

2 | Basic Concepts

If our brains were so simple that we could understand them, we would be so simple that we could not.

Anonymous [from dwt]

EXAMINING THE BRAIN

The usual clinical approach to the study of brain functions has, historically, been the neurological examination, which includes extensive study of the brain's chief product—behavior. The neurologist examines the strength, efficiency, reactivity, and appropriateness of the patient's responses to commands, questions, discrete stimulation of particular neural subsystems, and challenges to specific muscle groups and motor patterns. The neurologist also examines body structures, looking for such evidence of brain dysfunction as swelling of the retina or atrophied muscles due to insufficient neural stimulation. In the neurological examination of behavior, the clinician reviews behavior patterns generated by neuroanatomical subsystems, measuring patients' responses in relatively coarse gradations or noting their absence.

Following the development of scanning techniques, beginning in the mid-1970s, imaging has become a critical part of the diagnostic workup for most patients. *Computerized tomography (CT)* and *magnetic resonance imaging (MRI)* techniques reconstruct different densities and constituents of internal structures into clinically useful shadow pictures of the intracranial anatomy (Beauchamp and Bryan, 1997; R.O. Hopkins, Abildskov, et al., 1997; Hurley et al., 2002).

Neuropsychological assessment is another method of examining the brain by studying its behavioral product. Since the subject matter of neuropsychological assessment is behavior, it relies on many of the same techniques, assumptions, and theories as does psychological assessment. Also like psychological assessment, neuropsychological assessment involves the *intensive* study of behavior by means of interviews and standardized scaled tests and questionnaires that provide relatively precise and sensitive indices of behavior. The distinctive character of neuropsychological assessment lies in a conceptual frame of reference that takes brain function as its point of departure. Regardless of whether a behavioral study is undertaken for clinical or research

purposes, it is neuropsychological so long as the questions that prompted it, the central issues, the findings, or the inferences drawn from them ultimately relate to brain function. Like neurology, the findings are interpreted within the clinical context of the patient's presentation and other observations (see Chapter 5).

Direct observation of the fully integrated functioning of living human brains will probably always be impossible, although many rapidly evolving technological advances are bringing us closer to this goal (D'Esposito and Postle, 2002; Heeger and Ress, 2002). These newer examinations techniques rely upon indirect, typically noninvasive, methods. Originally developed for clinical examinations, these techniques are also used by cognitive neuroscientists and neuropsychologists to examine correlates of virtually all aspects of behavior in both patient populations and healthy subjects (e.g., see Gazzaniga, 2000, *passim*; Hugdahl and Davidson, 2003, *passim*).

The earliest instruments for studying brain function that continue to be in use are electrophysiological (e.g., see Daube, 2002, *passim*). These include *electroencephalography (EEG)*, *electrodermal activity*, and *evoked and event-related potential (EP, ERP)*. EEG frequency and patterns not only are affected by many brain diseases but have also been used to study aspects of cognition; e.g., high frequency has been associated with attentional activity for decades (Sheer and Schrock, 1986; Oken and Chiappa, 1985). Some investigators now hypothesize that it documents conscious awareness by demonstrating integrated neural activity across distributed cerebral regions (Crick and Koch, 1998; Meador, Ray, Echaz et al., 2002). *Magnetoencephalography* (the magnetic cousin of the EEG that records magnetic rather than electrical fields) has also been increasingly used to examine brain functions in patients and healthy volunteers alike (Reite, Cullum, et al., 1993; Reite, Teale, and Rojas, 1999).

Electrodermal activity (with components such as skin conductance level and galvanic skin response) reflects autonomic nervous system functioning, provides a measure of emotional response (Bauer, 1998; Critchley, 2002; Zahn and Mirsky, 1999), and has demonstrated recognition in the absence of conscious perception (Tranel and Damasio, 2000). Both EP and ERPs have demonstrated hemispheric specialization (Papanicolaou,

Moore, Deutsch, et al., 1988; Papanicolaou, Moore, Levin, and Eisenberg, 1987) and processing speed and efficiency (J.J. Allen, 2002; Picton et al., 2000; Zappoli, 1988).

The large volumes of data generated by these techniques may be quantified and analyzed using a variety of statistical methods. Quantified data may then be displayed on a stylized head or brain image or MRI, a practice that is often referred to as *brain mapping* (F.H. Duffy, Iyer, and Surwillo, 1989; Nuwer, 1989). Numerous technological and methodological problems in this practice, however, lead to a high rate of erroneous interpretations. Thus, whether EEG/EP brain mapping techniques should be employed in routine clinical assessments of many different kinds of patients has become a controversial issue (Nuwer, 1997).

Other noninvasive methods that permit the study (and visualization) of ongoing brain activity (Friedland, 1990; Oder et al., 1996) are often collectively called *functional brain imaging*. These techniques have proven useful for exploring both normal brain functioning and the nature of specific brain disorders (Buckner, 2000; Pincus and Tucker, 2003, *passim*; P. Zimmerman and Leclercq, 2002). Neuropsychologists interested in these methods should become familiar with the many assumptions and methodological concerns about them (see Papanicolaou, 1999, for a review).

Regional cerebral blood flow (rCBF), one of the older functional brain imaging techniques, reflects the brain's metabolic activity indirectly as changes in the magnitude of blood flow in different brain regions. rCBF provides a relatively inexpensive means for visualizing and recording brain function (D.J. Brooks, 2001; Deutsch et al., 1988; Nobler, Mann, and Sackeim, 1999; Nobler, Olivet, and Sackeim, 2002; Risberg, 1989). *Positron emission tomography (PET)* visualizes brain metabolism directly as glucose radioisotopes emit decay signals, their quantity indicating the level of brain activity in a given area (Oder et al., 1996; J.C. Patterson and Kotrla, 2002; C. Price and Friston, 2003). PET not only contributes valuable information about the functioning of diseased brains but has also become an important tool for understanding normal brain activity (Aguirre, 2003; Cabeza and Nyberg, 2000; George et al., 2000; Reiman et al., 2000; Rugg, 2002; Tzourio et al., 1995). *Single photon emission computed tomography (SPECT)* is similar to PET but less expensive and involves a contrast agent that is readily available. Comparison of interictal and ictal SPECT scans in epilepsy surgery candidates has been valuable for identifying seizure onset (So, 2000). In studies of cognition and other forms of behavior, these procedures typically compared data obtained during an activation task of interest (e.g., verbalizations) to data from a resting or other control state. The contribution

of the activation task is inferred from the differences between the two task conditions.

However, each of these procedures has limitations. For example, PET applications are limited by their dependence on radioisotopes that must be generated in a nearby cyclotron and have only a short half-life (J.C. Patterson and Kotrla, 2002) and by their cost (Reiman et al., 2000). SPECT generally does not have the necessary temporal and spatial resolution for use in activation studies of cognition. Moreover, many studies using these methods to investigate brain-behavior relationships report group findings rather than for individuals, thus limiting their clinical application.

Functional magnetic resonance imaging (fMRI) has already produced an impressive number of studies, and this can be expected to increase at an ever expanding rate. In addition to the many current clinical applications of motor and language mapping, fMRI has become a popular method for investigating traditional psychological processes such as time perception (S.M. Rao, Mayer, and Harrington, 2001), semantic processing (Bookheimer, 2002), emotional processing (M.S. George et al., 2000; R.C. Gur, Schroder, et al., 2002), response inhibition (Durstun et al., 2002), face recognition (Joseph and Gathers, 2002), somatosensory processing (Meador, Allison, Loring et al., 2002), sexual arousal (Arnold et al., 2002), and many others. More than the other procedures discussed, fMRI will greatly affect neuropsychology as well as cognitive neuroscience in general, in part due to its widespread use.

The clinical need to identify cerebral language and memory dominance in neurosurgery candidates led to the development of invasive techniques such as the *Wada test* (intracarotid injection of amobarbital for temporary pharmacological inactivation of one side of the brain) and electrical cortical stimulation mapping to minimize the surgical risk to these functions (Loring, Meador, Lee, and King, 1992; Branch, Milner, and Rasmussen, 1964; Ojemann, Cawthon, and Lettich, 1990; Penfield and Rasmussen, 1950). Not only have these procedures significantly reduced cognitive morbidity following epilepsy surgery, but they have also greatly enhanced our knowledge of brain-behavior relationships. Atypical language representation, for example, alters the expected pattern of neuropsychological findings, even in the absence of large cerebral pathology (Loring, Strauss, et al., 1999).

These procedures have impediments in that they are invasive and afford only a limited range of assessable behavior due to the restrictions on patient response in an operating theater and the short duration of medication effects. Generalizability of data obtained by this technique is further limited by the atypical functioning of these patients' diseased or damaged brains.

Many of the same questions addressed by the Wada test and cortical stimulation mapping in patients may be answered in studies of healthy volunteers using such noninvasive techniques as *transcranial magnetic stimulation* (C.M. Epstein et al., 1999; L.C. Robertson and Rafal, 2000), *functional transcranial Doppler* (Knecht et al., 2000), *magnetoencephalography/magnetic source imaging* (Papanicolaou et al., 2001; Simos, Breier et al., 1999; Simos, Castillo, et al., 2001), and *fMRI* (J.R. Binder, Swanson, et al., 1996; Desmond et al., 1995; Detre et al., 1998; W.D. Gaillard et al., 2000; Jokeit et al., 2001).

"BRAIN DAMAGE" AND "ORGANICITY"

Throughout the 1930s and 40s and well into the 50s, most clinicians treated "brain damage" or brain dysfunction as if it were a unitary phenomenon—"organicity." The determination of whether a patient was "organic" was often the reason for neuropsychological consultation. It was well recognized that brain damage resulted from many different conditions and had different effects (Babcock, 1930; Klebanoff, 1945) and that certain specific brain-behavior correlates, such as the role of the left hemisphere in language functions, appeared with predictable regularity. Yet much of the work with "brain damaged" patients was based on the assumption that organicity was characterized by one central and therefore universal behavioral defect (K. Goldstein, 1939; Yates, 1954). Even so thoughtful an observer as Teuber could say in 1948 that, "Multiple-factor hypotheses are not necessarily preferable to an equally tentative, heuristic formulation of a general factor—the assumption of a fundamental disturbance . . . which appears with different specifications in each cerebral region. In fact, the assumption of a fundamental disturbance may have definite advantages at the present state of knowledge" (pp. 45–46).

The early formulations of brain damage as a unitary condition that is either present or absent were reflected in the proliferation of single function tests of "organicity" that were evaluated, in turn, solely in terms of how well they distinguished "organic" from psychiatric patients or normal control subjects (e.g., Klebanoff, 1945; Spreen and Benton, 1965; Yates, 1954). The "fundamental disturbance" of brain damage, however, turned out to be exasperatingly elusive. Despite many ingenious efforts to devise a test or examination technique that would be sensitive to organicity per se—a neuropsychological litmus paper, so to speak—no one behavioral phenomenon could be found that was shared by all brain injured persons but by no one else. This one-dimensional approach to neuropsychological

assessment continues to show up occasionally in the literature and in clinical assumptions.

In neuropsychology's next evolutionary stage, "brain damage" was still treated as a unitary phenomenon but was given measurable extension. The theoretical basis for this position had been provided by Karl Lashley in his *Law of Mass Action* and *Principle of Equipotentiality* (1929). Lashley knew that even in rats certain functions, such as visual discrimination, were predictably compromised by lesions involving well-defined cortical areas of the brain. However, his experiments with rats led him to conclude that by and large the effectiveness of an animal's behavior and the extent to which its cortex was intact were directly correlated regardless of the site of damage, and that the contributions of different parts of the cortex were interchangeable.

In their once popular paper, L.F. Chapman and Wolff (1959) reviewed the literature on localization of function, presented data on their patients, and concluded, with Lashley, that sheer extent of cortical loss played a greater role in determining the amount of cognitive impairment than did the site of the lesion. "Brain damage" (or "organicity" or "organic impairment"—the terms varied from author to author but the meaning was essentially the same) took on a one-dimensionality and lack of specificity similar to that of the concept "sick." Neither "brain damage" nor "sickness" has etiological implications; neither implies the presence or absence of any particular symptoms or signs, nor can predictions or prescriptions be made on the basis of either term. Still, "brain damage" as a unitary but measurable condition remains a vigorous concept, reflected in the many test and battery indices, ratios, and quotients that purport to represent some quantity or relative degree of neurobehavioral impairment.

Advances in diagnostic medicine, with the exception of certain cases with mild or questionable cognitive impairment, have changed the typical referral question to the neuropsychologist from one that attempts to determine if the patient has neurologic disease or not. In most cases, the presence of "brain damage" has been clinically established and often verified radiologically. However, the *behavioral* repercussions of brain damage vary with the nature, extent, location, and duration of the lesion; with the age, sex, physical condition, and psychosocial background and status of the patient; and with individual neuroanatomical and physiological differences (see Chapters 3, 7, and 8). Not only does the pattern of neuropsychological deficits differ with different lesion characteristics and locations, but two persons with similar pathology and lesion sites may have distinctly different neuropsychological profiles (DeBleser, 1988; Howard, 1997; Luria, 1970). In contrast, patients with damage at different sites may pre-

sent similar deficits (Naeser, Palumbo, et al., 1989). Thus, although “brain damage” may be useful as an organizing concept for a broad range of behavioral disorders, when dealing with individual patients the concept of brain damage only becomes meaningful in terms of specific behavioral dysfunctions and their implications regarding underlying brain pathology.

