders having more ability to modulate aggressive behavior. Conversely, affective offenders lack prefrontal control, which results in unregulated aggression.

3.3. Subgroups in the forensic samples

Subgroups of aggressive offenders, such as psychopaths, stalkers, domestic batterers, and bombers, have been studied for patterns and modes of aggressive behavior. As described previously, physiological substrates (e.g., EEG, heart rate, skin conductance, neurotransmitters) frequently relate to behavioral patterns in these groups.

3.3.1. Psychopathy

Psychopathy has often been confused with, or used interchangeably for, the DSM categorization of antisocial personality disorder. There is, however, well-founded research that clearly distinguishes the two behaviors in both biogenic and psychogenic origins. Compared with other criminals, psychopaths represent a disproportionately higher rate of criminality. In comparison to the acts of other offenders, their crimes are more aggressive and violent (Hare & McPherson, 1984), and are typically callous and cold. Yet, psychopaths are also rageful at times. A disinhibitory response to even slight provocations appears evident in both their calculated and impulsive violence (Elliott, 1992). Consistent research indicates that the Psychopathy Checklist-Revised (PCL-R; Hart, Hare, & Forth, 1994) distinguishes a subgroup of psychopathic criminals who are more resistant to treatment, prone to recidivism, and more maladjusted than nonpsychopathic offenders. Hart, Kropp, and Hare (1988) found that, after release from incarceration, offenders ranking in the top third of the PCL were nearly three times more likely to violate conditions of parole and nearly four times more likely to commit another violent crime. Hare's (1991) construction of the PCL-R is a 20-item structured interview that measures the severity of psychopathy along two factors. Factor 1, *aggressive narcissism*, a term coined by Meloy (1988), assesses a callous and remorseless disregard for the feelings and rights of others. It encompasses behavior, such as lack of remorse or guilt, shallow affect, and callousness or lack of empathy. Factor 2, "chronically unstable and antisocial lifestyle," reflects more observable behavior, such as poor behavioral control, impulsivity, irresponsibility, and revocation of conditional release. A need for stimulation and proneness to boredom are robust findings in the psychopathy research (Hare, 1970; Millon, Simonsen, Dirket-Smith, & Davis, 1998).

Hare's descriptions depict psychopathy as predatory in nature. Shallow affect, low emotional reactivity, proneness to boredom, and poor behavioral control seem strongly related to predatory aggression. Studies confirm that psychopaths tend to engage in predatory rather than affective violence, while nonpsychopathic violent criminals are involved in more affective violence (Cornell et al., 1996; Meloy 1997, 1988; Serin, 1991; Williamson et al., 1987). Serin (1991) for example found that violent psychopaths were qualitatively different in their use of violence, compared to nonpsychopaths. Violent psychopaths were more likely to use instrumental aggression (commonly seen as predatory aggression), threats, and weapons, than were violent nonpsychopaths.

Predatory violence and psychopathy appear closely associated. The forensic sciences are gaining increased understanding about the types of behavior of various aggressive offenders. An ability to detect or predict the most violent and habitual offenders has implications for both treatment and public policy.

3.3.2. Stalking

Several typologies have been created to classify individuals who stalk (Harmon, Rosner & Owens, 1995; Mullen, Pathe, Purcell, & Stuart, 1999; Zona, Sharma, & Lane, 1993). The frequency of violence among stalkers ranges from 25% to 35%, which is high in relation to other populations of violent offenders (Meloy, 1998). Most stalkers however are not physically violent and when violence is present, the violence does not typically involve a weapon and the resulting injuries are not serious (Meloy, 1998). The mode of violence involved in the behavior of staking appears somewhat dependent upon the nature of the target (Meloy, 2002). Meloy (2002) distinguishes between two groups of targets, public and private. Public targets are those individuals who have attained some level of recognition or status in the public eye. These individuals may have local prominence (mayor) or sustain national or international recognition (movie star). Private figures consist of everyone else, and have typically been prior sexual intimates or acquaintances of the stalker (Meloy, 2002).

