61

Brain disease: forensic neuropsychiatric issues

MACE BECKSON AND GEORGE BARTZOKIS

Behavioral abnormalities can be a central issue in criminal and civil legal proceedings. Typical examples could include the case of a young woman claiming no recollection for alleged shoplifting; an unusual, problematic contract signed by a wealthy elderly gentleman; a confessed murderer with a recent history of impulsive violent acts; and an otherwise respectable family man accused of sexually deviate behavior. Issues of competence, responsibility, insanity, and mitigating factors require the careful evaluation of behavioral abnormalities.

Brain disease is frequently the basis for behavioral abnormalities involving disturbances of thinking, perceptions, emotions, and actions. In the examples above, the shoplifter may have had a temporal lobe seizure; the elderly gentleman may be suffering from dementia; the murderer may have an undetected frontal lobe tumor, and the family man may have undiagnosed multiple sclerosis. The lay public, attorneys, and even physicians commonly judge the behavior as volitional or as 'mental illness' and fail to consider a medical cause for the behavior or the potential for treating the underlying disease. Of great importance, any distinctly altered behavior or significantly changed personality must be considered evidence of organic brain disorder until ruled out by thorough and appropriate neuropsychiatric evaluation.

THE DIAGNOSTIC PROCESS: AN OVERVIEW

Neuropsychiatry is a medical discipline that specializes in the evaluation and treatment of behavioral disturbance that results from brain disease. Those neurologists and psychiatrists who have specialized training in brain–behavior relationships are capable of undertaking such an investigation and making appropriate determinations. Diagnostic work-up usually includes a detailed medical and behavioral history, complete neurological/physical examination, a thorough psychiatric interview, and an organic mental status examination, augmented by appropriate laboratory tests, such as structural and/or functional brain imaging, electrophysiological testing, neuropsychological testing, and/or other specialized medical tests. Expert assessment of the data and formulation of a probable diagnosis complete this process.

The neuropsychiatric history: clues

As in all of medicine, the history of the current problem is of prime importance in making the proper diagnosis of a behavioral abnormality. Sources of the history may include the individual with the behavioral problem, family members, involved friends, treating physicians, medical records, school records, and legal records. Evidence that a brain disorder has been acquired at some point or has existed from birth should be sought from symptoms, events, illnesses, family history, and growth and developmental history.

Certain important historical clues increase the likelihood of brain disorder:

1 Cognitive difficulties:
   - alertness, attention, or concentration problems;
   - difficulty with comprehension, speech, reading, or writing;
   - memory problems;
   - getting lost or misplacing things; or
   - new difficulty balancing checkbook or handling money.
2 Personality change:
   - apathy or loss of motivation; or
   - socially inappropriate or impulsive behavior.
3 Unusual behavior or change in emotions:
   - agitation;
   - wandering;
   - emotional liability;
   - hallucinations; or
   - paranoia.
4 Recent difficulty doing usual work at job or at home.
5 Known, diagnosed neurological illness, for example, multiple sclerosis, seizures, Alzheimer's disease, encephalitis, stroke.

6 Family history of a familial neurological illness, for example, Huntington's disease, epilepsy.
7 Known head trauma or brain injury, for example, motor vehicle accident resulting in a coma, being knocked unconscious with a blunt object, multiple fights, neurosurgery.
8 Diagnosed psychiatric illness, for example, schizophrenia, manic-depressive illness, especially if:
   - no family history of the illness;
   - atypical age of onset or course of the illness;
   - atypical symptoms or signs for the psychiatric diagnosis;
   - poor response to treatment; or
   - no thorough neuropsychiatric assessment ever performed.
9 Diagnosed or family history of medical illness known to have neurological sequelae, for example, diabetes, liver disease, hypertension, thyroid disease.
10 Family history of psychiatric illness.
11 Treatment with a medication known to have neurological side effects, for example, blood pressure medication, appetite suppressants.
12 Abuse of psychoactive substances, for example, alcohol, stimulants, hallucinogens.
13 Illness or trauma associated with the pregnancy or birth of the individual.
14 Delayed developmental milestones, for example, walking, talking.
15 Poor school achievement, especially known learning disabilities or markedly poorer performance than siblings.
16 Poor social development, for example, behavioral problems in school, fighting with peers.

THE NEUROLOGICAL/PHYSICAL EXAMINATION

The neurological examination may uncover both obvious and subtle signs of brain dysfunction: visual disturbances or other cranial nerve abnormalities; asymmetric or pathologic deep tendon reflexes; lateralized loss of strength, coordination, or sensation; abnormal balance or gait; or abnormal involuntary movements. Physical examination may demonstrate cranial or spinal abnormalities, centers of chronic pain, evidence of congenital or developmental defects, and other signs of underlying disease. The neurological findings may also allow anatomic localization of the dysfunction within the nervous system; this, combined with the other data, may provide clues as to the disease process involved. Both anatomic localization and the disease process may prove pivotal to understanding the associated behavioral abnormalities. Neurological 'soft signs' refer to non-specific abnormalities, for example, posturing while walking. Such signs do not have a definitive anatomic correlate and therefore differ from 'hard' findings (e.g., lateralized hyperreflexia and weakness indicative of a lesion in the motor pathways of the spinal cord or brain), which are clear indicators of specific acquired neurological damage. Isolated soft signs are only weak evidence of neurological disorder.