CONCERNING TERMINOLOGY

The experience of wading through the older neuropsychological literature shares some characteristics with exploring an archaeological dig into a long-inhabited site. Much as the archaeologist finds artifacts that are both similar and different, evolving and discarded, so a reader can find, scattered through the decades, descriptions of the various neuropsychological disorders in terms (usually names of syndromes or behavioral anomalies) no longer in use and forgotten by most, terms that have evolved from one meaning to another, and terms that have retained their identity and currency pretty much as when first coined. Moreover, not all earlier terms given to the same neuropsychological phenomena over the past ten decades have been supplanted or fallen into disuse so that even the relatively recent literature may contain two or more expressions for the same or similar observations (e.g., see p. 33 for current aphasia terms, pp. 26, 34–35 for varieties of “working memory”). This rich terminological heritage can be very confusing, as Newcombe and Ratcliffe (1989) point out in their discussion of the terminological problems attendant on the study of disorders of visuospatial analysis. (See also Lishman’s [1997] discussion of the terminological confusion surrounding “confusion,” for example, and other common terms that are variously used to refer to mental states, to well-defined diagnostic entities, or to specific instances of abnormal behavior.)

In this book we have made an effort to use only terms that are currently widely accepted. Some still popular but poorly defined terms have been replaced by simpler and more apt substitutes for some of the classical terminology. For example, in order to distinguish those constructional disorders that have been called “constructional apraxia” from the neuropsychologically meaningful concept of praxis, which “in the strict sense, refers to the motor integration employed used to execute complex learned movements” (Strub and Black, 2000). We follow Strub and Black’s lead by maintaining a terminological distinction between these functional classes. Thus we use “constructional defects” or “constructional impairment” to refer to these kinds of disorders; the term “apraxia” is reserved for the special class of dysfunctions characterized by a breakdown

in the direction or execution of complex motor acts. “Apraxia” has problems of its own as different investigators define and use such terms as “ideational apraxia,” “ideomotor apraxia,” and “ideokinetic apraxia” in confusingly different ways (compare, for example, Hécaen and Albert, 1978; Heilman and Rothi, 2003; Walsh and Darby, 1999; M. Williams, 1979). Kimura and Archibald (1974) suggested that the forms of apraxia that have been called “ideational, ideomotor, ideokinetic, and so on” do not relate to “behaviorally different phenomena, but [to] disturbances at different points in a hypothetical sequence of cognitive events involved in making a movement.” Rather than attempt to reconcile the many disparities in the use of these terms and their definitions, we call these disturbances simply “apraxias” (see also Hanna-Pladdy and Rothi, 2001).

DIMENSIONS OF BEHAVIOR

Behavior may be conceptualized in terms of three functional systems: (1) *cognition*, which is the information-handling aspect of behavior; (2) *emotionality*, which concerns feelings and motivation; and (3) *executive functions*, which have to do with how behavior is expressed. Components of each of these three sets of functions are as integral to every bit of behavior as are length and breadth and height to the shape of any object. Moreover, like the dimensions of space, each one can be conceptualized and treated separately. The early Greek philosophers were the first to conceive of a tripartite division of behavior, postulating that different principles of the “soul” governed the rational, appetitive, and animating aspects of behavior. Present-day research in the behavioral sciences tends to support the philosophers’ intuitive insights into how the totality of behavior is organized. These classical and scientifically meaningful functional systems lend themselves well to the practical observation, measurement, and description of behavior and constitute a framework for organizing behavioral data generally.

In neuropsychology, the cognitive functions have received more attention than the emotional and control systems. This is partly because the cognitive defects of brain injured patients can figure so prominently in their symptomatology; partly because they can be so readily conceptualized, measured, and correlated with neuroanatomically identifiable systems; and partly because the structured nature of most medical and psychological examinations does not provide much opportunity for subtle emotional and control deficits to become evident.

However, brain damage rarely affects just one of these systems. Rather, the disruptive effects of most

brain lesions, regardless of their size or location, usually involve all three systems (Lezak, 1994).

For example, Korsakoff's psychosis, a condition most commonly associated with severe chronic alcoholism, has typically been described only in terms of cognitive dysfunctions; e.g., "The characteristic feature of Korsakow's [sic] syndrome is a certain type of amnesia. The patient has a gross defect of memory for recent events so that he has no recollection of what has happened even half an hour previously. He is disoriented in space and time and he fills the gaps in his memory by confabulation, that is, by giving imaginary accounts of his activities" (Walton, 1994; see also Squire, 1987; Tranel and Damasio, 2002). Yet chronic Korsakoff patients also exhibit profound changes in affect and executive, or control, functions that may be more crippling and more representative of the psychological devastations of this disease than the memory impairments.

Patients with this condition tend to be emotionally flat, to lack the impulse to initiate activity, and, if given a goal requiring more than an immediate one- or two-step response, to be unable to organize, set into motion, and carry through a plan of action to reach it (Heindel, Salmon, and Butters, 1991). Everyday frustrations, sad events, or worrisome problems, when brought to their attention, will arouse a somewhat appropriate affective response, as will a pleasant happening or a treat; but the arousal is only transitory, subsiding with a change in topic or distraction such as someone entering the room. When not stimulated from outside or by physiological urges, these responsive, comprehending, often well-spoken and well-mannered patients sit quite comfortably doing nothing, not even attending to a TV or nearby conversation. When they have the urge to move, they walk about aimlessly. Even those who talk about wanting to visit a relative, for instance, or call a lawyer, make no effort to do so, although doors are unlocked and the public telephone is in full view.

The behavioral defects characteristic of many patients with right hemisphere damage also reflect the involvement of all three systems. It is well known that these patients are especially likely to show impairments in such cognitive activities as spatial organization, integration of visual and spatial stimuli, and comprehension and manipulation of percepts that do not readily lend themselves to verbal analysis. Right hemisphere damaged patients may also experience characteristic emotional dysfunctions such as an indifference reaction (ignoring, playing down, or being unaware of mental and physical disabilities and situational problems), uncalled-for optimism or even euphoria, inappropriate emotional responses and insensitivity to the feelings of others, and loss of the self-perspective needed for accurate self-criticism, appreciation of limitations, or making constructive changes in behavior or attitudes (Cummings and Mega, 2003; Cutting, 1990; Gainotti, 1972). Furthermore, despite strong, well-expressed motivations and demonstrated knowledgeability and capability, impairments in the capacity to plan and organize complex activities and thinking immobilize many of the same right hemisphere damaged patients who have difficulty performing visuospatial tasks (Brownell and Martino, 1998; Lezak, 1994).

Behavior problems may also become more acute and the symptom picture more complex as secondary reactions to the specific problems created by the brain injury further involve each system. Additional repercussions and reactions may then occur as the patient attempts to cope with succeeding sets of reactions and the problems they bring.

The following case of a man who sustained relatively minor brain injuries demonstrates some typical interactions between impairments in different psychological systems.

A middle-aged clerk, the father of teenaged children, incurred a left-sided head injury in a car accident and was unconscious for several days. When examined three months after the accident, his principal complaint was fatigue. His scores on cognitive tests were consistently *high average* (between the 75th and 90th percentiles). The only cognitive difficulty demonstrated in the psychological examination was a slight impairment of verbal fluency exhibited by a few word-use errors on a sentence-building task. This verbal fluency problem did not seem grave, but it had serious implications for the patient's adjustment.

Because he could no longer produce fluent speech automatically, the patient had to exercise constant vigilance and conscious effort to talk as well as he did. This effort was a continuous drain on his energy so that he fatigued easily. Verbal fluency tended to deteriorate when he grew tired, giving rise to a vicious cycle in which he put out more effort when he was tired, further sapping his energy at the times he needed it the most. He felt worn out and became discouraged, irritable, and depressed. Emotional control too was no longer as automatic or effective as before the accident, and it was poorest when he was tired. He "blew up" frequently with little provocation. His children did not hide their annoyance with their grouchy, sullen father, and his wife became protective and overly solicitous. The patient perceived his family's behavior as further proof of his inadequacy and hopelessness. His depression deepened, he became more self-conscious about his speech, and the fluency problem frequently worsened.

COGNITIVE FUNCTIONS

Cognitive abilities (and disabilities) are functional properties of the individual that are not directly observed but instead are *inferred* from . . . behavior. . . . All behavior (including neuropsychological test performances) is multiply determined: a patient's failure on a test of abstract reasoning may not be due to a specific impairment in conceptual thinking but to attention disorder, verbal disability, or inability to discriminate the stimuli of the test instead.

Abigail B. Sivan and Arthur L. Benton, 1999

The four major classes of cognitive functions have their analogues in the computer operations of input, storage,

processing (e.g., sorting, combining, relating data in various ways), and output. Thus, (1) *receptive functions* involve the abilities to select, acquire, classify, and integrate information; (2) *memory and learning* refer to information storage and retrieval; (3) *thinking* concerns the mental organization and reorganization of information; and (4) *expressive functions* are the means through which information is communicated or acted upon. Each functional class comprises many discrete activities—such as color recognition or immediate memory for spoken words. Although each function constitutes a distinct class of behaviors, normally they work in close, interdependent concert. Despite the seeming ease with which the classes of cognitive functions can be distinguished conceptually, more than merely interdependent, they are inextricably bound together—different facets of the same activity. A.R. Damasio, H. Damasio, and Tranel (1990) describe the memory (information storage and retrieval) components of visual recognition. They also call attention to the role that thinking (concept formation) plays in the seemingly simple act of identifying a visual stimulus by name. Both practical applications and theory-making benefit from our ability to differentiate these various components of behavior.

Generally speaking, within each class of cognitive functions a division may be made between those functions that mediate verbal/symbolic information and those that deal with data that cannot be communicated in words or symbols, such as complex visual or sound patterns. These subclasses of functions differ from one another in their neuroanatomical organization and in their behavioral expression while sharing other basic neuroanatomical and psychometric relationships within the functional system.

The identification of discrete functions within each class of cognitive functions varies with the perspective and techniques of the investigator. Examiners using simple tests that elicit discrete responses can study highly specific functions. Multidimensional tests call for complex responses and thus measure broader and more complex functions. Verbal functions enter into verbal test responses. Motor functions are demonstrated on tests involving motor behavior. When practical considerations of time and equipment limit the functions that can be studied or when relevant tests are not administered, the examiner may remain ignorant of the untested functions or how their impairment contributes to a patient's deficits (Finger et al., 1988; Teuber, 1969). Although different investigators may identify or define some of the narrower subclasses of functions differently, they agree on the major functional systems and the large subdivisions.

However, these functional divisions are, to some extent, conceptual constructions that help the clinician

understand what goes into the typically very complex behaviors and test responses of their brain damaged patients. Discrete functions described here and in Chapter 3 rarely occur in isolation; but rather contribute to the much more commonly seen larger patterns of dysfunction due to damage to a “continuous, graded functional[ly]” organized cerebral cortex (E. Goldberg, 1995).

Academic psychology studies attentional functions within the framework of cognitive psychology. However, attentional functions differ from the functional groups listed above in that they underlie and, in a sense, maintain the activity of the cognitive functions. To carry the computer analogy a step further, attentional functions serve somewhat as command operations, calling into play one or more cognitive functions. For this reason, they are classified as *mental activity variables* here (see pp. 33–35).

Neuropsychology and the Concept of Intelligence: Brain Function Is Too Complex To Be Communicated in a Single Score

General intelligence is as valid as the “strength of soil” concept is for plant growers. It is not wrong but archaic.

J.P. Das, 1989

Cognitive activity was originally attributed to a single function, *intelligence*. Early investigators treated the concept of intelligence as if it were a unitary variable which, like physical strength, increased at a regular rate in the course of normal childhood development (Binet and Simon, 1908; Terman, 1916) and decreased with the amount of brain tissue lost through accident or disease (L.F. Chapman and Wolff, 1959; Lashley, 1938). As refinements in testing and data-handling techniques have afforded greater precision and control over observations of cognitive activity, it has become evident that the behavior measured by “intelligence” tests involves specific cognitive and executive functions (Ardila, 1999a; Feinberg and Farah, 2003a; Frackowiak, Friston, Frith, 1997; Rains, 2002; see also Chapter 3).

Neuropsychological research has contributed significantly to the redefinition of the nature of “intelligence” (Gazzaniga, 2000, *passim*; Kolb and Wishaw, 1996; Mesulam, 2000b). One of neuropsychology's earliest findings was that the summation scores (i.e., “intelligence quotient” [“IQ”] scores) on standard intelligence tests do not bear a predictably direct relationship to the size of brain lesions (Hebb, 1942; Maher, 1963). When a discrete brain lesion produces deficits involving a broad range of cognitive functions, these functions may

be affected in different ways. Abilities most directly served by the damaged tissue may be destroyed; associated or dependent abilities may be depressed or distorted, while some others may appear to be heightened or enhanced (e.g., see p. xx).

