Amnesia and Crime: A Neuropsychiatric Response

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Bourget and Whitehurst's "Amnesia and Crime," published in a prior issue of the *Journal*, addresses a conceptually complex and clinically challenging subject. Their treatment emphasizes psychiatric conditions in which memory disturbances may arise that are relevant to criminal proceedings. However, their consideration of the neurobiology of memory, memory disturbances, and the neurobiological bases of interactions between psychiatric symptoms and memory merit further elaboration. The relevance of memory impairment to criminal matters requires forensic psychiatric experts to possess a basic understanding of the phenomenology and neurobiology of memory. The present authors describe briefly the phenomenology and neuroanatomy of memory, emphasizing first that memory is not a unitary cognitive domain, clinically or neurobiologically. The assertion that psychotic delusions produce memory impairment is challenged, and the description of "organic" amnesia, both semantically and in terms of its clinical features, is reframed. Resources on which to build a neuropsychiatric foundation for forensic psychiatric opinions on memory impairment surrounding criminal behavior are offered.

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The neurosciences are developing rapidly. While there is still much to be learned, considerable advances have been made in defining the neurobiology that underlies cognition, emotion, and behavior. Among the cognitive domains, memory stands out as a function that has been highly investigated, yielding many fruitful insights into its nature, as well as the neuroanatomy and neurochemistry that make this vital cognitive function possible. With this background in mind, we read with great interest Bourget and Whitehurst's article, "Amnesia and Crime."[1] The authors clearly identify a concern of vital importance to forensic psychiatric practice. Indeed, the evaluation of reported amnesia, particularly among criminal defendants, is very challenging and requires a precise approach to the assessment of memory and an understanding of its neurobiology. While the authors' thoughtful synthesis of this exigent topic is to be highly commended, we offer here some observations on this work from a neuropsychiatric perspective and discuss their implications for the practice of forensic psychiatry.

Bourget and Whitehurst,[1] near the end of their paper, indicate that an in-depth review of the neuropsychology and neuroanatomy of memory is beyond the scope of their article. We agree that an in-depth review of this subject is an undertaking of enormous scope. Nevertheless, readers of this journal interested in the topic of amnesia and crime may benefit from an additional review of the phenomenology and neurobiology of memory and memory impairments.

Key Concepts in the Phenomenology of Memory

At the outset, it is important to acknowledge that memory is neither a unitary concept nor a single neuropsychological function.[2,3] The term memory is generally used to refer to the ability to learn, store, and retrieve information. However, there are several different, albeit sometimes overlapping and interrelated, ways of categorizing memory. Such categorization is generally based on the type of information

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learned and retrieved, as well as the duration of the interval between learning and retrieving.

With regard to the type of information learned, two general categories of memory are well accepted: explicit (or declarative) and implicit (or procedural). Declarative memory refers to the ability to learn, encode, and retrieve factual (semantic) information, as well as information regarding events (episodic) or oneself (autobiographical). In other words, explicit memory pertains to who, what, where, and when. This domain of memory is also sometimes divided into spatial and verbal subcategories, with spatial memory denoting the learning, storage, and retrieval of visuospatial information and verbal memory denoting the learning, storage, and retrieval of verbal-linguistic information. Declarative memory is highly associative and subject to representational flexibility (and hence to *post hoc* modification or error).

Implicit, or procedural, memory denotes the ability to learn, store, and recall (by action) skills and procedures, as well as some other nondeclarative sensory events. It generally denotes memory for "how" things are done, and is demonstrated by execution rather than by explanation. Learning in this context implies the tuning and modification of task-related sensorimotor systems (e.g., how one's fingers are moved when playing a violin) rather than the encoding of task outcomes (e.g., the song played by that series of movements). As a result, procedural memory is neither associative nor flexible, and its retrieval is limited to the context in which it was acquired (i.e., actually playing a violin, as opposed to playing another stringed instrument or "air violin"). Both conceptually and clinically, retrieval of procedural memory overlaps considerably with praxis, the execution of a skilled purposeful movement on demand.