The mode of violence most typically displayed by stalkers who pursue public targets is predatory in nature (Meloy, 2002). Similar to previous descriptions of predatory behavior, the behavior of this stalker is typically premeditated, goal-directed, and is primarily cognitively motivated (Meloy, 2002). Public figures targeted by a stalker are less likely to receive warnings or threatening communications, and therefore are often unaware they are being pursued (Meloy, 2002). This method has tactical advantages, as it adds an element of surprise any may increase the probability of success (Meloy, 2002). Public targets also form one subgroup of a larger cluster of targets labeled strangers. Within this subgroup of stranger targets, there is a greater likelihood that the stalkers are more likely to be psychotic than those who stalk prior sexual intimates, a group typically targeted by stalkers who purse private targets (Meloy, 2002).

Stalkers pursuing private figures typically display affective violence, and rarely does the violence involve a weapon (Meloy, 2002). As such, the violence is characterized by a high level of autonomic arousal, anger, or fear, and is an unplanned response to a perceived threat. The threat typically involves rejection or abandonment by the individual being stalked. The

violent behavior can take on many forms but typically involves pushing, punching, slapping, choking, fondling, shoving, or hair pulling (Meloy, 2002).

3.3.3. Domestic violence

Holtzworth-Munroe (2000) and Holtzworth-Munroe and Stuart (1994), in a review of battering studies, suggest that there are three categories of batterers: dysphoric–borderline, family only, and generally violent–antisocial batters. The aforementioned study by Gottman et al. (1995) showed evidence for two subgroups of batterers: those who are violent outside the home and those who are not. Gottman et al. found that heart rate distinguished between the two groups. In addition, Gottman et al. suggested type 1 and type 2 personality profiles of batterers. The former lowered their heart rates in negative interactions, whereas the latter increased their heart rates. Type 1 men were more likely that type 2 men to be violent outside the home. Type 1 men were also lower on dependency needs and higher on antisocial behavior and sadistic aggression. Thus, lower heart rates and increased violence outside the home can be hypothesized as markers of predatory violence. Babcock, Jacobson, Gottman, and Yerington (2000) observed two modes of violence in their sample of 23 domestically violent and nonviolent husbands who were classified as being secure, preoccupied, or dismissing in their level of attachment. They labeled the violence as expressive violence and instrumental violence. This bimodal classification of violence can be equated to affective and predatory violence, respectively. Those batterers who were evaluated to be preoccupied in their level of attachment exhibited expressive violence (affective), typically in response to a fear of abandonment. Dismissing batterers exhibited instrumental or predatory violence as a way to assert authority and establish control over their wives.

3.3.4. Bombers

There is limited research on bombers. Macdonald (1977) interviewed more than 30 bombers and 100 arsonists. He discovered that six groups were needed to describe the small group of bombers he studied: the psychotic bomber, the sociopathic bomber, the political bomber, the compulsive bomber, the Mafia bomber, and the military bomber. Three other descriptions of bombers have been presented by Brussel (1968), Dietz (1987), and Douglas & Olshaker (1996). Meloy and McEllistrem (1998) linked anecdotal and descriptive data about bombers to what is known about psychopaths (Meloy & McEllistrem, 1998). Both observational and biological data suggest a possible connection between predatory violence and those who bomb. In particular, Raine (1993) found that bomb disposal experts who had been decorated for bravery and fearlessness had lower heart rates than other bomb disposal experts. He also found that the decorated bomb disposal experts had lower heart rates than a control group of soldiers. Clearly, their low resting heart rate, commonly seen in those who commit predatory violence, may help these bomb disposal experts to tolerate extreme stress, hence, setting them apart from their peers. There is currently no direct evidence for equivalent biological processes in bomb disposal experts and actual bombers. There is, however, enough evidence to support the presence of some psychopathic traits in the later, traits that we know are sometimes expressed as predatory aggression.