Differences in the vulnerability of specific mental abilities also characterize the effects of deteriorating brain disease (e.g., Filley, 2001; Mesulam, 2000a; Parks, Zec, and Wilson, 1993; see Chapter 7, *passim*). Not only are some functions disrupted in the early stages while others may remain relatively intact for years, but the affected functions also deteriorate at different rates. Differential deterioration of diverse psychological functions also occurs in aging (see pp. 296–300). Moreover, citing the lateral frontal lobes as the seat of “intelligence” because they are involved in various abstraction and conceptualizing tasks (J. Duncan et al., 2000) does not identify a cerebral locus for “intelligence” but rather illustrates one of the many different definitions of intelligence and is a nice example of circular reasoning. In sum, neuropsychological studies have not found a general cognitive or intellectual function, but rather many discrete ones that work together so smoothly in the intact brain that cognition is experienced as a single, seamless attribute.

From a neuropsychological perspective, Piercy (1964) defined intelligence as a “tendency for cerebral regions subserving different intellectual functions to be proportionately developed in any one individual. According to this notion, people with good verbal ability will tend also to have good non-verbal ability, in much the same way as people with big hands tend to have big feet” (p. 341). The performance of most adults on cognitive ability tests reflects both this tendency for test scores generally to converge around the same level and for some test scores to vary in differing degrees from the central tendency (Carroll, 1993; J.D. Matarazzo and Prifitera, 1989; Neisser et al., 1996). J.R. Flynn (1987, 1999), finding significant increases in large group “IQ” scores from generation to generation (the “Flynn effect”), concluded that these increases reflected changing environmental factors, presumably the same factors that contribute to intraindividual score convergence.

In cognitively intact adults, specialization of interests and activities and singular experiences contribute to intraindividual differences (Halpern, 1997). Socialization experiences, personal expectations, educational limitations, emotional disturbance, physical illness or handicaps, and brain dysfunction tend to magnify intraindividual test differences to significant proportions (e.g., see A.S. Kaufman, McLean, and Reynolds, 1988; Razani et al., 2001; Suzuki and Valencia, 1997).

Thus, the unitary concept of intelligence has only limited application in neuropsychological assessment. The

concept of intelligence may seem to justify the practice of using the level of a cognitively impaired patient’s best educational or vocational achievement or performance on an “intelligence” test as a best indicator of premorbid functioning—against which to compare current activities, observations, and test performances (see Chapter 4). However, psychologists have defined the concept of intelligence in so many different ways as to have lost what central meaning it originally had in philosophical discourse (Ardila, 1999a; Garcia, 1981). “Cognitive abilities” or “mental abilities” are the terms we will use when referring to those psychological functions dedicated to information reception, processing, and expression, and to executive functions—the abilities necessary for metacognitive control and direction of mental experience.

“IQ” and other summation or composite scores

The term IQ is bound to the myths that intelligence is unitary, fixed, and predetermined. . . . As long as the term IQ is used, these myths will complicate efforts to communicate the meaning of test results and classification decisions.

D.J. Reschly, 1981

“IQ” refers to a derived score used in many test batteries designed to measure a hypothesized general ability, intelligence. Because of the multiplicity of cognitive functions assessed in these batteries, IQ scores are not useful in describing cognitive test performances. IQ scores obtained from such tests represent a composite of performances on different kinds of items, on different items in the same tests when administered at different levels of difficulty, on different items in different editions of test batteries bearing the same name, or on different batteries contributing different kinds of items (Anastasi and Urbina, 1997; M.H. Daniel, 1997). If nothing else, the variability in sources from which the scores are derived should lead to serious questioning of their meaningfulness. Such composite scores are often good predictors of academic performance, which is not surprising given their heavy loading of school-type and culturally familiar items. Yet they represent so many different kinds of more or less confounded functions as to be conceptually meaningless (Lezak, 1988b).

In neuropsychological assessment in particular, IQ scores are often unreliable indices of neuropathic deterioration. Specific defects restricted to certain test modalities, for example, may give a totally erroneous impression of significant intellectual impairment when actually many cognitive functions may be relatively intact and lower total scores are a reflection of impairment of specific functional modalities. Conversely, IQs

may obscure selective defects in specific tests (A. Smith, 1966, p. 56). Leatham (1999) illustrates this point with the case of a postencephalitic man who "could not learn anything new" but achieved an IQ score of 128.

In fact, any derived score based on a combination of scores from two or more measures of different abilities results in loss of data. Should the levels of performance for the combined measures differ, the composite score—which will be somewhere between the highest and the lowest of the combined measures—will be misleading (Lezak, 2002). Averaged scores on a Wechsler Intelligence Scale battery provide just about as much information as do averaged scores on a school report card. Students with a four-point average can only have had an A in each subject; those with a zero grade point average obviously failed all subjects. Excluding these extremes, it is impossible to predict a student's performance in any one subject. In the same way, it is impossible to predict specific disabilities and areas of competency or dysfunction from averaged ability test scores (e.g., "IQ" scores). Thus composite scores of any kind have no place in neuropsychological assessment.

"IQ" may also stand for the concept of intelligence; e.g., in statements like "IQ is a product of genetic and environmental factors." It may refer to the idea of an inborn quantity of mental ability residing within each person and demonstrable through appropriate testing; e.g., "Harry is a good student, he must have a high IQ" (Lezak, 1988b). Moreover, interpretations of IQ scores in terms of what practical meaning they might have can vary widely, even among professionals, such as high school teachers and psychiatrists, whose training should have provided a common understanding of these scores (L. Wright, 1970).

S.E. Folstein (1989) called attention to how, in the United States, current use of IQ scores contributes further misery to already tragic situations. She explained that many patients with Huntington's disease whose mental abilities have deteriorated beyond the point that they can continue working will still perform sufficiently well on enough of the tests in Wechsler Intelligence Scale batteries to achieve an IQ score above 70, the number selected by the Social Security Disability Insurance (SSDI) agency as separating those able to work from those too mentally impaired for competitive employment. Thus, SSDI may refuse benefits to cognitively disabled persons simply on the grounds that their IQ score is too high, even when appropriate assessment reveals a pattern of disparate levels of functioning that preclude the patient from earning a living. Similar problems affect severely injured *traumatic brain injury* (TBI) patients who have been refused benefits because their summed test score is too high, even though they lack the judgment, social graces, self-control, mental flexi-

bility, memory and attentional abilities, and stamina to hold down even a routine kind of job.

One must never misconstrue a normal intelligence test result as an indication of normal intellectual status after head trauma, or worse, as indicative of a normal brain; *to do so would be to commit the cardinal sin of confusing absence of evidence with evidence of absence* [italics, mdl]. (Teuber, 1969)

In sum, "IQ" as a score is inherently meaningless and not infrequently misleading as well. "IQ"—whether concept, score, or catchword—has outlived whatever usefulness it may once have had and should be discarded.

Unfortunately, other summary scores also obscure actual test performances. Probably the most venerable of these summary scores is the Digit Span score which, in the Wechsler batteries (Wechsler Intelligence Scales for Adults [p. 649]; Wechsler Memory Scales [p. 481]), includes not only both forward and reversed span scores—each examining different aspects of short-term auditory memory and attention (see pp. 351–352)—but further confounds the data by adding a reliability measure (i.e., two trials at each span length).

Combined scores such as the "Index Scores" in Wechsler batteries may also obscure important information obtainable only by examining discrete scores. The Wechsler Adult Intelligence Scale-III (WAIS-III) features several Index Scores which are compilations of scores of individual tests that load on the same factor *in large-scale population* studies (The Psychological Corporation, 1997). However, in the individual case, large differences between the discrete test scores can illuminate core problems which would be submerged by the summation Index Score (see p. 653).

CLASSES OF COGNITIVE FUNCTIONS

As more is learned about how the brain processes information, it becomes more difficult to make theoretically acceptable distinctions between the different functions involved in human information processing. In the laboratory, precise discriminations between sensation and perception may depend upon whether incoming information is processed by analysis of superficial physical and sensory characteristics or through pattern recognition and meaningful associations. The fluidity of theoretical models of perception and memory in particular becomes apparent in the admission of A.R. Damasio, Tranel, and Damasio (1989) that "We have no way of distinguishing what might be conceived of as the higher echelons of perception from the lower echelons of recognition. . . . [T]here is no definable point of demarcation between perception and recognition" (p. 317).

Further, studies of perception without awareness, such as *blindsight* (Weiskrantz, 1986) or covert face recognition in *prosopagnosia* (defective face recognition) (Farah, 2001; J.E. McNeil and Warrington, 1993) indicate how perception and awareness each depend upon intercellular networks in which information is transferred in both parallel and serial processing networks (Farah, O'Reilly, and Vecera, 1993; Fuster, 2003; Pashler, 1998).

Efforts to conceptualize memory functions come up against this same kind of theoretical problem. "Memory research in cognitive neuroscience has literally exploded within the past few years, leaving clinical neurologists and psychologists with a complex array of fragmented perspectives on memory, a variety of subdivisions of mnemonic capacities, and a bundle of rivaling or just redundant 'models' and 'network' hypotheses" (Helmstaedter and Kurthen, 2001).

Rather than entering theoretical battlegrounds on ticklish issues that are not material to most practical applications in neuropsychology, we shall discuss these functions within a conceptual framework that has proven useful in psychological assessment generally and in neuropsychological assessment particularly.

Receptive Functions

Entry of information into the central processing system proceeds from sensory stimulation, i.e., *sensation*, through *perception*, which involves the integration of sensory impressions into psychologically meaningful data, and thence into memory. Thus, light on the retina creates a visual *sensation*; *perception* involves encoding the impulses transmitted by the aroused retina into a pattern of hues, shades, and intensities recognized as a daffodil in bloom.

Neuroscientists have discovered that the components of sensation can be splintered into ever smaller receptive units. The Nobel Prize winning research of Hubel and Weisel (1962, 1968) demonstrated that neurons in the visual cortex are arranged in columns that respond preferentially to stimuli at specific locations and at specific orientations. How discrete these subsystems may be is shown by the report of A.R. Damasio, Damasio, and Tranel (1990) that, "when fragments of a face are presented in isolation, for example, eyes or mouth, different neurons respond to different fragments."

Sensory reception

Sensory reception involves an arousal process that triggers central registration leading to analysis, encoding, and integrative activities. The organism receives sensation passively, shutting it out only, for instance, by

holding the nose to avoid a stench. Even in soundest slumber, a stomach ache or a loud noise will rouse the sleeper. However, sensations are rarely experienced in themselves, and perceptions depend greatly on attentional factors (Meador, Allison, et al., 2002; Meador, Ray et al., 2001). Most sensory data enter neurobehavioral systems as perceptions already endowed with previously learned meanings (Forde and Humphreys, 1999; Goodale, 2000; Shiffrin and Schneider, 1977). Neuropsychological assessment and research focus primarily on the five traditionally recognized senses: sight, hearing, touch, taste, and smell. Berthoz (2000) calls attention to other senses such as movement, space, balance, and effort.

Perception and the agnosias

Perception involves active processing of the continuous torrent of sensations as well as their inhibition or filtering from consciousness. This processing comprises many successive and interactive stages. Those that deal with the simplest physical or sensory characteristics, such as color, shape, or tone, come first in the processing sequence and serve as foundations for the more complex, "higher" levels of semantic and visuoconceptual processing that integrate sensory stimuli with one another at each moment, successively, and with the organism's past experience (Fuster, 2003; A. Martin, Ungerleider, and Haxby, 2000; Rapp, 2001, *passim*).

Normal perception in the healthy organism is a complex process engaging many different aspects of brain functioning (Coslett and Saffran, 1992; Goodale, 2000; Löwel and Singer, 2002). Like other cognitive functions, the extensive cortical distribution and complexity of perceptual activities make them highly vulnerable to brain injury. Perceptual defects resulting from brain injury can occur through loss of a primary sensory input such as vision or smell and also through impairment of specific integrative processes. Although it may be difficult to separate the sensory from the perceptual components of a behavioral defect in some severely brain injured patients, sensation and perception each has its own functional integrity. This can be seen clearly when perceptual organization is maintained despite very severe sensory defects or when perceptual functions are markedly disrupted in patients with little or no sensory deficit. The nearly deaf person can readily understand speech patterns when the sound is sufficiently amplified, whereas some brain damaged persons with keen auditory acuity cannot make sense out of what they hear.

The perceptual functions include such activities as awareness, recognition, discrimination, patterning, and orientation. Impairments in perceptual integration ap-

pear as disorders of recognition, the *agnosias* (literally, no knowledge). Teuber (1968) clarified the distinction between sensory and perceptual defects by defining agnosia as “a normal percept stripped of its meanings.” Moreover, “True agnosia . . . relates to the whole perceptual field, whether right or left,” in contrast to unilateral imperception phenomena where the patient is unaware of sensations or events on only one side (see pp. 66–67). Since a disturbance in any one perceptual activity may affect any of the sensory modalities as well as different aspects of each one, a catalogue of discrete perceptual disturbances can be quite lengthy. Benson (1989) listed six different kinds of visual agnosias. Bauer and Demery (1993) identified three distinctive auditory agnosias, and M. Williams (1979) described another three involving various aspects of body awareness.