With regard to the duration between learning and recall, there are several temporally based types of memory. Working memory describes the process of keeping information in mind or on-line for short-term use in processing additional information and overlaps both conceptually and nosologically with immediate memory. Short-term memory refers to the ability to retain information for a period of several minutes to a few days, and long-term memory refers to the retention of information over a period of days to years. Unfortunately, the interval between learning and recall denoted by short- and long-term memory varies among studies. Accordingly, these terms are potential sources of nosological confusion when the interval between learning and recall they denote is not stated explicitly by the writer or speaker using them.

The Neuroanatomy of Memory in Brief

Declarative memory requires first an intact sensory-cortical pathway through which to acquire (learn) new information. From primary and secondary association cortices, highly processed multimodal sensory information is transmitted from parietal heteromodal association cortices to the entorhinal-hippocampal complex. When that incoming multimodal sensory information produces a sufficiently robust signal in the hippocampus (a process that, at least in part, necessitates amygdala-hippocampal interactions that attach a motivational/emotional, or "survival-related," valence to that information), the process of long-term potentiation (LTP) within the network processing that information is initiated. By forming stable synapses within that network, LTP, a glutamatergically mediated and cholinergically dependent process, serves as the neural basis for encoding.[4,5] Because the hippocampus is necessary for encoding declarative information, the process of new learning of declarative information is described as hippocampally dependent.

The processing stream into which the hippocampus projects via the hippocampal-forniceal-mamillothalamic pathway extends to the frontal areas (and particularly the dorsolateral prefrontal cortex) involved in the process of consolidating new memories. After consolidation, volitional retrieval of declarative information requires prefrontal structures to activate the selective distributed networks in which that information was originally encoded. In contrast to new learning, volitional retrieval of previously learned (consolidated) information is not hippocampally dependent, but is instead frontally dependent. This type of memory is highly associative: reactivation of nearly any part of the network involved in the original encoding of that information, or activation of other networks whose constituent elements are shared by the network involved in the original encoding of that information, will result in automatic (nonvolitional) retrieval of that information.

In general, encoding and retrieving verbal-linguistic declarative information is a left (dominant) hemisphere function. Nonautobiographical episodic memory appears to engage both hemispheres,[6] whereas spatial memory and autobiographic declar-

ative memory appear to be relatively, but not exclusively, more strongly lateralized to the right hemisphere. These latter two types of memory also appear to engage specific additional neuroanatomic areas. In the case of spatial memory, the parahippocampal area is engaged[7]; this area is present bilaterally, but appears to be larger on the right. In the case of autobiographic memory, a predominantly right hemispheric network including the right temporomesial (hippocampal, parahippocampal, and amygdala), temporopolar, and temporolateral cortices, the right posterior cingulate areas, the right insula, and the right prefrontal areas are engaged.[6,8,9]

Unlike declarative memory, procedural memory is predicated on the development and fine-tuning of the sensorimotor-frontal-subcortical-cerebellar networks that are necessary for learning and efficiently retrieving complex sensorimotor routines. Procedural memory therefore is not hippocampally dependent, and its function and dysfunction are dissociable from declarative memory.

Defining Amnesia

Amnesia denotes an impairment of memory. The impairment may be of declarative or procedural memory or of encoding/new learning or recall/retrieval of previously learned information or it may be anterograde (impairment in new learning), retrograde (impairment in the recall of previously learned information), or both (global). The neuroanatomy of memory offers a foundation for interpreting the putative neurobiological bases for amnesia in its various forms. Impaired declarative new learning is generally associated with dysfunction of the hippocampal-forniceal-mammillothalamic pathway, whereas impaired volitional retrieval of declarative information is associated with dysfunction of the frontal-subcortical systems necessary for reactivation of the neural network in which such information is represented. Impaired procedural memory is generally associated with subcortical dysfunction (e.g., as in Parkinson's or Huntington's diseases) and manifests as difficulty learning new motor routines.

When declarative memory impairments develop, anterograde amnesia is the rule. Although rare cases of pure retrograde amnesia due to mechanical trauma or vascular injury have been reported, when retrograde amnesia is present, it is typically accompanied by anterograde amnesia of greater severity. With regard to the content of the retrograde amnesia, information acquired proximate to the time of onset of memory dysfunction is more severely affected than information acquired more remotely (Ribot's law). In other words, events immediately preceding the acquisition of the condition producing the memory impairment (e.g., traumatic brain injury or hypoxic-ischemic brain injury) may be lost, but previously learned semantic and autobiographical information are relatively preserved.