The mental status examination for organic brain disease

A key aspect in the evaluation of aberrant behavior is the mental status examination with particular stress on those elements reflecting abnormal brain function. This examination includes:

1 Observation of appearance and behavior.
2 Questions regarding the individual's thoughts, perceptions, and emotions.
3 Assessment of insight and judgment.
4 Determination of attentional capabilities and concentration.
5 Assessment of basic language and comprehension abilities.
6 Testing of orientation, learning, and memory.
7 Testing of visuospatial skills, such as copying diagrams.
8 Tests of calculations and abstract interpretation (e.g., proverbs).
9 Testing of ability to perform sequencing tasks, inhibit impulses, and other so-called frontal systems tests.

The results may reveal a wide variety of abnormalities consistent with brain dysfunction. Data from the mental status examination can discover or confirm a confusional state, aphasia, amnesia, a frontal lobe syndrome, and so on. This exam can also provide crucial data for determining which brain structures are malfunctioning, the nature of the disease process affecting them, and the abnormal behaviors that could be anticipated. Abnormalities on the mental status examination can be quantitated with formal neuropsychological testing.

Laboratory tests

In most instances, the diagnosis of behavioral abnormality caused by brain disease is derived from data obtained from the history, the neurological/physical examination, and the mental status examination. Appropriate laboratory tests can be selected to provide confirmatory evidence or to rule out various diagnostic possibilities. In general, laboratory tests function only as supportive adjuncts to the neuropsychiatric examination. Positive results that confirm clinical impressions are of considerable value; the opposite, laboratory tests that disagree with the neuropsychiatric diagnosis, are of questionable value and negative laboratory results are usually of no consequence. 'Normal' brain images [computed tomography (CT) or magnetic resonance imaging (MRI)] or EEGs do not indicate an absence of brain disease. On the other hand, if results of these tests agree with the clinical impression, the diagnosis is probable.

Brain imaging

Brain imaging studies can provide structural or functional information. CT and MRI are structural imaging techniques; positron emission tomography (PET) and single photon emission computed tomography (SPECT) are functional imaging techniques that are used clinically. Structural brain imaging assesses the integrity of brain tissue. Loss of brain tissue or damage to particular structures becomes visually evident with such a procedure. Current clinically available functional imaging assesses metabolism or blood flow. A functional image may reveal loss of function in a brain area; however, it cannot determine whether a particular area has been damaged. Comparison with a structural image would be needed to answer this question. CT scanning is the least expensive, followed by MRI and SPECT, with PET being most expensive.

All imaging studies must be interpreted with caution. Significant functional changes may occur in the absence of abnormality on the structural image and small areas of damage on a structural image are often correlated with wide areas of abnormality on functional image. Which image more accurately reflects the brain basis of the behavioral syndrome demands knowledge of the techniques, as well as the probable pathophysiology of the behavior. A structural imaging study is performed as part of most neuropsychiatric evaluations. A functional image may be added in certain circumstances.

Imaging can be helpful for evaluation in the following:

- Abnormal neurological/physical examination.
- Abnormal mental status examination.
- New onset or atypical features of psychiatric disorder such as psychosis or mood disorder (including atypical age of onset, symptoms, course, or treatment response).
- Personality change.
- New onset of seizures.
- Unexplained confusional state.
- Dementia.
- Movement disorder (e.g., chorea).
- Catatonia.
- Weight loss and behavioral abnormality.
- Abnormal childhood or adolescent development or chronic social or occupational dysfunction.

CT SCAN

The CT scan utilizes ionizing radiation (X-rays) and computed reconstruction to provide a structural image in three dimensions (Oldendorf 1980; Adams and Ropper 2001). The image is produced in a series of thin horizontal slices through the brain (tomograms), outlining structures by their relative specific density. The CT scan can demonstrate acute hemorrhages, old strokes, tissue loss secondary to aging, trauma or degeneration, most tumors, calcified lesions, excessive build-up of cerebrospinal fluid, and lateral pressure causing shift of brain structures. Iodine contrast material, injected intravenously, enhances the image by outlining blood vessels, areas of breakdown of the blood–brain barrier, and extravasations of blood. Contrast enhancement also helps highlight arteriovenous malformation, certain tumors, meningoencephalitis, subarachnoid hemorrhage, abscess, hematoma, contusion, and neoplastic metastases. CT scan, both with and without contrast, is used in most neuropsychiatric work-ups.

MRI

Magnetic resonance imaging also produces three-dimensional tomograms of the brain, but without ionizing radiation. Instead, the image is constructed by a computer from magnetic resonance signals produced by the brain tissue's hydrogen nuclei when 'energized' by radio waves (Garber *et al.* 1988). Bone is visualized as a black void because it is relatively 'inert' (very short relaxation times). MRI can visualize areas obscured by bone artifacts on the CT scan, particularly the anterior temporal lobes (typical source area for seizures), deep subcortical structures (often involved in neuropsychiatric disorders), the cerebellum, and the brainstem. The MRI can also be enhanced by the addition of intravenous gadolinium to mark breakdown of the blood–brain barrier, tumors and metastases, abscess, hematoma, and stroke. MRI requires use of a powerful magnetic field, and so is contraindicated if there is metal within the skull, a cardiac pacemaker is fitted, or internal ferromagnetic surgical clips or devices have been used.