This list could be expanded, for within most of these categories of perceptual defect there are functionally discrete subcategories; e.g., Heilman and Valenstein (2003) list 25 in the index, and the *INS Dictionary of Neuropsychology* (Loring, 1999) defines 14. For instance, loss of the ability to recognize faces (*prosopagnosia* or *face agnosia*), one of the visual agnosias, may be manifested in at least two different forms: inability to recognize familiar faces and inability to recognize unfamiliar faces, which usually do not occur together (Benton, 1980; De Haan, 2001; Warrington and James, 1967b). Moreover, prosopagnosia can occur with or without intact abilities to recognize associated characteristics such as a person’s facial expression, age, and sex (Tranel, Damasio, and Damasio, 1988) and thus lends itself to subcategories. A.R. Damasio (1990) suggested that the highly discrete dissociations that can occur within the visual modality (e.g., inability to recognize a person’s face with intact recognition for the same person’s gait) or between categories presented visually (e.g., man-made tools vs. natural objects; printed words vs. multidigit numbers) reflect the processing characteristics of the neural systems that form the substrates of knowledge (e.g., Riddoch and Humphreys, 2001). The fine degree to which brain organization is specialized becomes apparent in patients with similarly placed lesions who can identify inanimate objects but not animate ones, or comprehend words that are abstract better than those that are concrete (Warrington and Shallice, 1984).

Rather than offering a list of the many different forms that agnosias can take, E. Goldberg (1990) organized the various agnosias into two major categories: *associative agnosias* arise from a breakdown in one or more aspects of the patient’s information store or “generic” knowledge and *apperceptive agnosias* are due to higher level perceptual disturbances. The specific content of an

agnosic disorder depends on individual variations in the specific functions involved in a lesion site.

Memory

If any one faculty of our nature may be called more wonderful than the rest, I do think it is memory. There seems something more speakingly incomprehensible in the powers, the failures, the inequalities of memory, than in any other of our intelligences. The memory is sometimes so retentive, so serviceable, so obedient—at others, so bewildered and so weak—and at others again, so tyrannic, so beyond control!—We are to be sure a miracle every way—but our powers of recollecting and forgetting, do seem peculiarly past finding out.

Jane Austen, Mansfield Park, 1814 (1961)

Memory is a cortical network, an array of connective links formed by experience between neurons of the neocortex . . . the function of cortical neurons in memory derives exclusively from their being part of such networks.

Joaquin M. Fuster, 1995

Central to all cognitive functions and probably to all that is characteristically human in a person’s behavior is the capacity for memory, learning, and intentional access to this knowledge store. Memory frees the individual from dependency on physiological urges or situational happenstance for pleasure seeking; dread and despair do not occur in a memory vacuum. Severely impaired memory isolates patients from emotionally or practically meaningful contact with the world about them and deprives them of a sense of personal continuity, rendering them passive and helplessly dependent. Mildly to moderately impaired memory has a disorienting effect.

How many memory systems?

Perhaps the most important recent contribution to the evolution of our understanding of memory has been the demonstration that, with other mammals, we have a number of distinctly different systems that serve our memories (Squire and Knowlton, 2000). Intimations of a dual nature of memory have cropped up in the literature since the 1960s, when B. Milner (1962, 1965) and Corkin (1968) demonstrated that the now famous patient, H.M., could learn and retain some new skills despite profound amnesia (literally, no memory). Ever since surgery for epilepsy had unexpectedly left him with no hippocampus (paired structures necessary for learning about objects, ideas, and the course of one’s life), H.M. has had memory deficits that severely compromise access to previously learned information as well as a complete inability to learn new information

or recall ongoing events. The possibility of more memory systems, each with its own relatively discrete neurotransmitters or neuroanatomic underpinnings, may also be entertained (Mayes, 2000a; Schacter, Wagner, and Buckner, 2000).

A functional duality of memory systems, so vividly demonstrated in amnesic patients and patients with degenerative disorders (e.g., see N. Butters and Stuss, 1989) has provided the basis for conceptualizing memory functions in terms of two long-term storage and retrieval systems: a *declarative* system, or *explicit memory* which deals with facts and events and is available to consciousness, and a *nondeclarative* or *implicit* system which is “nonconscious” (Squire and Knowlton, 2000). However, depending on one’s perspective, the count of systems or kinds of memory varies. In a clinical perspective, Mayes (2000a) divides declarative memory into *semantic* (fact memory) and *episodic* (autobiographic memory), and nondeclarative memory into *item-specific implicit memory* (ISIM) and *procedural*—also implicit—memory (see also Baddeley, 2002). This classification gives four long-term storage systems plus one system for *short-term* (what Mayes calls *working*) memory which is supported by neuroimaging studies (Schacter, Wagner, and Buckner, 2000). As of July 25, 2002, Tulving (2002b) had counted “134 different named types of memory.”

While the dual system classification remains at the core of most other ways of conceptualizing memory systems and subsystems, some variations have been offered. For example, in listing long-term storage systems by their neuroanatomic sites, “thought to be especially important for each form of declarative and nondeclarative memory,” Squire and Zola (1996) raise the count of systems to six by adding *classical conditioning* (two systems, one for emotional and one for skeletal responses) and *reflex learning*, while merging semantic and episodic memory into one declarative system and not including short-term memory in their count. However, for clinical purposes, the dual system conceptualization—into declarative (explicit) and nondeclarative (implicit) memory with its major subsystems—provides a useful framework for observing and understanding patterns of memory competence and deficits presented by our patients. The discussion of memory here generally follows the dual system framework of Baddeley (2002) and Mayes (2000a).

Declarative (explicit) memory

Most memory research and theory has focused on abilities to learn about and remember information, objects, and events. This is the kind of memory that patients refer to when complaining of memory problems, that

teachers address for most educational activities, that is the “memory” of common parlance. It has been described as “the mental capacity of retaining and reviving impressions, or of recalling or recognizing previous experiences . . . act or fact of retaining mental impressions” (J. Stein, 1966) and, as such, always involves awareness (Moscovitch, 2000). Referring to it as “explicit memory,” Demitrack and his colleagues (1992) point out that declarative memory involves “a conscious and intentional recollection” process.

Stages of memory processing

Despite the plethora of theories about stages (R.C. Atkinson and Shiffrin, 1968; G.H. Bower, 2000; R.F. Thompson, 1988) or processing levels (S.C. Brown and Craik, 2000; Craik, 1979), for clinical purposes a three-stage or elaborated two-stage model of declarative memory provides a suitable framework for conceptualizing and understanding dysfunctional memory (Balota et al., 2000; McGaugh, 1966; Parkin, 2001). Moreover, clinically, three kinds of memory are distinguishable. Two are succeeding stages of *short-term storage* (see also Baddeley, 2002; Loring, 1999; R.C. Petersen and Weingartner, 1991, for discussions of memory terminology). Recent research has questioned whether short-term storage is a distinct memory function as considerable evidence indicates that it is essentially one aspect of the consolidation process (Fuster, 2003; Parkin, 2001). However for clinical purposes it remains a useful concept (e.g., Baddeley, 2002).

1. *Registration*, or *sensory memory* holds large amounts of incoming information briefly (on the order of seconds) in *sensory store* (Balota et al., 2000; Valler and Papagno, 2002). It is neither strictly a memory function nor a perceptual function but rather a selecting and recording process by which perceptions enter the memory system. Registration involves the programming of acquired sensory response patterns (perceptual tendencies) in the recording and memorizing center of the brain (Nauta, 1964). The first traces of a stimulus may be experienced as a fleeting visual image (*iconic memory*, lasting up to 200 msec) or auditory “replay” (*echoic memory*, lasting up to 2,000 msec), indicating early stage processing in terms of sensory modality (Fuster, 1995; Koch and Crick, 2000). The affective, set (perceptual and response predisposition), and attention-focusing components of perception play an integral role in the registration process (Brain, 1969; S.C. Brown and Craik, 2000; Markowitsch, 2000). Either information being registered is further processed as short-term memory or it quickly decays.

2a. *Immediate memory*, the first stage of *short-term*

memory (STM) storage, temporarily holds information retained from the registration process. While theoretically distinguishable from attention, in practice, short-term memory may be equated with simple immediate span of attention (Baddeley, 2000; Howieson and Lezak, 2002; see pp. 34, 350–351). Immediate memory represents neuronal activation in which the relevant perceptual components have been integrated (Doty, 1979; Mishkin and Appenzeller, 1987). It serves “as a limited capacity store from which information is transferred to a more permanent store” and also “as a limited capacity retrieval system” (Fuster, 1995; see also Squire, 1986). Having shown that immediate memory normally handles about seven bits of information at a time, give or take two, G.A. Miller (1956) observed that this restricted holding capacity of “immediate memory impose[s] severe limitations on the amount of information that we are able to perceive, process, and remember.” Immediate memory is of sufficient duration to enable a person to respond to ongoing events when more enduring forms of memory have been lost (Talland, 1965a; Victor et al., 1971). It typically lasts from about 30 seconds up to several minutes.

Although immediate memory is usually conceptualized as a unitary process, Baddeley (1986, 2002) shows how it may operate as a set of subsystems “controlled by a limited capacity executive system,” which together is *working memory*. It is hypothesized that working memory consists of two subsystems, one for processing language—the “phonological loop”—and one for visuospatial data—the “visuospatial sketch pad” (see also Vallar and Papagno, 2002). The functions of working memory are “to hold information in mind, to internalize information, and to use that information to guide behavior without the aid of or in the absence of reliable external cues” (Goldman-Rakic, 1993, p. 15; see also Andrade, 2001; Fuster, 2003).

R.D. Morris and Baddeley (1988) suggested that a *primary memory* component of short-term storage can be differentiated from working memory in that the former is highly attention dependent, dissipating rapidly with distraction. Early stage Alzheimer patients and patients with frontal lobe lesions, for example, may demonstrate a relatively intact working memory but a very fragile primary memory.

Numerous studies have supported Hebb’s (1949) insightful hunch that information in immediate memory is temporarily maintained in *reverberating neural circuits* (self-contained neural networks that sustain a nerve impulse by channeling it repeatedly through the same network) (Dudai, 1989; Fuster, 1995; McGaugh et al., 1990, *passim*; Rosenzweig and Leiman, 1968; Shepherd, 1998). It appears that, if not converted into a more stable biochemical organization for longer last-

ing storage, the electrochemical activity that constitutes the immediate memory trace spontaneously dissipates and the memory is not retained. For example, only the rare reader with a “photographic” memory will be able to recall verbatim the first sentence on the preceding page although almost everyone who has read this far will have just seen it.

2b. *Rehearsal* is any repetitive mental process that serves to lengthen the duration of a memory trace (S.C. Brown and Craik, 2000). With rehearsal, a memory trace may be maintained for hours. Rehearsal increases the likelihood that a given bit of information will be permanently stored but does not ensure it (Baddeley, 1986).

2c. Another kind of short-term memory may be distinguished from immediate memory in that it lasts from an hour or so to one or two days—longer than a reverberating circuit could be maintained by even the most conscientious rehearsal efforts, but not yet permanently fixed as learned material in long-term storage (Fuster, 1995; Rosenzweig and Leiman, 1968; Tranel and Damasio, 2002). This may be evidence of an intermediate step “in a continuous spectrum of interlocked molecular mechanisms of . . . the multistep, multichannel nature of memory” (Dudai, 1989).

3. *Long-term memory (LTM) or secondary memory*—i.e., *learning*, the acquisition of new information—refers to the organism’s ability to store information. Long-term memory is most readily distinguishable from short-term memory in amnesic patients, i.e., persons unable to retain new information for more than a few minutes without continuing rehearsal. Although amnesic conditions may have very different etiologies (see Chapter 7, *passim*), they all have in common a relatively intact short-term memory capacity with significant long-term memory impairments (Baddeley and Warrington, 1970; O’Connor and Verfaellie, 2002; Parkin, 2001).

The process of storing information as long-term memory, *consolidation*, may occur quickly or continue for considerable lengths of time without requiring active involvement (Lynch, 2000; Mayes, 1988; Squire, 1987). *Learning* implies consolidation—what is learned is consolidated. “Consolidation best refers to a hypothesized process of reorganization within representations of stored information, which continues as long as information is being forgotten” (Squire, 1986, p. 241). Memory acquisition and retention result from the interaction of multiple networks distributed through time and space. Many theories of memory consolidation propose a gradual transfer of memory that requires processing from hippocampal and medial temporal lobe structures to the neocortex for longer term storage (Kapur and Brooks, 1999).

“Learning” often implies effortful or attentive activity on the part of the learner. Yet when the declarative memory system is intact, much information is also acquired without directed effort, by means of *incidental learning* (Dudai, 1989; Kimball and Holyoak, 2000). Incidental learning tends to be susceptible to impairment with some kinds of brain damage (S. Cooper, 1982; C. Ryan, Butters, Montgomery, et al., 1980). Much of the information in the long-term storage system appears to be organized on the basis of meaning and associations, whereas in the short-term storage system it is organized in terms of contiguity or of sensory properties such as similar sounds, shapes, or colors (G.H. Bower, 2000; Craik and Lockhart, 1972). However, Baddeley (1978) observed that rote repetition and association built on superficial, relatively meaningless stimulus characteristics can lead to learning too.