4. Conclusion

Research on aggression and violence has been ongoing since the mid-1920s. Early observational data in humans and animals led researchers on a quest for neuroanatomical and neurochemical substrates of such behavior. Even in these early studies, clear distinctions were made between modes of aggression. Further research confirmed multiple modes of violence corresponding to specific biogenic and psychogenic substrates.

The physiological sites critical for eliciting and inhibiting both predatory and affective aggression have been well established in animals. The afferent and efferent connections for the expression and regulation of these behaviors have been identified, along with the corresponding physical manifestations of these distinct behaviors. In addition, information regarding corresponding neurochemical systems involved in the production and modulation of these behaviors has slowly been revealed.

The application of these findings to the pathological human condition is precarious, but it has yielded prolific avenues of research and promising results. Although it is difficult to match the level of rigor and sophistication seen in the animal literature to the study of human aggression, several constructs have been researched involving human subjects. The study of natural disease processes that inflict damage on the limbic system has allowed a window of observation on and comparison to neuroanatomical substrates already demonstrated in animal models. Studies of the role of specific neurotransmitter systems have been useful in understanding not only chemical contributions but also avenues to treat and attenuate aggression in humans. Both anatomical and neurochemical studies support at least a bimodal, *predatory* and *affective*, classification system of aggression, which corresponds to unique physiological substrates and phenotypic expression. Although this bimodal classification system describes distinct forms of aggression and recognizes particular physiological substrates, it does not necessarily point to effective treatments. Eichelman and Hartwig (1990) have recognized the multiplicity of factors that can contribute to destructive behavior and have developed a coherent, standardized classification system to unite researchers and clinicians through a common nosology. With this system, a centralized database should be created whereby independent researchers can compare effective treatments in relation to very specific manifestations of destructive behavior.

Finally, these modes of behavior can be applied to forensic cases to better explicate the nature of a violent act. Forensic criteria offered by Meloy (1988, 1997, 1998) allows for better identification of particular manifestations of aggression displayed by an offender. With the support of biochemical, neuroanatomical, and pharmacological findings from the literature on animal and human aggression, forensic experts will be better able to establish the severity of pathological aggression evidenced in a crime. This level of differentiation may then offer clues as to the motivation, level of dangerousness, and risk of recidivism posed by individuals who enact aggression.

Acts of ethnic cleansing, school shootings, bombings, hostage takings, incidents of terrorism—these and other violent acts appear with regularity in both print and visual media. Indeed, worldwide communication can now broadcast scenes of violence from every corner of the world to anyone with a television, radio, or Internet connection. Moreover, advancing

technology has increased the chance that at any given incident, someone with a video camera will capture the scene live, leaving little to the imagination. At least in part because of these developments, greater attention is being paid to those who inflict injury on others and to how such acts are perpetrated.

The victims of such events and the public at large can find redress from the courts. However, many questions about the nature of the person who committed the act are often left unanswered. There appears to be a subjective awareness by many that certain acts of violence are qualitatively different from others, and considerable objective data support the veracity of this observation. The legal system has recognized a distinction for years. For example, murder is prosecuted at different levels, depending on the degree to which the perpetrator planned the crime and on the level of cruelty. Legislative and judicial bodies have also attempted to address this distinction by passing hate-crime legislation and by redefining old behaviors as new crimes (e.g., stalking). Clinicians and researchers alike have been studying these behaviors since the beginning of the 20th century. Even early observers noted distinct forms and etiologies of aggression. After nearly eight decades of study, numerous experts have produced a voluminous literature revealing the complexity of aggression, which will contribute to its detection, control, and alleviation.

Acknowledgements

Thanks to Reid Meloy, PhD, for his patience and indispensable help and guidance in producing this article, and to my wife, Lisa Keating, PhD, for her scholarly help in writing and editing this article. I am thankful to my mentors at the Menninger Clinic who helped guide my in my writing and research, including Lisa Lewis, PhD, Phil Beard, and Maryann Clift.

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