MRI VERSUS CT

In general, MRI is preferable to CT for soft tissue structural imaging unless specific contraindications for MRI are present (Garber *et al.* 1988). It is more sensitive in detecting stroke, seizure focus, tumors, vascular malformations, and degenerative changes. As noted above, MRI shows structures frequently involved in neuropsychiatric syndromes that are obscured by bone in the CT scan. Also, midline structures often involved in neuropsychiatric syndromes (medial temporal lobes, limbic areas) can be imaged with the MRI by utilizing different orientations of tomograms (e.g., sagittal and coronal), which the CT scan cannot provide. White matter lesions (small infarcts, demyelinating lesions, infiltrating tumors) are visualized particularly well with MRI. MRI avoids exposure to ionizing radiation and, in most cases, intravenous contrast is not required. The CT scan is routinely used in emergency situations, such as acute head trauma or intracranial hemorrhage. MRI is often preferred if brain disease is suspected and a CT scan has been normal. The cost of an MRI scan is about the cost of a CT with contrast enhancement.

PET AND SPECT SCANS

PET and SPECT are functional imaging techniques that can be used to detect brain dysfunction that may

not appear as structural damage (Volkow, Brodie, and Bendriem 1991; Holman *et al.* 1991). By measuring energy emitted by rapidly decaying radiolabeled compounds, conclusions can be drawn about the relative functioning of brain areas. For example, labeled glucose can reveal aspects of cellular metabolism, while inhaled inert gases or other tracers carried by the circulatory system can demonstrate characteristics of blood flow. The intensity of activity can be color-coded to more easily visualize abnormalities of metabolism or blood flow. The early stages of dementia (e.g., Alzheimer's disease or multi-infarct dementia) and other degenerative diseases such as Huntington's disease can be demonstrated with these techniques. Small lesions, such as a seizure focus, may appear on a functional but not structural image. Functional imaging can be considered when the history and/or examination imply brain dysfunction, but structural imaging does not reveal pathology.

FUNCTIONAL MRI AND MR SPECTROSCOPY

Based on the same nuclear magnetic resonance technology as MRI, functional MRI (fMRI) and magnetic resonance spectroscopy (MRS) are exciting research tools that hold great promise for the elucidation of normal and pathological brain function and, ultimately, clinical diagnostic applications (Prichard and Cummings 1997). fMRI non-invasively detects increased neuronal activity when the brain is activated; increased blood flow (which correlates with increased activity) causes an increase in T2-weighted signal when newly arriving oxyhemoglobin dilutes the paramagnetic deoxyhemoglobin that suppresses the NMR signal from nearby water molecules (Castelijns *et al.* 2000). MRS provides chemical information on metabolites and offers clues to normal and pathological cerebral metabolic processes (Rudkin and Arnold 1999). Neither fMRI nor MRS has a role in the courtroom at this time.

EEG AND BRAIN MAPPING

The brain transmits information through electrical signals. Voltage potentials can be measured with scalp electrodes. The electroencephalogram (EEG) provides a sensitive recording of brain cortical (surface) electrical activity (Adams and Ropper 2001). EEG is useful for suspected seizure disorder, confusional states, and altered states of awareness. It may be useful for assessing degenerative disorders (slowing of activity) and structural lesions (focal abnormal electrical activity). Because seizures are paroxysmal, an individual EEG record can be normal because no seizure activity occurred at the time of the recording. Furthermore, many seizure foci that lie in deep structures and cause behavioral abnormalities cannot be detected from the scalp. EEG telemetry can be used to provide continuous EEG recording over days or weeks. Special leads, such as sphenoidal or depth eletrodes, can improve proximity to deep brain structures and thus improve detection of seizure foci.

Primarily a research tool, quantitative EEG (QEEG) (sometimes referred to as EEG brain mapping) mathematically processes digitally recorded EEG in order to highlight specific waveform components or transform the EEG into a format that elucidates relevant information (Nuwer 1997). QEEG can provide topographic representation of quantified brain cortical electrical activity that is considerably easier for the non-expert to visualize (Zappulla 1991). Unfortunately, there are many ways in which the data can be misleading. 'Abnormal' results in normal subjects and incorrect diagnoses in patients, i.e., false positives, remain a major disadvantage to QEEG and can create confusion, abuse, and false impressions if used in court, where it has not been accepted under *Frye* or *Daubert* rules (Nuwer 1997). The standard EEG, interpreted by an expert, remains the 'gold standard.'

Neuropsychological testing

Neuropsychological testing and interpretation by a trained neuropsychologist (a clinical psychologist with specialized training in brain function and cognition) can provide a quantitative report of cognitive deficits as compared with age- and sex-adjusted normative values, providing strong evidence of brain disease (Strub and Black 1985). Neuropsychological testing is covered in depth in Chapter 62.

Additional laboratory tests

A wide variety of laboratory tests can be selected, based on the history, examination, and other test results. Such tests include, for example, examination of the cerebrospinal fluid in cases of suspected neurosyphilis; analysis of urine toxicology in suspected substance abusers; HIV serology; blood cell count for evidence of vitamin deficiency; plasma ammonia level as a reflection of liver failure; hormone levels to assess effects of certain brain tumors; continuous cardiac monitoring for evidence of abnormal rhythms; sleep electroencephalography to demonstrate periodic apnea during the night. Although it is beyond the scope of this chapter to detail indications and usefulness of all such tests, they may prove crucial to the establishment of a definitive diagnosis.