Long-term memory storage involves a number of processes occurring at the cellular level. These include neurochemical alterations in the *neuron* (nerve cell), neurochemical alterations of the *synapse* (the point of interaction between nerve cell endings) that may account for differences in the amount of neurotransmitter released or taken up at the synaptic juncture, elaboration of the dendritic (branching out) structures of the neuron to increase the number of contacts made with other cells (Fuster, 1995; D. Johnston and Amaral, 1998; Levitan and Kaczmarek, 2002; Löwel and Singer, 2002; Lynch, 2000), and perhaps *pruning* or *apoptosis* (programmed cell death) of some connections with disuse (Edelman, 1989; Huttenlocher, 2002). There is no single local site for stored memories; instead, memories involve neuronal contributions from many cortical and subcortical centers (Fuster, 1995; Markowitsch, 2000; Penfield, 1968; Thatcher and John, 1977), with “different brain systems playing different roles in the memory system” (R.F. Thompson, 1976). Encoding, storage, and retrieval of information in the memory system appear to take place according to both principles of association (Levitan and Kaczmarek, 2002; McClelland, 2000) and “characteristics that are unique to a particular stimulus” (S.C. Brown and Craik, 2000, p. 98). Breakdown in the capacity to store or retrieve material results in distinctive memory disorders.

Recent and *remote* memory are clinical terms that refer, respectively, to *autobiographical memories* stored within the last few hours, days, weeks, or even months and to older memories dating from early childhood (e.g., Strub and Black, 2000; see also Neisser and Libby, 2000). In intact persons it is virtually impossible to determine where recent memory ends and remote memory begins, for there are no major discontinuities in memory from the present to early wisps of infantile rec-

ollection. Recent memory and remote memory become meaningful concepts when dealing with problems of *amnesia* (literally, no memory), periods for which there is no recall, in contrast to memory impairments which may involve specific deficits. Then remote memory becomes recall of information stored prior to the amnesic episode or state.

Amnesia

When registration or storage processes are impaired by disease or accident, acquisition of new information or recall of old may range from spotty to nonexistent (Kapur, 1988a; T.M. Lee et al., 2002; O'Connor and Verfaellie, 2002; Tulving, 2002a; Zola-Morgan, 2003). The nature of these deficits is largely determined by lesion site, as memory impairments can result from injuries to many different parts of the brain (Bogen, 1997). Temporary disruption of these processes, which often follows head injury or *electroconvulsive therapy* (ECT) for psychiatric conditions, obliterates memory for the period of impairment (Cahill and Frith, 1995; Y. Stern and Sackeim, 2002). Destruction of these capacities results in a permanent memory vacuum from the time of onset of the disorder.

The inability or impaired ability to remember one's life events beginning with the onset of a condition is called *anterograde amnesia*. Patients with anterograde amnesia are, for most practical purposes, unable to learn and have defective recent memory. The kind and severity of the memory defect vary somewhat with the nature of the disorder (Kopelman, Stanhope, and Kingsley, 1999; O'Connor and Verfaellie, 2002; Y. Stern and Sackeim, 2002).

Loss of memory for events preceding the onset of brain injury, most often due to trauma, is called *retrograde amnesia*. It tends to be relatively short (30 minutes or less) with TBI but can be extensive (E. Goldberg and Bilder, 1986). When retrograde amnesia occurs with brain disease, loss of one's own history and events may go back years and even decades (M.S. Albert, Butters, and Brandt, 1981; N. Butters and Cermak, 1986; Corkin, Hurt, et al., 1987) and typically follows a temporal gradient in which newer memories are more vulnerable to loss than older ones (M.S. Albert, Butters, and Levin, 1979; Beatty, Salmon, Butters, et al., 1988; Kapur, Millar, et al., 1998; Squire, Clark, and Knowlton, 2001). The dissociation of anterograde and retrograde memory problems in patients with memory disorders has shown that the anatomical structures involved in new learning and in retrieval of old memories are different (E. Goldberg, Antin, et al., 1981; Markowitsch, 2000). Hippocampal damage is implicated in the defective storage processes of antero-

grade amnesia (see pp. 50–51). The retrieval problems of retrograde amnesia have been associated with diencephalic lesions, most specifically with nuclei in the mammillary bodies and/or the thalamus (see p. 46) and interconnecting pathways (N. Butters and Stuss, 1989; Markowitsch, 2000; Y. Stern and Sackeim, 2002), and also with other subcortical structures and cortical regions (Nyberg and Cabeza, 2000).

Long-enduring retrograde amnesia that extends back for years or decades is usually accompanied by an equally prominent anterograde amnesia; these patients neither recall much of their history nor learn much that is new. For a dense retrograde amnesia to occur on an organic basis with learning ability remaining fully intact is relatively rare (E. Goldberg, Antin, et al., 1981; Kopelman, 1987a).

A 52-year-old machine maintenance man complained of “amnesia” a few days after his head was bumped in a minor traffic accident. He knew his name but denied memory for any personal history preceding the accident while registering and retaining postaccident events, names, and places normally. This burly, well-muscled fellow moved like a child, spoke in a soft—almost lisping—manner, and was only passively responsive in interview. He was watched over by his woman companion who described a complete personality change since the accident. She reported that he had been raised in a rural community in a southeastern state and had not completed high school. With these observations and this history, rather than begin a battery of tests, he was hypnotized.

Under hypnosis, a manly, pleasantly assertive, rather concrete-minded personality emerged. In the course of six hypnotherapy sessions the patient revealed that, as a prize fighter when young he had learned to consider his fists to be “lethal weapons.” Some years before the accident he had become very angry with a brother-in-law who picked a fight and was knocked down by the patient. Six days later this man died, apparently from a previously diagnosed heart condition; yet the patient became convinced that he had killed him and that his anger was potentially murderous. Just days before the traffic accident, the patient’s son informed him that he had fathered a baby while in service overseas but was not going to take responsibility for baby or mother. This enraged the patient who reined in his anger only with great effort. He was riding with his son when the accident occurred. A very momentary loss of consciousness when he bumped his head provided a rationale—amnesia—for a new, safely ineffectual personality to evolve, fully dissociated from the personality he feared could murder his son. Counseling under hypnosis and later in his normal state helped him to learn about and cope with his anger appropriately.

Aspects and elements of declarative memory

Recall vs. recognition. The effectiveness of the memory system also depends on how readily and completely information can be retrieved. Information retrieval is *remembering*, which may occur through *recall* involv-

ing an active, complex search process (S.C. Brown and Craik, 2000; Mayes, 1988). The question, “What is the capital of Oregon?” tests the recall function. When a like stimulus triggers awareness, remembering takes place through *recognition*. The question “Which of the following is the capital of Oregon: Albany, Portland, or Salem?” tests the recognition function. Retrieval by recognition is much easier than free recall for both intact and brain impaired persons (N. Butters, Wolfe, Granholm, and Martone, 1986; M.K. Johnson, 1990). On superficial examination, retrieval problems can be mistaken for learning or retention problems, but the nature of an apparent learning problem can be determined by appropriate testing techniques (H.S. Levin, 1986; see pp. 414–415).

Elements of declarative memory. The many different kinds of memory function become apparent in pathological conditions of the brain (Shimamura, 1989; Stuss and Levine, 2002; Van der Werf et al., 2000; Verfaellie and O’Connor, 2000). Besides the overriding distinctions between short-term and long-term memory, patients may display deficits that are specific to the nature of the information to be learned, i.e., *material specific*. Such deficits are specific to either verbal or non-verbalized information (Buckner, 2000; Jones-Gotman, 1991a; B. Milner, 1974), or to motor skill learning (Corkin, 1968; Mayes, 2000b), cutting across sensory modalities. Further, a similar distinction is made for *modality specific* memory, which depends on the specific sensory modality of testing, and is most often identified when examining working memory (Conant et al., 1999; Fastenau, Conant, and Lauer, 1998).

Brain disease affects different kinds of memories in long-term storage differentially so that a motor speech habit, such as organizing certain sounds into a word, may be wholly retained while rules for organizing words into meaningful speech are lost (H. Damasio and Damasio, 1989; Geschwind, 1970). Stored memories involving different sensory modalities, knowledge categories, and output mechanisms are also differentially affected by brain disease (Farah, Hammond, et al., 1989; K. Patterson and Hodges, 1995). For example, recognition of printed words or numbers may be severely impaired while speech comprehension and picture recognition remain relatively intact. Differences between what learned information is affected or not by brain disease may be so fine that access to one category of words is retained while words in a similar category are lost, e.g., proper names relating to specific people vs. proper names with a general referent (Warrington and McCarthy, 1987; see also A.R. Damasio, 1990; Warrington and Shallice, 1984), living vs. non-living things (E. Strauss, Semenza, et al., 2000), or

memory for landmarks vs. route recall (Schacter and Nadel, 1991). Thus, some very focal brain lesions reveal that large material-specific categories, such as semantic or spatial memory, break down into ever more discrete subsystems following the parallel fragmentation of perceptual processes into the same material-specific subsystems, and that the content categories of both memory and perception are differentially vulnerable to brain damage (Schacter, 1990a; Shelton and Caramazzo, 2001).

Another distinction can be made between *episodic* or *event* memory, also called *declarative* memory, and *semantic memory* (Mayes, 1988; Tulving, 1985, 2000; Wheeler, 2002). The former refers to memories of one's own experiences and is therefore unique and localizable in time and space. Semantic memory, i.e., what is learned as knowledge, is "timeless and spaceless," as, for instance, the alphabet or historical data unrelated to a person's life. The clinical meaningfulness of this distinction becomes evident in patients whose posttraumatic or postencephalitic retrograde amnesia may extend back weeks and even years, although their fund of information, language usage, and practical knowledge may be quite intact (Warrington and McCarthy, 1988).

Yet another distinction, between *automatic* and *effortful* memory, rests on whether learning involves active, effortful processing or passive acquisition (Balota, Dolan, and Duchek, 2000; Hasher and Zacks, 1979; M.K. Johnson and Hirst, 1991). Clinically, the difference between automatic and effortful memory commonly shows up in a relatively normal immediate recall of digits or letters that is characteristic of many brain disorders (e.g., head trauma, Alzheimer's dementia, multiple sclerosis), a recall that requires little processing in contrast to reduced performance on a task requiring effort, such as reciting a string of digits in reverse, a phenomenon that also appears with advanced age. That these are distinctive memory processes is shown by facilitation of the effortful task when the dopamine neurotransmitter system is stimulated with no corresponding improvement in the automatic memory task (R.P. Newman et al., 1984).

In selected patient groups, other kinds of memory that can be distinguished from the usual categories of declarative memory have been identified. *Source memory* (K.J. Mitchell and Johnson, 2000; Schacter, Harbluk, and McLachlan, 1984; Shimamura, 2002) or *contextual memory* (J.R. Anderson and Schooler, 2000; Parkin, 2001; Schacter, 1987) refers to knowledge of where or when something was learned, i.e., the contextual information surrounding the learning experience. Source memory may be a form of incidental memory.

Prospective memory is a recently distinguished capacity that involves both the "what" knowledge of de-

clarative memory and executive functioning. It is the ability "to remember to do something at a particular time" (Baddeley, Harris, et al., 1987; see Brandimonte et al., 1996, *passim*; Shimamura, Janowsky, and Squire, 1991). The importance of prospective memory becomes apparent in those patients with frontal lobe injuries whose memory abilities in the classical sense may be relatively intact but whose social dependency is due, at least in part, to their inability to remember to carry out previously decided upon activities at designated times or places (Sohlberg and Mateer, 2001; see p. 81). For example, it may not occur to them to keep appointments they have made, although when reminded or cued it becomes obvious that this information was not lost but rather was not recalled when needed.

Nondeclarative (implicit) memory

The knowledge and skills in nondeclarative memory have been defined as "knowledge that is expressed in performance without subjects' phenomenal awareness that they possess it" (Schacter, McAndrews, and Moscovitch, 1988). Two subsystems are clinically relevant: *procedural memory* and *priming* or *perceptual learning* (Baddeley, 2002; Mayes, 2000b; Squire and Knowlton, 2000). *Classical conditioning* is also considered a form of nondeclarative memory (Squire and Knowlton, 2000). Different aspects of implicit memory and learning activities are processed within neuroanatomically different systems (Fuster, 1995; Heindel, Salmon, et al., 1989; Squire and Knowlton, 2000; Tranel and Damasio, 2000).

Procedural, or *skill memory*, includes motor and cognitive skill learning and perceptual, "how to," learning; *priming* refers to a form of cued recall in which, without the subject's awareness, prior exposure facilitates the response; and classical conditioning (Mayes, 1988; Squire, 1987). Two elements common to these different aspects of memory are their preservation in most amnesic patients (Ewert et al., 1989; Martone, Butters, Payne, et al., 1984; O'Connor and Verfaillie, 2002) and that they are acquired or used without awareness or deliberate effort (Graf et al., 1984; Nissen and Bullemer, 1987).

Aspects of procedural memory have always been available through our observations of patients who remember nothing of ongoing events and little of their past history yet retain abilities to walk and talk, dress and eat, etc.; i.e., their well-ingrained habits that do not depend on conscious awareness remain intact (Fuster, 1995; Gabrieli, 1998; Mayes, 2000b). Mishkin and Petri (1984) considered procedural memory "a habit system." Moreover, procedural memory has been repeatedly demonstrated in intact subjects taught un-

usual skills, such as reading inverted type (Kolars, 1976) or learning the sequence for a set of changing locations (Willingham et al., 1989). Now that procedural memory has been not just identified but well-studied, it holds some promise for specific kinds of rehabilitative interventions for memory impaired patients (Donaghy and Williams, 1998; Farina et al., 2002; Glisky et al., 1986).