Because the term amnesia may be used to refer to any of the types of memory impairment, its use is generally discouraged in favor of offering specific descriptions of type and severity of the memory functions that are impaired. When clarity on the definition and referent of amnesia is lacking, erroneous inferences and conclusions regarding the relationship between amnesia and other concurrently experienced psychiatric symptoms are inevitable.

Amnesia for Traumatic and/or Stressful Events

The use of the term dissociative amnesia to describe a dense impairment in the recall of all details of a discrete (usually traumatic) event is inconsistent with the phenomenology and neurobiology of trauma-related memory disturbances. Although neurotransmitter and neurohormonal excesses occurring during traumatic experiences may interfere with the normal operation of the hippocampus, and hence with declarative new learning, Brewin[10] notes that lower-level representations of sensory (primarily visuospatial or image-based) information about the trauma generally remain intact. These visuospatial or image-based representations are not dependent on hippocampal processing, but instead are predicated on consolidation of pathways linking cortical and subcortical areas directly to the amygdala (a form of implicit memory). These sensory representations are informationally encapsulated, encode temporal information poorly, and are subject to cued reactivation (experienced as flashbacks) despite poorly consolidated or fragmentary declarative memory of the events to which they are related. When an individual is amnestic for all aspects of a traumatic event, including both its declarative and sensory elements, such amnesia is inconsistent with the effects of stress on memory formation.

This type of trauma-related amnesia may superficially resemble a memory disorder, but it is more accurately understood as a psychological defense

(e.g., repression or suppression), Vaillant's[11] distinctions between these ego defenses notwithstanding. In fact, suppression of emotional memories appears to be an active inhibitory process involving right prefrontal areas (Brodmann area 10 and the right inferior, middle, and superior frontal gyri).[12] Accordingly, suppression of emotional memories does not represent a memory deficit syndrome (amnesia) but instead demonstrates a right frontally mediated ability to inhibit access to (activation of) otherwise normal underlying memories. This amnesia represents active suppression of traumatic information and is most accurately described with the term psychogenic amnesia.

Delusions and Memory

Bourget and Whitehurst[1] suggest, on the basis of two reports (Bradford and Smith[13] and O'Connell[14]), that delusion-related attentional impairments may result in impaired encoding of events that thereby create, albeit indirectly, the possibility of amnesia due to a psychotic (delusional) episode. However, a review of more recent evidence generated from neuroscientific research suggests otherwise.

Delusions are neither the cause nor the consequence of attention and memory impairments, although all of these are common neuropsychiatric comorbidities among persons with conditions in which delusions are prominent, such as schizophrenia.[15] Delusions are linked more closely with impairments in executive function, particularly with failures of error recognition and detection referable to dysfunction in frontoparietal systems.[16] By contrast, the temporal poles, which figure prominently in the storage of personal semantic and episodic memories, appear to be functionally normal among deluded individuals.[6,8,9] Concordant with the imaging of episodic memory systems in this population, patients with schizophrenia are no more likely to produce false memories than are control subjects.[17] When these patients make such errors, they may be more likely to assert them with higher confidence than are control subjects,[17] again a probable reflection of impairments in frontally mediated cognition, or executive function, rather than in temporoparietally dependent declarative new learning.

The notion that delusions result in memory impairments is challenged further by studies of the cooccurrence of these problems in Alzheimer's disease (AD). By definition, typical AD involves, among other cognitive impairments, amnesia, which is defined in this context as an impairment in the encoding (new learning) of declarative (semantic and episodic/autobiographical) information.[18] Delusions commonly arise among persons with AD, at a median frequency of about 37 percent.[19] The presence of misidentification, but not of paranoid delusions, in AD is associated with relatively greater impairments in verbal fluency and visuospatial function; however, persons with or without delusions due to AD do not differ with respect to the severity of their declarative memory impairments.[20]

So, while delusions and memory impairments may co-occur at times, the neuropsychiatric literature does not support the suggestion that delusions result in amnesia, whether in the context of a psychotic disorder such as schizophrenia or an amnestic disorder such as AD. Any suggestion to the contrary will require the expert to produce credible scientific evidence of a causal negative influence of delusions on memory function.