TYPICAL SITUATIONS REQUIRING NEUROPSYCHIATRIC EVALUATION

Memory loss and amnesia

The claim by an accused individual of no recollection of an illegal event requires careful consideration. Memory impairment can occur in many disorders, only one of

which is amnesia. By definition, amnesia has four characteristics: (i) normal immediate recall; (ii) serious impairment in learning new information; (iii) relatively intact ability to recall old information (except for a variable period of retrograde amnesia); and (iv) relatively unimpaired cognition and personality (Benson and Blumer 1982). Events occurring during the period of amnesia are not recalled later. A frequent claim is one of loss of recall of events during a circumscribed period of time in the past, although currently the individual is able to learn and recall new information. Transient, short-lived episodes of memory loss, usually lasting a few hours or less, can occur in a variety of conditions (Cummings 1985). Most of these are not amnesias by definition: they primarily involve alteration of attention during a confusional state. Lesions that produce amnesia involve the limbic structures necessary for new learning and recent memory: the hippocampal formation of the medial temporal lobe, the fornix, and the mammillary bodies of the hypothalamus. In the confusional state or dementia, more widespread processes cause multiple brain dysfunctions.

The neuropsychiatric evaluation of possible amnesia proceeds as discussed previously, focusing on elements of history, neurological/physical examination, and mental status examination. Neuropsychological testing can accurately document memory dysfunction as well as the presence or absence of other cognitive deficits. Structural brain imaging and EEG are generally required because of the specific etiologies that must be considered. Other specific-laboratory tests may be required as part of the diagnostic work-up for toxic and metabolic abnormalities. The following discussion covers common circumstances of memory loss.

Alcoholic blackouts can produce transient memory loss and should be suspected in the setting of alcohol abuse. Physical examination may elicit the classic findings of alcoholism with or without the typical denial of abuse. The mental status examination may be normal except for recall of the events that transpired during the blackout period (usually hours to days in duration). Not infrequently, however, mental status examination will reveal impaired cognition reflecting chronic alcohol abuse. Blood alcohol testing can detect current use of alcohol, while blood cell count and serum chemistries may demonstrate pathophysiological effects of chronic alcoholism. Structural brain imaging may reveal cortical atrophy in the chronic alcoholic.

The confusional state, defined by impairment in attention, will produce subsequent memory loss for the period in which the confusional state was present. Toxic and metabolic causes are most common (Cummings 1985; Yudofsky and Hales 1997). In particular, drug or medication ingestion is frequently the cause of confusional state and associated memory lapse. Commonly responsible substances include, among a host of others, barbiturates, tranquilizers, and sleeping pills; analgesics; illicit psychotropic drugs; atropine or related agents; steroids; and anti-parkinsonism medications. The sudden withdrawal of drugs and medications can precipitate a transient confusional state. Medical conditions, such as hypoglycemia, cardiac rhythm abnormalities with compromised cerebral blood flow, hepatic or renal failure, or other metabolic disorders may produce transient confusional states.

Most seizures produce altered consciousness followed by confusion, and seizure must be considered as a potential cause of memory loss – particularly in individuals with previous head trauma or a known diagnosis of epilepsy. The presence of symptoms commonly associated with seizures, such as aura, automatic behaviors, incontinence, or confusion, should be sought. Integrated, purposeful behavior or appropriate responsiveness to questions or commands is not characteristic of seizure states, which produce an alteration of awareness or consciousness. History may reveal previous occurrences of other seizures or an underlying medical condition or injury associated with seizures. Neurological examination and structural brain imaging may determine an underlying condition, for example, brain tumor or stroke. Routine EEG may identify seizure activity or abnormal electrical activity consistent with a seizure focus. PET scanning may identify a seizure focus.

Migraine headache may also be associated with transient confusional state and memory loss. Migrainous symptoms, including headache, nausea, photophobia, and visual hallucinations, should be sought. Memory problems occur in dementia syndromes where the memory deficits are part of an overall loss of intellectual function, and are thereby distinguished from the amnesias.

Transient disorders of learning include posttraumatic amnesia, transient global amnesia, and psychogenic amnesia. Head trauma routinely produces an amnestic syndrome that is usually short-lived and resolves spontaneously. A history is pivotal for such a circumstance; other signs or symptoms of brain injury may or may not be present upon physical or mental status examination or upon laboratory testing such as brain imaging. Transient global amnesia is a disorder of the middle-aged or elderly in which memory loss persists for hours and then resolves. This condition may be associated with cerebrovascular disease in the posterior circulation, but it has also been reported in association with other etiologies including diazepam overdose, tumors, and seizures; in almost half of reported cases, however, no specific cause is demonstrated.

Psychogenic amnesia is a conversion symptom in which personally emotionally charged information is selectively lost. It is generally short-lived, remitting spontaneously, but almost invariably indicates a significant underlying psychiatric disorder. It may be overcome with the help of amobarbital (Amytal) interview or hypnosis. This diagnosis should be considered only when the memory disorder involves personal data (e.g., name) with little or no problem learning other information. The

temptation may be great to assume that a memory loss is the result of a psychological process, especially if it appears that the patient stands to gain psychologically or monetarily by not remembering. This temptation must be resisted pending a formal evaluation.

Feigned memory loss, involving purposeful deceit, is another distinct possibility. Mental status examination may reveal no abnormalities, or may reveal inconsistencies suggestive of lying. For example, severe deficits may be shown on mental status examination despite objectively intact behavioral function; new learning may be demonstrated while the individual claims no recall of recent events. Neuropsychological testing may be helpful in making this determination. In addition to cognitive evaluation, other psychological test instruments such as the Minnesota Multiphasic Personality Inventory (MMPI) and Rorschach Ink Blot may reveal a conscious effort to deceive. All of these tests, however, must be validated against the clinical impression: the neuropsychological tests can be manipulated sufficiently to provoke misinterpretation. Other laboratory tests demonstrate only normal findings in cases of feigned memory loss.