Forgetting

Some loss of or diminished access to information—both recently acquired and stored in the past—occurs continually as normal *forgetting*. Normal forgetting rates differ with such psychological variables as personal meaningfulness of the material and conceptual styles, as well as with age differences and probably some developmental differences. Normal forgetting differs from amnesic conditions in that only amnesia involves the inaccessibility or nonrecording of large chunks of personal memories.

What the process(es) of normal forgetting might be is still unclear. The Freudian view hypothesized that nothing is lost from memory and the problem lies in faulty or repressed retrieval processes. However, systematic research has shown that forgetting really happens and that what is forgotten is lost from memory through disuse or interference by more recently or vividly learned information or experiences (Mayes, 1988; Squire, 1987). Perhaps most important of these processes is “autonomous decay . . . due to physiologic and metabolic processes with progressive erosion of synaptic connections” (G.H. Bower, 2000). Fuster (1995) points out that initial “poor fixation of the memory” accounts for some instances of forgetting. This becomes most apparent in clinical conditions in which attentional processes are so impaired that passing stimuli (in conversation or as events) are barely attended to and weakly stored, if at all (Howieson and Lezak, 2002).

What seems likely is that normally both kinds of processes are operative: psychodynamic suppression or repression of some unwanted or unneeded memories takes place along with organic dissolution of others. Forgetting proceeds more rapidly with certain neurobehavioral conditions, e.g., Alzheimer’s disease (Dannenberg et al., 1988), amnesia (Isaac and Mayes, 1999), frontotemporal dementia (Pasquier et al., 2001), aging (Tombaugh and Hubley, 2001), and vascular dementia (Vanderploeg, Yuseph, and Schinka, 2001).

Thinking

Thinking may be defined as any mental operation that relates two or more bits of information explicitly (as

in making an arithmetic computation) or implicitly (as in judging that this is bad, i.e., relative to that) (Fuster, 2003). A host of complex cognitive functions is subsumed under the rubric of thinking, such as computation, reasoning and judgment, concept formation, abstracting and generalizing, ordering, organizing, planning, and problem solving (see Sohlberg and Mateer, 1989).

The nature of the information being mentally manipulated (e.g., numbers, design concepts, words) and the operation (e.g., comparing, compounding, abstracting, ordering) define the category of thinking. Thus, “verbal reasoning” comprises several operations done with words; it generally includes ordering and comparing, sometimes analyzing and synthesizing (e.g., Cosmides and Tooby, 2000). “Computation” may involve operations of ordering and compounding done with numbers (Dehaene, 2000; Fasotti, 1992), and distance judgment involves abstracting and comparing ideas of spatial extension.

The concept of “higher” and “lower” mental processes originated with the ancient Greek philosophers. This concept figures in the hierarchical theories of brain functions and mental ability factors in which “higher” refers to the more complex mental operations and “lower” to the simpler ones. Thinking is at the high end of this scale. The degree to which a concept is *abstract* or *concrete* also determines its place on the scale. For example, the abstract idea “a living organism” is presumed to represent a higher level of thinking than the more concrete idea “my cat Pansy;” the abstract rule “file specific topics under general topics” is likewise considered to be at a higher level of thinking than the instructions “file ‘fir’ under ‘conifer,’ file ‘conifer’ under ‘tree.’”

The higher cognitive functions of abstraction, reasoning, judgment, analysis, and synthesis tend to be relatively sensitive to diffuse brain injury, even when most specific receptive, expressive, or memory functions remain essentially intact (Knopman and Selnes, 2003; Mesulam, 2000a). They may also be disrupted by any one of a number of lesions in functionally discrete areas of the brain at lower levels of the hierarchy (Gitelman, 2002). Thus the higher cognitive functions tend to be more “fragile” than the lower, more discrete functions. Conversely, higher cognitive abilities may remain relatively unaffected in the presence of specific receptive, expressive, and memory dysfunctions (E. Goldberg, 2001; Pincus and Tucker, 2003; Teuber et al., 1951; Wepman, 1976; for case examples, see Ogden, 1996).

Problem solving can take place at any point along the complexity and abstraction continua. The simplest issues of daily living call upon it, such as inserting tooth

brushing into the morning routine or determining what to do when the soap dish is empty. Einstein did the same in his efforts to account for light distortions in the solar system. Problem solving involves executive functions (see pp. 35–37, and Chapter 16) as well as thinking, since a problem first has to be identified. Patients with executive disorders can look at an empty soap dish without recognizing that it presents a problem to be solved and yet be able to figure out what to do once the problem has been brought to their attention.

Unlike other cognitive functions, thinking is not tied to specific neuroanatomical systems, although the disruption of feedback, regulatory, and integrating mechanisms can affect complex cognitive activity more profoundly than other cognitive functions (Luria, 1966). “There is no . . . anatomy of the higher cerebral functions in the strict sense of the word. . . . Thinking is regarded as a function of the entire brain that defies localization” (Glöning and Hoff, 1969).

Arithmetic concepts and operations, however, are basic thinking tools that can be disrupted in quite specific ways by more or less localized lesions (Denburg and Tranel, 2003; Fasotti, 1992; Grafman and Rickard, 1997). Their vulnerability to different lesion loci has revealed at least three distinctive aspects to arithmetic activity; each, when impaired, gives rise to a specific kind of *acalculia* (literally, no counting) (E. Goldberg, 1990; Grafman, 1988; Spiers, 1987): (1) appreciation and knowledge of number concepts (*acalculias* associated with verbal defects); (2) ability to organize and manipulate numbers spatially as in long division or multiplication of two or more numbers (*spatial dyscalculia*); and (3) ability to perform arithmetic operations (*anarithmetria*). Neuroimaging studies have further fractionated components of number processes in showing associations of specific components with different cerebral regions (Dehaene, 2000; Gitelman, 2002).

As with other cognitive functions, the quality of any complex operation will depend in part on the extent to which its sensory and motor components are intact at the central integrative (cortical) level (E. Goldberg, 1990; Riddoch and Humphreys, 2001). For example, patients with certain somatosensory perceptual defects tend to do poorly on reasoning tasks involving visuospatial concepts (Farah, 2003a; Teuber, 1959); patients whose perceptual disabilities are associated with lesions in the visual system are more likely to have difficulty solving problems involving visual concepts (B. Milner, 1954; Tranel, 2002). Verbal defects tend to have more obvious and widespread cognitive consequences than defects in other functional systems because task instructions are frequently verbal, self-regulation and self-critiquing mechanisms are typically

verbal, and ideational systems—even for nonverbal material—are usually verbal (Luria, 1973a).

Expressive Functions

Expressive functions, such as speaking, drawing or writing, manipulating, physical gestures, facial expressions or movements, make up the sum of observable behavior. Mental activity is inferred from them.

Apraxia

Disturbances of purposeful expressive functions are known as *apraxias* (literally, no work) (Liepmann, [1900] 1988). The apraxias typically involve impairment of learned voluntary acts despite adequate motor innervation of capable muscles, adequate sensorimotor coordination for complex acts carried out without conscious intent (e.g., articulating isolated spontaneous words or phrases clearly when volitional speech is blocked, brushing crumbs or fiddling with objects when intentional hand movements cannot be performed), and comprehension of the elements and goals of the desired activity. Given the complexity of purposeful activity, it is not surprising that apraxia occurs with disruption of pathways at different stages (initiation, positioning, coordination, and/or sequencing of motor components) in the evolution of an act or sequential action (Grafton, 2002; Heilman and Rothi, 2003; Roy and Square, 1985).

Apraxic disorders may appear when pathways have been disrupted that connect the processing of information (e.g., instructions, knowledge of tools or acts) with centers for motor programming or when there has been a breakdown in motor integration and executive functions integral to the performance of complex learned acts (De Renzi, Faglioni, and Sorgato, 1982; Luria, 1966, 1973b). Thus, when asked to show how he would use a pencil, an apraxic patient who has adequate strength and full use of his muscles may be unable to organize finger and hand movements relative to the pencil sufficiently well to manipulate it appropriately. He may even be unable to relate the instructions to hand movements although he understands the nature of the task (Geschwind, 1975; Heilman and Rothi, 2003). “[T]he hallmark of apraxia is the appearance of well-executed but incorrect movements” (Bogen, 1993).

Apraxias tend to occur in clusters of disabilities that share a common anatomical pattern of brain damage (Dee et al., 1970; Geschwind, 1975). For example, apraxias involving impaired ability to perform skilled tasks on command or imitatively and to use objects appropriately and at will are commonly associated with lesions near or overlapping speech centers, and they

typically appear concomitantly with communication disabilities (Heilman and Rothi, 2003; Kertesz, 1996; Meador, Loring, Lee, et al., 1999). A more narrowly defined relationship between deficits in expressive speech (Broca's aphasia, see pp. 33, 77–78) and facial apraxia further exemplifies the anatomical contiguity of brain areas specifically involved in verbal expression and facial movement (Kertesz, 1996; Kertesz and Hooper, 1982; Verstichel et Cambier, 1996), even though these disorders have been dissociated in some cases (Heilman and Rothi, 2003). Apraxia of speech, too, may appear in impaired initiation, positioning, coordination, and/or sequencing of the motor components of speech (Square-Storer and Roy, 1989). These problems can be mistaken for or occur concurrently with defective articulation (*dysarthria*). Yet language (symbol formulation) deficits and apraxic phenomena often occur independently of one another (Haaland and Flaherty, 1984; Heilman and Rothi, 2003; Roy, 1983).

Constructional disorders

Constructional disorders, often classified as apraxias, are actually not apraxias in the strict sense of the concept. Rather, they are disturbances "in formulative activities such as assembling, building, drawing, in which the spatial form of the product proves to be unsuccessful without there being an apraxia of single movements" (Benton, 1969a). They are more often associated with lesions of the nonspeech hemisphere of the brain than with lesions of the hemisphere that is dominant for speech (De Renzi, 1997b), and they frequently appear with defects of spatial perception (Benton, 1973, 1982). Just as constructional disorders and those involving space perception tend to go together but can each be present as a relatively isolated impairment, so the different constructional disorders may appear in relative isolation. Thus, some patients will experience difficulty in performing all constructional tasks; others who make good block constructions may consistently produce poor drawings; still others may copy drawings well but be unable to do free drawing, etc. Certain constructional tasks, such as clock drawing, are useful bedside examination procedures as the multiple factors required for success (planning, spatial organization, motor control, etc.) make simple construction tasks sensitive to cognitive impairments resulting from a variety of conditions (M. Freedman, Leach, et al., 1994; Strub and Black, 2000; Tuokko, Hadjistavropoulos et al., 1992).

Aphasia

Defects of symbol formulation, the *aphasias* and *dysphasias* (literally, no speech and impaired speech, re-

spectively) were traditionally considered to be apraxias, for the end product of every kind of aphasic or language disturbance is expressive, appearing as defective or absent speech or defective symbol production (F.L. Darley, 1967; Poeck, 1983). An influential older classification of aphasic disorders defined auditory and visual agnosias for symbolic material as receptive aphasias and defined verbal apraxias as expressive aphasias (Brodal, 1981). With expansion and refinements in the systematic observation and treatment of aphasic disturbances, this simplistic two-part classification has lost its usefulness (Benson and Ardila, 1996; Mazzucchi, 2000). Today most investigators identify many more types of aphasia (e.g., Benson, 1993 [ten types]; A.R. Damasio and Damasio, 2000 [eight types]; Kertesz, 2001 [ten types]; Verstichel et Cambier, 1996 [nine types]). Some investigators describe a variety of subtypes as well (e.g., E. Goldberg, 1989; Goodglass, Kaplan, and Barresi, 2000; Luria, 1973b) or decry the usual typologies as having outlived both their usefulness and much contradictory new data (A. Basso, 2003; D. Caplan, 2003; Caramazza, 1984; Howard, 1997).

Analysis of the discrete patterns of defective language processing that can occur with circumscribed brain lesions have identified component processes necessary for normal speech and suggest a regularity in their neuroanatomical correlates (Crosson, 1985; A.R. Damasio and Damasio, 2000; H. Damasio and Damasio, 1989; Naeser, 1982). Broad patterns of correlation between types of language dysfunction and neuroanatomical structures appear with sufficient regularity to warrant the development of aphasia typologies (A.R. Damasio and Damasio, 2000; Geschwind, 1970, 1972; Kertesz, 2001). However, the presentation of aphasic symptoms also varies enough from patient to patient and in individual patients over time that clear distinctions do not hold up in many cases (M.P. Alexander, 2003; Howard, 1997). Thus, it is not surprising that the identification of aphasia *syndromes* (sets of symptoms that occur together with sufficient frequency as to "suggest the presence of a specific disease" or site of damage [Geschwind and Strub, 1975]) is complicated both by differences of opinion as to what constitutes an aphasia syndrome and differences in the labels given those symptom constellations that have been conceptualized as syndromes. The major subdivisions named in much of the literature are presented in Table 2.1, p. 33. It is of interest to note that only four aphasia syndromes are identified by all the authors named in Table 2.1; if all syndromes named in these references were included, the list would be considerably longer.