Moving Beyond the Organic Versus Functional Dichotomy

Another concern surrounds the authors' framing of organic amnesia and the statement that "as organic pathology is usually indicated by failures of retention of information, problems with memory storage rather than retrieval may underlie the memory dysfunction in organic amnesia" (Ref. 1, p 471).

The DSM-based system has, since the advent of the Diagnostic and Statistical Manual of Mental Disorders (DSM-III-R),[21] explicitly discouraged the organic-versus-functional dichotomy. The rationale for this position is that "the term organic mental disorder is no longer used because it incorrectly implies that 'nonorganic' mental disorders do not have a biological basis" (Ref. 22, p 135). As noted earlier, whether one is discussing memory disturbances in the context of neurological disorders, psychological trauma, or ego defenses, there is a neurobiological (organic) basis for them all. With this in mind, we agree with the American Psychiatric Association's position that the organic-versus-functional dichotomy be abandoned, particularly when introducing evidence into courts of law.

If Bourget and Whitehurst[1] are not using the term amnesia in this section more restrictively than in other sections of their article, then their description of organic amnesia also fails to com-

port with the neuropsychiatric literature. Many neurological conditions in which frontal-subcortical dysfunction figures prominently—traumatic brain injury, cerebrovascular disease, HIV/AIDS, and multiple sclerosis being among the most common and obvious of them—produce prominent impairments in the retrieval of previously (including recently) learned declarative information.[2,23,24] Suggesting that problems with the storage rather than the retrieval of information are the characteristic type of memory impairment due to neurological disorders (organic amnesia) increases the likelihood that the memory impairments of persons with frontal-subcortical dysfunction will be misunderstood as nonorganic or, worse, malingered. This possibility again highlights the necessity for careful description of memory impairments and attention to the neurobiology on which such impairments are predicated.

Additional Resources on Memory and Memory Disorders for the Forensic Psychiatrist

Claims of amnesia are certainly a common and challenging aspect of forensic psychiatric practice. Again, the efforts of Bourget and Whitehurst[1] to elucidate this difficult topic are appreciated. The authors have aptly set the stage for a much-needed exploration of the subject. At the same time, the neuropsychiatric literature offers important insights into the phenomenology and neurobiology of memory and its disorders, as well as the distinctions between memory impairments and other psychiatric symptoms that may mimic or interact with such impairments.

An optimal discussion of matters of memory can occur only if forensic psychiatric experts interested in crime-related amnesia become experts in the brain anatomy and chemistry that subserve this cognitive function. Precision in the discussion of the terminology, phenomenology, and putative neurobiology of memory and memory impairments is essential if the forensic relevance and biological plausibility of any failure of memory are to be asserted in legal proceedings. Conclusions not rigorously tied to the neuroscience of memory are bound to suffer from imprecision and may mislead the trier of fact.

Fortunately, there are many excellent resources detailing the neuropsychiatric approach to cognition in general, and memory in particular. A broad and accessible review of cognitive impairment is offered in the *Guide to Neuropsychiatric Therapeutics*.[2] This text is rigorously tied to the scientific evidence and is authored by many leaders in the field. The fifth edition of the *American Psychiatric Publishing Textbook of Neuropsychiatry and Behavioral Neurosciences*[25] represents a comprehensive manual. This book, too, is authored by leaders in the field, and represents a more dense and challenging, but highly rewarding, treatise. The *Principles of Behavioral and Cognitive Neurology*[3] features a detailed and gripping chapter on memory and amnesia and is highly regarded within the neuropsychiatric community. Forensic psychiatrists should also be aware of The Law and Neurosciences Project. Supported by a $10 million MacArthur Foundation Grant, the project has been undertaken to investigate the impact of emerging neuroscience on the legal system and to effect thoughtful integration. The project maintains an excellent Web site[26] worthy of exploration. Expertise in the area of memory, particularly its application to expert testimony on amnesia and crime, mandates familiarity with the science delineated in such sources.

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