Acquired neurological disorders and dementia

An individual may demonstrate clear abnormalities of intellectual functioning, leading to questions regarding competence, or may demonstrate behavioral abnormalities, which upon closer examination are part of a syndrome produced by acquired neurological disease. An individual may suffer a loss of cognitive or behavioral function as a result of focal structural lesions (e.g., tumor, stroke) or more diffuse or global processes (e.g., degenerative disease, head trauma, infection). The loss may be specific and demarcated, for example, a loss of memory function (amnesia) as discussed above, or it may be more progressive and generalized, simultaneously involving multiple cognitive domains (dementia). Depending on lesion localization, a variety of neuropsychiatric syndromes have been described (Beckson and Cummings 1991). Mental retardation is not an acquired loss; it refers to a diminished level of functioning from birth that may nevertheless be associated with behavioral abnormalities of legal consequence.

Language disorder (aphasia) presents in many forms, sometimes simplified as disturbances of comprehension (fluent aphasia) or verbal output (non-fluent aphasia). Specific areas of the temporal and frontal cortices, usually in the left hemisphere, are crucial for the comprehension and output of language, respectively. Fluent aphasias, in which the individual produces nonsense jargon and cannot comprehend what others are saying or what is written, are easily confused with psychotic illness because of inappropriate responses and agitated or paranoid behavior. There may be no gross neurological deficit that might otherwise prompt neurological suspicions. Non-fluent aphasias are frequently associated with severe depression. While the individual understands some written and spoken language, his or her difficulty communicating, further complicated by depressive withdrawal and apathy, may suggest dementia. Aphasic patients are not necessarily demented; in fact, their other cognitive domains may be perfectly intact if tested in a way that allows appropriate responses. A depressed, non-fluent aphasic patient may improve sufficiently with speech therapy and antidepressant medication to be self-reliant in a way that a demented patient could not. Disturbances in language should be addressed by a thorough neuropsychiatric evaluation to determine the nature of the clinical syndrome, particularly to establish whether other cognitive domains are involved, and to identify the causative etiology of the disturbance. The testamentary capacity of aphasics has been studied and relatively firm guidelines have been established (Critchley 1970; Benson 1992).

Visuospatial disturbances, often reflecting parietal lobe disease, may disrupt an individual's ability to properly perceive or work with spatial orientation and relationships, thereby causing substantial functional impairment. The functional ramifications for the patient's occupation and life can be significant but, like language deficit, can exist in the absence of dementia or any other additional cognitive deficit.

Frontal lobe injury routinely produces marked behavioral disorders, often in the absence of any obvious neurological findings (Stuss and Benson 1986). Orbitofrontal injury may lead to marked personality change with emotional lability, poor insight and judgment, and disinhibited behavior, including antisocial acts. Medial frontal damage leads to apathy and indifference, which may be easily mistaken for depression, though there may also be short-lived outbursts of aggression. Lateral convexity lesions produce distractibility, sequencing and categorization difficulties, and loss of ability to effectively plan and execute complex tasks. Despite the intactness of basic areas such as language, frontal brain damage produces severe functional limitations. Neuropsychological testing often fails to demonstrate any basic cognitive deficits. The most common source of frontal injury is blunt head trauma. Tumors such as gliomas, meningiomas, and pituitary adenomas, as well as anterior communicating artery aneurysms, also cause frontal dysfunction.

The dementia syndrome consists of acquired deficits in at least three of the following cognitive domains: speech/language, memory, visuospatial, calculation/abstraction, and personality (Cummings and Benson 1992). The deficits are greater than the mild intellectual decline often associated with normal aging. The early stages may be insidious, with personality change or behavioral abnormalities dominating the clinical picture and the problem may be confused with psychiatric disorders such as depression and psychosis. Some dementia

syndromes are reversible, depending on the underlying cause. Once the dementia syndrome has been established from the history and mental status examination, a correct determination of etiology follows from combining these data with the results of neurological/physical examination, blood tests, cerebrospinal fluid analysis, EEG, structural and functional brain imaging. Neuropsychological testing can quantitate the severity of the cognitive deficits and provide a baseline for future assessment for evidence of a progressive, deteriorating course. While dementia most commonly results from Alzheimer's disease or cerebrovascular disease (multi-infarct dementia), other less frequent etiologies include extrapyramidal diseases, hydrocephalus, demyelinating diseases, and toxic-metabolic, traumatic, infectious (including HIV), and neoplastic causes. Treatment and prognosis follow from correct diagnosis.

Violence

Violence, aggression, impulsive acts, and conduct disorders are of great forensic import. Various neuropsychiatric disorders have been associated with violent behavior. Human aggression can result from hypothalamic, temporolimbic, and frontal cortical lesions (Weiger and Bear 1988; Elliott 1992). The episodic dyscontrol syndrome is marked by attacks of explosive rage and violence directed at people or objects, often with a primitive quality and remarkable displays of strength (Rickler 1982; Elliott 1990). The attacks are recalled and remorse is usually expressed. Some (Mark and Ervin 1970) have included as part of the syndrome episodes of pathological intoxication (relatively small quantities of alcohol produce bizarre behavior and amnesia), reckless driving, and sexual impulsiveness. Episodic dyscontrol has been related to a number of different etiologies including traumatic brain injury, temporal lobe epilepsy, minimal brain dysfunction, encephalitis, meningitis, midline tumors, multiple sclerosis, stroke, subarachnoid hemorrhage, normal pressure hydrocephalus, hypoglycemia, hyponatremia, and premenstrual syndrome, but in many instances no specific etiology is determined.