Several different ways of comprehending the aphasias have been suggested. Benson's (1993) format classifies each of eight relatively common types of aphasia on the basis of whether the patient can repeat what is

TABLE 2.1 Most Commonly Defined Aphasic Syndromes

Aphasia Type	DISTINGUISHING SYNDROME FEATURES				
	Fluency	Comprehension	Repetition	Naming	Other Names
Broca's*	Poor	Good	Poor	Poor	Expressive, motor
Wernicke's*	Good	Poor	Poor	Poor	Receptive, sensory
Global*	Poor	Poor	Poor	Poor	—
Conduction*	Good	Good	Poor	Good	—
Anomic	Good	Good	Good	Poor	Amnesic, semantic
Transcortical motor	Poor	Good	Good	Poor	—
Transcortical	Good	Poor	Good	Poor	—
Subcortical	Fair to good	Variable	Variable	Variable	—

For syndrome descriptions see Benson, 1993; A.R. Damasio and Damasio, 2000; Goodglass and Kaplan, 1983a; Kertesz, 2001; Verstichel et Cambier, 1996.

*Denotes syndromes named in all the above references.

heard. In his schema, the most common aphasia syndromes, except anomic aphasia, are characterized by "abnormal repetition;" *transcortical aphasias* (in which receptive and expressive speech areas remain connected but are isolated from other brain areas necessary for normal speech and language)—which differ from one another in degree of fluency and comprehension—plus anomic aphasia make up the "normal repetition" grouping. Another categorization of the aphasias discriminates between defects in linguistic components of speech such as loss of word meaning (semantic deficits) and agrammatic speech (syntactic deficits) (Saffran, 2003; Marin and Gordon, 1979). Yet another organization format rests on the degree to which the "language-processing systems" are anatomically near or involved with sensory or motor systems (Dronkers et al., 2000; E. Goldberg, 1990). However, Poeck (1983) pointed out that "the syndromes of aphasia . . . are, to a large extent, artifacts produced by the vascularization of the language area" (p. 84). It is possible to define aphasia syndromes because of the large interindividual similarities in brain organization and arterial distribution, which, Poeck estimated, hold for "about 80%" of aphasic patients.

Like other kinds of cognitive defects, language disturbances usually appear in clusters of related dysfunctions. "Impairment of any of the cerebral systems essential to language processes is usually reflected in more than one language modality; conversely impairment of any modality often reflects involvement of more than one process" (Schuell, 1955, p. 308). Thus, *agraphia* (literally, no writing) and *alexia* (literally, no reading) only rarely occur alone. They are most often found together and in association with other language disturbances, typically appearing as impairment rather than total loss of function and in many different forms (Coslett, 2003; Kertesz, 2001; Roeltgen, 2003). In contrast to alexia, which denotes reading defects in persons who could read before the onset of brain damage or disease, *dyslexia* typically

refers to developmental disorders in otherwise competent children who do not make normal progress in reading (Coltheart, 1987; Galaburda, 2001; Lovett, 2003). Developmental dysgraphia differs from agraphia on the same etiological basis (Ellis, 1982). Language disturbances may also occur in confusional states arising from metabolic or toxic disorders rather than from a focal brain lesion (Chédru and Geschwind, 1972).

Mental Activity Variables

These are behavior characteristics that concern the efficiency of mental processes. They are intimately involved in cognitive operations but do not have a unique behavioral end product. They can be classified roughly into three categories: *level of consciousness*, *attentional functions*, and *activity rate*.

Consciousness

The concept of consciousness has eluded a universally acceptable definition (R. Carter, 2002; Dennett, 1991; Farah, 2001). Thus it is not surprising that efforts to identify its neural substrate system and neurobiology are still at the hypothesis-making stage (e.g., Koch and Crick, 2000; Metzinger, 2000, *passim*). Consciousness generally concerns the level at which the organism is receptive to stimulation or is awake. The words "conscious" or "consciousness" are also often used to refer to *awareness* of self and surroundings and in this sense can be confused with "attention." To maintain a clear distinction between "conscious" as indicating an awake state and "conscious" as the state of being aware of something, we will refer to the latter concept as "awareness" (Merikle et al., 2001; Sperry, 1984; Weiskrantz, 1997). In the sense used in this book, specific aspects of awareness can be blotted out by brain damage, such as awareness of one's left arm or some implicit skill memory (Farah, 2000; Schacter, McAndrews, and

Moscovitch, 1988). Awareness can even be divided, with two awarenesses coexisting, as experienced by “split-brain” patients (Baynes and Gazzaniga, 2000; Kinsbourne, 1988; Loring, Meador, and Lee, 1989). Yet consciousness is also a general manifestation of brain activity that may become more or less responsive to stimuli but has no separable parts.

Level of consciousness ranges over a continuum from full alertness through drowsiness, somnolence, and stupor, to coma (Plum and Posner, 1980; Strub and Black, 2000; Trzepacz et al., 2002). Even slight depressions of the alert state may significantly affect a person’s mental efficiency, leading to tiredness, inattention, or slowness. Levels of alertness can vary in response to organismic changes as in metabolism, circadian rhythms, fatigue level, or other organic states (e.g., tonic changes) (Stringer, 1996; van Zomeren and Brouwer, 1987). Variations in brain electrophysiology measured by such techniques as electroencephalography and evoked potentials are seen with altered levels of consciousness (Andreassi, 1995; Daube, 2002; Frith and Dolan, 1997). Although disturbances of consciousness may accompany a functional disorder, they usually reflect pathological conditions of the brain (Lishman, 1997; Strub, 1996b; Trzepacz et al., 2002).

Attention

Attention refers to several different capacities or processes that are related aspects of how the organism becomes receptive to stimuli and how it may begin processing incoming or attended-to excitation (whether internal or external) (Parasuraman, 1998). Showing how widely divergent definitions of attention may be are Mirsky’s (1989) placement of attention within the broader category of “information processing” and Gazzaniga’s (1987) conclusion that “the attention system . . . functions independently of information processing activities and [not as] . . . an emergent property of an ongoing processing system.” Many investigators seem most comfortable with one or more of the characteristics that William James (1890) and others ascribed to attention (e.g., see Leclercq, 2002; Pashler, 1998; Parasuraman, 1998). These include its two aspects, “reflex” (i.e., automatic processes) and “voluntary” (i.e., controlled processes). On the basis of a large-scale factor analysis, Spikman, Kiers, and their collaborators (2001) called them “Stimulus-driven Reaction” and “Memory-driven Action,” respectively, especially noting that the subject’s *control* is a primary characteristic of the latter. Other characteristics of attention that have been identified are its finite resources and the capacities both for disengagement to shift focus and for responsivity to either sensory or semantic stimulus characteristics. An-

other kind of difference between attentional activities has to do with whether it is sustained *tonic attention* as occurs in *vigilance* or it shifts responsively as *phasic attention*, which orients the organism to changing stimuli.

Most investigators conceive of attention as a system in which processing occurs sequentially in a series of stages within different brain systems involved in attention (Butter, 1987; Luck and Hillyard, 2000). This system appears to be organized in a hierarchical manner in which the earliest entries are modality specific while late-stage processing—e.g., at the level of awareness—is supramodal (Butter, 1987; Posner, 1990). Disorders of attention may arise from lesions involving different points in this system (L.C. Robertson and Rafal, 2000; Rousseaux et al., 2002).

A salient characteristic of the attentional system is its *limited capacity* (Lavie, 2001; Pashler, 1998; Posner, 1978; van Zomeren and Brouwer, 1994). Only so much processing activity can take place at a time, such that engagement of the system in processing one attentional task calling on controlled attention can interfere with a second task having similar processing requirements. Thus, one may be unable to concentrate on a radio newscast while closely following a sporting event on television yet can easily perform an automatic (in this case, highly overlearned) attention task such as driving on a familiar route while listening to the newscast.

Attentional capacity varies not only between individuals but also within each person at different times and under different conditions. Depression or fatigue, for example, can temporarily reduce it in intact adults (Landrø, Stiles, and Sletvold, 2001; P. Zimmerman and Leclercq, 2002); old age (Parasuraman and Greenwood, 1998; Van der Linden and Collette, 2002) and brain injury may reduce attentional capacity more lastingly (L.C. Robertson and Rafal, 2000; Rousseaux, Fimm, and Cantagallo, 2002; van Zomeren and Brouwer, 1994).

Simple immediate span of attention—how much information can be grasped at once—is a relatively effortless process that tends to be resistant to the effects of aging and of many brain disorders. It may be considered a form of working memory but is an integral component of attentional functioning (Howieson and Lezak, 2002). Four other aspects of attention are more fragile and thus often of greater clinical interest (Leclercq, 2002; Mateer, 2000; Posner, 1988; Van der Linden and Collette, 2002; van Zomeren and Brouwer, 1994). (1) *Focused or selective attention* is probably the most studied aspect and the one people usually have in mind when talking about attention. It is the capacity to highlight the one or two important stimuli or ideas being dealt with while suppressing awareness of competing distractions. It is commonly referred to as

concentration. Sohlberg and Mateer (1989) additionally distinguished between focused and selective attention by attributing the “ability to respond discretely” to specific stimuli to the focusing aspect of attention and the capacity to ward off distractions to selective attention. (2) *Sustained attention*, or *vigilance*, refers to the capacity to maintain an attentional activity over a period of time. (3) *Divided attention* involves the ability to respond to more than one task at a time or to multiple elements or operations within a task, as in a complex mental task. It is thus very sensitive to any condition that reduces attentional capacity. (4) *Alternating attention* allows for shifts in focus and tasks.

While these different aspects of attention can be demonstrated by different examination techniques, even discrete damage involving a part of the attentional system can create alterations that affect more than one aspect of attention. Underlying many patients’ attentional disorders is *slowed processing*, which can have broad-ranging effects on attentional activities (Gronwall and Sampson, 1974; Ponsford, 1995; Saffran, Dell, and Schwartz, 2000; van Zomeren and Brouwer, 1994).

Patients with brain disorders associated with slowed processing—certain traumatic brain injuries and multiple sclerosis, for example—often complain of “memory problems,” although memory assessment may demonstrate minimal if any diminution in their abilities to learn new or retrieve old information. On questioning, the examiner discovers that these “memory problems” typically occur when the patient is bombarded by rapidly passing stimuli. These patients miss parts of conversations (e.g., a time or place for meeting, part of a story). Many of them also report misplacing objects as an example of their “memory problem.” What frequently has happened is that on entering the house with keys or wallet in hand they are distracted by children or a spouse eager to speak to them or by loud sounds or sight of some unfinished chore. With no recollection of what they have been told or where they set their keys, they and their families naturally interpret this as a “memory problem” rather than one of slowed processing speed which also makes difficult the processing of multiple simultaneous stimuli. When the true nature of this problem is appreciated, patients and families can alter ineffective methods of exchanging messages and conducting activities, with beneficial effects on the patient’s “memory.” (Howieson and Lezak, 2002)

Impaired attention and concentration are among the most common mental problems associated with brain damage (Leclercq, Deloche, and Rousseaux, 2002; Lezak, 1978b, 1989). When attentional deficits occur, all the cognitive functions may be intact and the person may even be capable of some high level performances, yet overall cognitive productivity suffers from inattentiveness, faulty concentration, and consequent fatigue (e.g., Stuss, Ely, Hugenholtz, et al., 1985; Stuss, Stethem, Hugenholtz, et al., 1989).

Activity rate

Activity rate refers to the speed at which mental activities are performed and to speed of motor responses. Behavioral slowing is a common characteristic of both aging and brain damage (see Chapter 7 and pp. 297–298). Motor response slowing is readily observable and may be associated with weakness, poor coordination, or—in testing writing or tracing speed—a prior hand or arm injury. Slowing of mental activity shows up most clearly in delayed reaction times and in longer than average total performance times in the absence of a specific motor disability. It can be inferred from *patterns* of mental inefficiency, such as reduced auditory span plus diminished performance accuracy plus poor concentration, although each of these problems can occur on some basis other than generalized mental slowing. Slowed processing speed appears to contribute significantly to the benign memory lapses of elderly persons (Luszcz and Bryan, 1999; D.C. Park et al., 1996; Salthouse, 1991a).

EXECUTIVE FUNCTIONS

The executive functions consist of those capacities that enable a person to engage successfully in independent, purposive, self-serving behavior. They differ from cognitive functions in a number of ways. Questions about executive functions ask *how* or *whether* a person goes about doing something (e.g., Will you do it and, if so, how and when?); questions about cognitive functions are generally phrased in terms of *what* or *how much* (e.g., How much do you know? What can you do?). So long as the executive functions are intact, a person can sustain considerable cognitive loss and still continue to be independent, constructively self-serving, and productive. When executive functions are impaired, the individual may no longer be capable of satisfactory self-care, of performing remunerative or useful work independently, or of maintaining normal social relationships regardless of how well-preserved the cognitive capacities are—or how high the person scores on tests of skills, knowledge, and abilities. Cognitive deficits usually involve specific functions or functional areas; impairments in executive functions tend to show up globally, affecting all aspects of behavior. However, executive disorders can affect cognitive functioning directly in compromised strategies to approaching, planning, or carrying out cognitive tasks, or in defective monitoring of the performance (P.W. Burgess et al., 1998; E. Goldberg, 2001; Lezak, 1982a; Ogden, 1996, *passim*).