Frontal lobe disease and violent behavior have a clear association. Orbitofrontal brain damage leads to irritability, disinhibition, and impulsiveness. Trivial provocation may cause outbursts of anger and impulsive actions that are short-lived but leave no resentment or remorse. Closed-head trauma is the most common etiology of frontal brain damage, but tumors, aneurysms, subarachnoid hemorrhage, encephalitis, and multiple sclerosis can also cause this syndrome. Aggressive behavior in dementia and mental retardation may reflect frontal involvement. In the Vietnam Head Injury Study of veterans who had suffered penetrating head injuries, patients with frontal ventromedial lesions consistently scored higher on aggression and violence scales compared with controls and patients with lesions in other brain areas (Grafman *et al.* 1996). Some have hypothesized that impulsive aggression and violence arise as the consequence of dysfunctional frontal lobe function relating to serotonergic dysregulation (Davidson, Putnam and Larson 2000). A study of community violence and inpatient assaults found that violence was related to poor performance on neuropsychological tests of frontal lobe function (Krakowski *et al.* 1997). Basal ganglia disorders, such as Huntington's Disease, which produce deficits in frontal systems function, have been associated with psychosis and violence (Beckson and Cummings 1992).

Mass lesions or brain tumor of the left temporal lobe can present as rage attacks (Piacente 1986). Tumor invasion of the hypothalamus has been associated with violent behavior in response to minimal provocation. Epilepsy may be a source of violent behavior and is considered in Chapter 60. Sleep-related violent behavior can be associated with parasomnias. In one study, serious and harmful violent acts were more likely to occur with males who showed sleep schedule disorder and abused drugs (Moldofsky *et al.* 1995).

Intoxication with alcohol or drugs is a common circumstance for violent behavior with impaired judgment and poor impulse control as hallmarks (see Chapter 70). Any metabolic derangement producing confusion can potentially be associated with violent behavior; the behavior is a result of poor judgment, and is not premeditated or organized. Psychotic paranoid delusional disorders resulting from brain disease may result in violent behavior consistent with the individual's delusional system.

Minimal brain dysfunction, often combined with attention deficit disorder as a syndrome complex, is found frequently in individuals who are prone to violence. The syndrome consists of deficits in attention, impulsivity, specific learning disabilities, and neurological soft signs, despite normal or even superior intelligence. It has been postulated to indicate a scatter of developmental or acquired areas of brain dysfunction (Elliott 1990).

A number of studies have examined the neurological signs, neuropsychological tests, EEGs, CT scans, and PET scans in violent offenders, most often producing evidence of non-specific abnormalities, often within the frontal and temporal lobes. A review of fourteen studies assessing EEG abnormalities in prison inmates and patients with antisocial behavior revealed increased frequency of EEG changes, in most studies ranging from 24 per cent to 78 per cent, and more commonly found in subjects who had committed violent acts and done so more than once, particularly if there was no apparent motive (Cummings 1985). EEG abnormalities have included generalized and focal slowing, as well as epileptiform irregularities, most often found in limbic areas including temporal and frontal lobes. A retrospective study of 372 male patients in a maximum-security mental hospital revealed that in the group of most violent patients, 20 per cent had focal temporal electrical abnormalities on EEG and 41 per

cent had structural abnormalities localized to temporal lobe on CT scan; such findings were relatively infrequent in the least violent group (Wong *et al.* 1994). Neurological soft signs and poorer performance on neuropsychological testing (especially frontal lobe tasks) have also been reported in criminally violent or impulsive populations. Whether the abnormalities reflect the sequelae of head trauma or some other underlying congenital, developmental, or acquired brain disease that results in personality and behavioral disturbance remains unclear (Cummings 1985).

Lewis *et al.* (1986) reported a series of fifteen death row inmates, finding a history of severe head injury in all fifteen, major neurological impairment in five, and less serious neurological impairment (e.g., blackouts, soft signs) in seven others; neuropsychological testing also revealed impairment. In a recent study of sixteen inmates on death row in California, twelve had histories of traumatic brain injury (Freedman and Hemenway 2000). Tancredi and Volkow (1988) demonstrated frontal and temporal lobe abnormalities on the PET scans of four violent patients; all four had neurological examinations that were normal, while two of the four had abnormal CT scans and abnormal EEGs. Additional hypotheses and investigation has been conducted on other biologic aspects of violence, but are beyond the scope of this chapter. Neuropsychiatric evaluation of the violent offender is often indicated to assess for evidence of brain disease; in some cases the disorder is treatable. Brain imaging and EEG should complement the history and neurological and mental status examination in attempting to discern brain dysfunction and its etiology. Neuropsychological testing may reveal mild but pertinent cognitive deficits.

REFERENCES

Adams, R.D., Ropper, A.H. 2001: *Adam and Victor's Principles of Neurology*, 7th edition. New York: McGraw-Hill.

Beckson, M., Cummings, J.L. 1991. Neuropsychiatric aspects of stroke. *International Journal of Psychiatry in Medicine* **21**, 1–15.

Beckson, M., Cummings, J.L. 1992. Psychosis in basal ganglia disorders. *Neuropsychiatry, Neuropsychology, and Behavioral Neurology* **5**, 126–31.

Benson, D.F. 1992: Psychiatric aspects of aphasia. In: Yudofsky, S.C., Hales, R.E. (eds), *American Psychiatric Association Textbook of Neuropsychiatry*, 2nd edition. Washington, DC: American Psychiatric Association Press.

Benson, D.F., Blumer, D. 1982: Aphasia: a clinical approach. In Benson, D.F., Blumer, D. (eds), *Psychiatric Aspects of Neurologic Disease.* Volume 2. New York: Grune & Stratton, 251–78.

Castelijns, J.A., Lycklama, A., Nijeholt, G.J., Mukherji, S.K. 2000. Functional MRI: background and clinical applications. *Seminars in Ultrasound CT and MR* **21**, 428–33.

Critchley, M. 1970: Testamentary capacity in aphasia. In Critchley, M. (ed.), *Aphasiology.* London: Edward Arnold Limited, 288–95.

Cummings, J.L. 1985: *Clinical Neuropsychiatry.* Orlando: Grune and Stratton.

Cummings, J.L., Benson, D.F. 1992: *Dementia: A Clinical Approach*, 2nd edition. Boston: Butterworth-Heinemann Medical.

Davidson, R.J., Putnam, K.M., Larson, C.L. 2000. Dysfunction in the neural circuitry of emotion regulation: a possible prelude to violence. *Science* **289**, 591–4.

Elliott, F.A. 1990. Neurology of aggression and episodic dyscontrol. *Seminars in Neurology* **10**, 303–12.

Elliott, F.A. 1992. Violence: the neurologic contribution. An overview. *Archives of Neurology* **49**, 595–603.

Freedman, D., Hemenway, D. 2000. Precursors of lethal violence: a death row sample. *Social Science and Medicine* **50**, 1757–70.

Garber, H.J., Weilburg, J.B., Buonanno, F.S., Manschreck, T.C., New, P.F.J. 1988. Use of magnetic resonance imaging in psychiatry. *American Journal of Psychiatry* **145**, 164–71.

Grafman, J., Schwab, K., Pridgen, A., Brown, H.R., Salazar, A.M. 1996. Frontal lobe injuries, violence, and aggression: a report of the Vietnam Head Injury Study. *Neurology* **46**, 1231–8.

Holman, L.B., Nagel, J.S., Johnson, K.A., Hill, T.C. 1991. Imaging dementia with SPECT. *Annals of the New York Academy of Sciences* **620**, 165–74.

Krakowski, M., Czobor, P., Carpenter, M.D., Libiger, J., Kunz, M., Papezova, H., Parker, B.B., Schmader, L., Abad, T. 1997. Community violence and inpatient assaults: neurobiological deficits. *Journal of Neuropsychiatry and Clinical Neuroscience* **9**, 549–55.

Lewis, D.O., Pincus, J.H., Feldman, M., Jackson, L., Bard, B. 1986. Psychiatric, neurological, and psychoeducational characteristics of 15 death row inmates in the United States. *American Journal of Psychiatry* **143**, 838–45.

Mark, V.H., Ervin, F.R. 1970: *Violence and the Brain.* New York: Harper and Row.

Moldofsky, H., Gilbert, R., Lue, F.A., MacLean, A.W. 1995. Sleep-related violence. *Sleep* **18**, 731–9.

Nuwer, M. 1997. Assessment of digital EEG, quantitative EEG, and EEG brain mapping: report of the American Academy of Neurology and the American Clinical Neurophysiology Society. *Neurology* **49**, 277–92.

Oldendorf, W.H. 1980: *The Quest for an Image of the Brain.* New York: Raven Press.

Piacente, G.J. 1986. Aggression. *Psychiatric Clinics of North America* **9**, 329–39.

Prichard, J.W., Cummings, J.L. 1997. The insistent call from functional MRI. *Neurology* **48**, 797–800.

Rickler, K.C. 1982: Episodic dyscontrol. In Benson, D.F., Blumer, D. (eds), *Psychiatric Aspects of Neurologic*

Disease. Volume 2. New York: Grune and Stratton, 49–74.

Rudkin, T.M., Arnold, D.L. 1999. Proton magnetic resonance spectroscopy for the diagnosis and management of cerebral disorders. *Archives in Neurology* **56**, 919–26.

Strub, R.L., Black, W.F. 1985: *The Mental Status Examination in Neurology*, 2nd edition. Philadelphia: F.A. Davis Company.

Stuss, D.T., Benson, D.F. 1986: *The Frontal Lobes*. New York: Raven Press.

Tancredi, L.R., Volkow, N.D. 1988. Neural substrates of violent behavior: implications for law and public policy. *International Journal of Law and Psychiatry* **11**, 13–49.

Volkow, N.D., Brodie, J., Bendriem, B. 1991. Positron emission tomography: basic principles and applications in psychiatric research. *Annals of the New York Academy of Sciences* **620**, 128–44.

Weiger, W.A., Bear, D.M. 1988. An approach to the neurology of aggression. *Journal of Psychiatric Research* **22**, 85–98.

Wong, M.T., Lumsclen, J., Fenton, G.W., Fenwick, P.B. 1994. Electroencephalography, computerized tomography, and violence ratings of male patients in a maximum-security mental hospital. *Acta Psychiatrica Scandinavica* **90**, 97–101.