For example, a young woman who survived a head-on collision displayed a complete lack of motivation with inability

to initiate almost all behaviors including eating and drinking, leisure or housework activities, social interactions, sewing (which she had once done well), or reading (which she can still do with comprehension). Although new learning ability is virtually nonexistent and her constructional abilities are significantly impaired, her cognitive losses are relatively circumscribed in that verbal skills and much of her background knowledge and capacity to retrieve old information—both semantic and episodic—are fairly intact. Yet she performs these cognitive tasks—and any other activities—only when expressly directed or stimulated by others, and then external supervision must be maintained for her to complete what she began.

Many of the behavior problems arising from impaired executive functions are apparent even to casual or naive observers. For experienced clinicians, these problems can serve as hallmarks of significant brain injury (Lezak, 1996). Among them are signs of a defective capacity for self-control or self-direction such as emotional lability or flattening, a heightened tendency to irritability and excitability, impulsivity, erratic carelessness, rigidity, and difficulty in making shifts in attention and in ongoing behavior. Deterioration in personal grooming and cleanliness may also distinguish these patients.

Other defects in executive functions, however, are not so obvious. The problems they occasion may be missed or not recognized as neuropsychological by examiners who see patients only in the well-structured inpatient and clinic settings in which psychiatry and neurology patients are ordinarily observed (Lezak, 1982a). Perhaps the most serious of these problems, from a psychosocial standpoint, are impaired capacity to initiate activity, decreased or absent motivation (*anergia*), and defects in planning and carrying out the activity sequences that make up goal-directed behaviors (Lezak, 1989; Luria, 1966; Sohlberg and Mateer, 2001; Walsh and Darby, 1999). Patients without significant impairment of receptive or expressive functions who suffer primarily from these kinds of control defects are often mistakenly judged to be malingering, lazy or spoiled, psychiatrically disturbed, or—if this kind of defect appears following a legally compensable brain injury—exhibiting a “compensation neurosis” that some interested persons may believe will disappear when the patient’s legal claim has been settled.

How crippling defects of executive functions can be is vividly demonstrated by the case of a hand surgeon who had had a hypoxic (*hypoxia*: insufficient oxygen) episode during a cardiac arrest that occurred in the course of minor facial surgery. His cognitive abilities, for the most part, were not greatly affected, but initiating, self-correcting, and self-regulating behaviors were severely compromised. He also displayed some difficulty with new learning—not so much that he lost track of the date or could not follow sporting events

from week to week but enough to render his memory, particularly prospective memory, unreliable for most practical purposes.

One year after the anoxic episode, the patient’s scores on Wechsler Intelligence Scale tests ranged from *high average* (75th percentile) to *very superior* (99th percentile), except on Digit Symbol, performed without error but at a rate of speed that placed this performance low in the *average* score range. His Trail Making Test speed was *within normal limits* and he demonstrated good verbal fluency and visual discrimination abilities—all in keeping with his highest educational and professional achievements. On the basis of a clinical psychologist’s conclusion that these high test scores indicated “no clear evidence of organicity” and a psychiatric diagnosis of “traumatic depressive neurosis,” the patient’s insurance company denied his claim (pressed by his guardian brother) for disability payments. Retesting six years later, again at the request of the brother, produced the same pattern of scores.

The patient’s exceptionally good test performance belied his actual behavioral capacity. Seven years after the hypoxic episode, this 45-year-old man who had had a successful private practice was working for his brother as a delivery truck driver. This youthful-looking, nicely groomed man explained, on questioning, that his niece bought all of his clothing and even selected his wardrobe for important occasions such as this examination. He knew neither where nor with what she bought his clothes, and did not seem to appreciate that this ignorance was unusual. He was well mannered and pleasantly responsive to questions but volunteered nothing spontaneously and made no inquiries in an hour-and-a-half interview. His matter-of-fact, humorless manner of speaking remained unchanged regardless of the topic.

When asked, the patient reported that his practice had been sold but he did not know to whom, for how much, or who had the money. This once briefly married man who had enjoyed years of affluent independence had no questions or complaints about living in his brother’s home. He had no idea how much his room and board cost or where the money came from for his support, nor did he exhibit any curiosity or interest in this topic. He said he liked doing deliveries for his brother because “I get to talk to people.” He had enjoyed surgery and said he would like to return to it but thought that he was too slow now. When asked what plans he had, his reply was, “None.”

His sister-in-law reported that it took several years of rigorous rule setting to get the patient to bathe and change his underclothes each morning. He still changes his outer clothing only when instructed. He eats when hungry without planning or accommodating himself to the family’s plans. If left home alone for a day or so he may not eat at all, although he fixes himself coffee. In seven years he has not brought home or asked for any food, yet he enjoys his meals. He spends most of his leisure time in front of the TV. Though once an active sports enthusiast he has made no plans to hunt or fish in seven years, but he enjoys these sports when taken by relatives.

Because the patient’s brother runs his own business, he is able to keep the patient employed. He explained that he can give his brother only routine assignments that require no judg-

ment, and these only one at a time. As the patient finishes each assignment, he calls into his brother's office for the next one. Although he knows that his brother is his guardian, the patient has never questioned or complained about his legal status. When the brother reinstituted suit for the patient's disability insurance, the company again denied the claim in the belief that the high test scores showed he was capable of returning to his profession. It was only when the insurance adjuster was reminded of the inappropriateness of the patient's life-style and the unlikelihood that an experienced, competent surgeon would contentedly remain a legal dependent in his brother's household for seven years that the adjuster could appreciate the psychological devastation the surgeon had suffered.

PERSONALITY/EMOTIONALITY VARIABLES

Some personality or emotional change usually follows brain injury (Greve et al., 2001; Koponen et al., 2002; Max et al., 2001). Some changes tend to occur as fairly characteristic behavior patterns that relate to specific anatomical sites (e.g., Baribeau and Roth, 1996; Davidson and Irwin, 2002; Gainotti, 1972, 1989; Lishman, 1997; Ruckdeschel-Hibbard et al., 1986). Among the most common direct effects of brain injury on personality are emotional dulling, disinhibition, diminution of anxiety with associated emotional blandness or mild euphoria, and reduced social sensitivity. Heightened anxiety, depressed mood, and hypersensitivity in interpersonal interactions may also occur (Blumer and Benson, 1975; Ghika-Schmid and Bogousslavsky, 2001; K. Goldstein, 1939; D.J. Stein and Hugo, 2002).

Profound personality changes frequently follow brain injury or occur with brain disease. These seem to be not so much a direct product of the illness as patients' reactions to their experiences of loss, chronic frustration, and radical changes in life style. Consequently, depression is probably the most common single emotional characteristic of brain damaged patients generally, with pervasive anxiety following closely behind (J.F. Jackson, 1988; Lezak, 1978b). When mental inefficiency (i.e., attentional deficits typically associated with slowed processing and diffuse damage) is a prominent feature, obsessive-compulsive traits frequently evolve (Lezak, 1989; D.J. Stein and Hugo, 2002). Some other common behavior problems of brain injured people are irritability, restlessness, low frustration tolerance, and apathy (Galbraith, 1985; Heilman, Blonder, et al., 2000, 2003).

Few brain damaged patients experience personality changes that are simply either direct consequences of the brain injury or secondary reactions to impairment and loss. For the most part, the personality changes, emotional distress, and behavior problems of brain damaged patients are the product of extremely com-

plex interactions involving their neurological disabilities, present social demands, previously established behavior patterns, and ongoing reactions to all of these (Gainotti, 1993). When brain injury is mild, personality and the capacity for self-awareness usually remain fairly intact so that emotional and characterological alterations for the most part will be reactive and adaptive (compensatory) to the patients' altered experiences of themselves. As severity increases, so do organic contributions to personality and emotional changes. With severe damage, little may remain of the premorbid personality and of reactive capabilities and responses.

Some brain injured patients display emotional instability characterized by rapid, often exaggerated affective swings, a condition called *emotional lability*. Three kinds of lability associated with brain damage can be distinguished.

1. The emotional ups and downs of some labile patients result from weakened controls and lowered frustration tolerance. This is often most pronounced in the acute stages of their illness and when they are fatigued or stressed. Their emotional expression and their feelings are congruent, and their sensitivity and capacity for emotional response are intact. However, emotional reactions, particularly under conditions of stress or fatigue, will be stronger and may last longer than was usual for them premorbidly (R.S. Fowler and Fordyce, 1974).
2. A second group of labile patients have lost emotional sensitivity and the capacity for modulating emotionally charged behavior. They tend to overreact emotionally to whatever external stimulation impinges on them. Their emotional reactivity can generally be brought out in an interview by abruptly changing the subject from a pleasant topic to an unpleasant one and back again, for these patients will beam or cloud up with each topic change. When left alone and physically comfortable, they typically seem emotionless (M.R. Bond, 1984; Prigatano, 1987).
3. A third group of labile patients differs from the others in that their feelings are generally appropriate, but brief episodes of strong affective expression—usually tearful crying, sometimes laughter—can be triggered by even quite mild stimulation. This is the *pseudobulbar state* (Heilman, Blonder, et al., 2003; Lieberman and Benson, 1977; R.G. Robinson and Starkstein, 2002). It results from structural lesions that involve the frontal cortex and connecting pathways to lower brain structures. This condition has been most usually observed with left-sided anterior damage (House et al., 1990). The feelings of patients with this condition are frequently not congruent with their appearance, and they generally can report the discrepancy. Because they tend

to cry with every emotionally arousing event, even happy or exciting ones, family members and visitors see them crying much of the time and often misinterpret the tears as evidence of depression. Sometimes the bewildered patient comes to the same mistaken conclusion and then really does become depressed. These patients can be identified by the frequency, intensity, and irrelevancy of their tears or guffaws; the rapidity with which the emotional reaction subsides; and the dissociation between their appearance and their stated feelings (B.W. Black, 1982; Heilman, Blonder, et al., 2003; Pino e Melo and Bogousslavsky, 2001).

Although most brain injured persons tend to undergo adverse emotional changes, for a few, brain damage seems to make life more pleasant. This can be most striking in those emotionally constricted, anxious, overly responsible people who become more easygoing and relaxed as a result of a pathological brain condition. A clinical psychologist wrote about himself several years after sustaining significant brain damage marked by almost a week in coma and initial right-sided paralysis:

People close to me tell me that I am easier to live with and work with, now that I am not the highly self-controlled person that I used to be. My emotions are more openly displayed and more accessible, partially due to the brain damage which precludes any storing up of emotion, and partially due to the maturational aspects of this whole life-threatening experience. . . . Furthermore, my blood pressure is amazingly low. My one-track mind seems to help me to take each day as it comes without excessive worry and to enjoy the simple things of life in a way that I never did before. (Linge, 1980)

However, their families may suffer instead. The following case illustrates this kind of personality change.

A young Vietnam veteran lost the entire right frontal portion of his brain in a land mine explosion. His mother and wife described him as having been a quietly pleasant, conscientious, and diligent sawmill worker before entering the service. When he returned home, all of his speech functions and most other cognitive abilities were intact. He was completely free of anxiety and thus without a worry in the world. He had also become very easygoing, self-indulgent, and lacking in both drive and sensitivity to others. His wife was unable to get him to share her concerns when the baby had a fever or the rent was

due. Not only did she have to handle all the finances, carry all the family and home responsibilities, and do all the planning, but she also had to see that her husband went to work on time and that he did not drink up his paycheck or spend it in a shopping spree before getting home on Friday night. For several years his wife tried to cope with the burdens of a carefree husband. She finally left him after he had ceased working and had begun a pattern of monthly drinking binges that left little of his considerable compensation checks.

One significant and relatively common concomitant of brain injury is a changed sexual drive (Boller and Frank, 1981; Foley and Sanders, 1997a,b; Perrigot et al., 1991; Wiseman and Fowler, 2002; Zasler, 1993). A married person who has settled into a comfortable sexual activity pattern of intercourse two or three times a week may begin demanding sex two and three times a day from the bewildered spouse. More frequently, the patient loses sexual interest or capability (Askin-Edgar et al., 2002; L.M. Binder, Howieson, and Coull, 1987; Bolderini et al., 1991; Lechtenberg, 1999; S. Newman, 1984). This leaves the partner feeling unsatisfied and unloved, adding to other tensions and worries associated with cognitive and personality changes in the patient (Lezak, 1978a; Zasler, 1993). For example, some brain damaged men are unable to achieve or sustain an erection, or they may have ejaculatory problems secondary to nervous tissue damage (D.N. Allen and Goreczny, 1995; Bray et al., 1981; Foley and Sanders, 1997b). Patients who become crude, boorish, or child-like as a result of brain damage no longer are welcome bed partners and may be bewildered and upset when rejected by their once affectionate mates. Younger persons brain damaged before experiencing an adult sexual relationship may not be able to acquire acceptable behavior and appropriate attitudes. Adults who were normally functioning when single often have difficulty finding and keeping partners because of cognitive limitations or social incompetence resulting from their neurological impairments. For all these reasons, the sexual functioning of many brain damaged persons will be thwarted (Griffith et al., 1990). Although some sexual problems diminish in time, for many patients they seriously complicate the problems of readjusting to new limitations and handicaps by adding another strange set of frustrations, impulses, and reactions.