Yudofsky, S.C., Hales, R.E. 1997: *American Psychiatric Association Textbook of Neuropsychiatry*, 3rd edition. Washington, DC: American Psychiatric Press.

Zappulla, R.A. 1991. Fundamentals and applications of quantified electrophysiology. *Annals of the New York Academy of Sciences* **620**, 1–21.

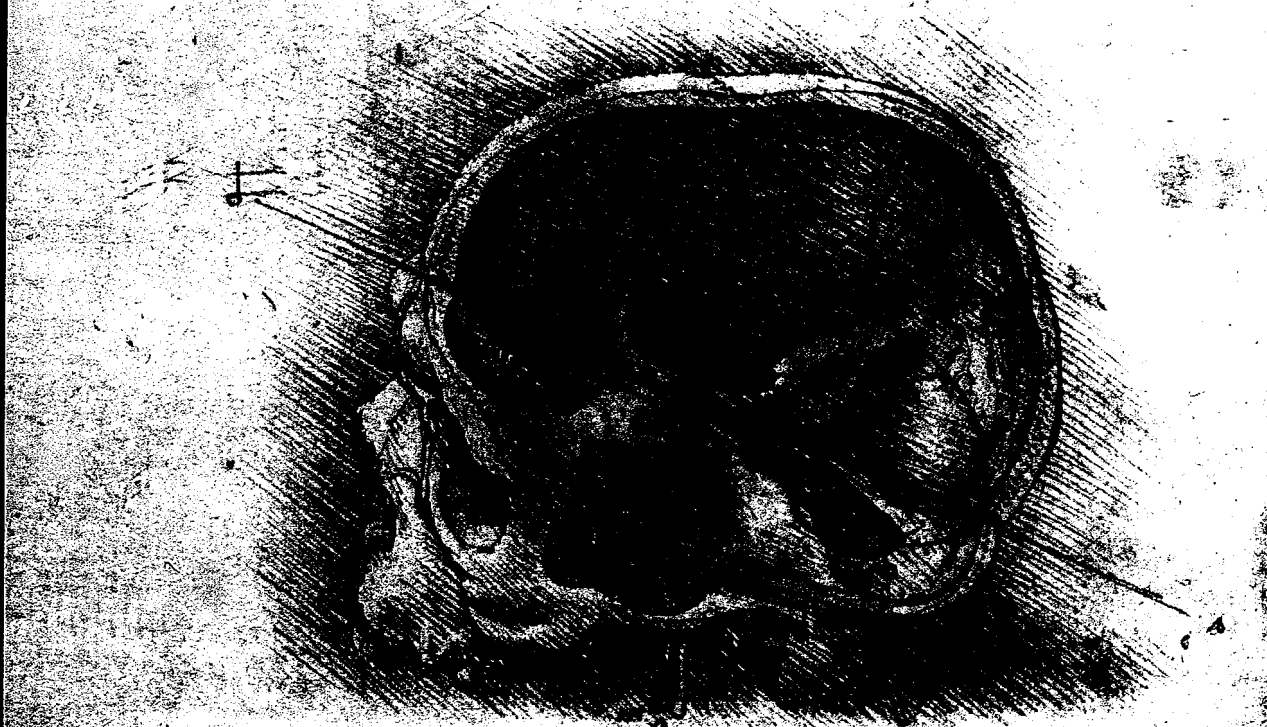
Suggested reading

Cummings, J.L. 1985: *Clinical Neuropsychiatry*. Orlando: Grune and Stratton.

Cummings, J.L., Benson, D.F. 1992: *Dementia: A Clinical Approach*, 2nd edition. Boston: Butterworth-Heinemann Medical.

Miller, B.L., Cummings, J.L. 1999: *The Human Frontal Lobes: Functions and Disorders*. New York: Guilford Press.

PRINCIPLES & PRACTICE OF FORENSIC PSYCHIATRY



Edited by

RICHARD ROSNER

EDITION 2

Principles and practice of forensic psychiatry

Second Edition

Edited by

RICHARD ROSNER MD
Clinical Professor, Department of Psychiatry,
New York University School of Medicine

Director, Forensic Psychiatry Residency,
New York University Medical Center

Medical Director, Forensic Psychiatry Clinic,
Bellevue Hospital Center, New York,
New York

A member of the Hodder Headline Group
LONDON

First published in Great Britain in 1994
Reprinted in 1998 by Arnold, a member of the Hodder Headline Group
338 Euston Road, London NW1 3BH
This edition published in 2003 by Arnold

http://www.arnoldpublishers.com

Distributed in the USA by
Oxford University Press Inc.,
198 Madison Avenue, New York, NY10016
Oxford is a registered trademark of Oxford University Press

British Library Cataloguing in Publication Data
A catalogue record for this book is available from the British Library

Library of Congress Cataloging-in-Publication Data
A catalog record for this book is available from the Library of Congress

ISBN 0 340 80664 8

1 2 3 4 5 6 7 8 9 10

Commissioning Editor: Serena Bureau
Development Editor: Tim Wale
Production Controller: Bryan Eccleshall
Production Editor: Anke Ueberberg
Cover Design: Terry Griffiths

Typeset in 10/12 Minion by Charon Tec. Pvt. Ltd, Chennai, India
Printed and bound in Italy

What do you think about this book? Or any other Arnold title?
Please send your comments to feedback.arnold@hodder.co.uk