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PSYCHIATRIC CONDITIONS ASSOCIATED WITH FOCAL LESIONS OF THE CENTRAL NERVOUS SYSTEM

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Introduction

Traditionally, focal pathology which involves the central nervous system has been the concern of the neurologist and neurosurgeon. It has long been realized, however, that focal pathology can produce a great variety of behavioral changes, some of which may directly involve the psychiatrist and are the subject of this chapter.

Most abnormalities of behavior caused by focal brain disease involve malfunction of major motor and sensory systems and produce little difficulty in diagnosis. Some behavioral manifestations, however, such as those involving the so-called higher functions, e.g., aphasia, memory loss, and attention, are sometimes not readily recognized and categorized. In addition, there is a group of behavioral disturbances produced by focal brain damage which are distinctly psychiatric in nature. The latter include such manifestations as aggressive behavior, compulsiveness, severe anxiety, euphoria, depression, paranoia, and others. This chapter will present some of the disturbances of the higher functions and psychiatric disorders produced by focal lesions. Admixture of symptoms is common and represents a considerable challenge to the diagnostician, particularly when one realizes

that many of the individual signs or symptoms seen with neurological disease also occur frequently in functional disorders.

Not all types of psychiatric symptomatology occur as the result of brain lesions, but the variety observed is extensive. Rejection of the possibility of organic causation because the clinical picture is, for example, typically schizophrenic, is unjustified. Furthermore, one should not automatically assume that psychiatric disorder is only indirectly related to an organic cause. Focal lesions often produce characteristic psychiatric disorders. For example, right hemisphere symptomatology is distinctly different from that of a temporal-lobe epilepsy, and Broca's and Wernicke's aphasics consistently show different emotional behavior.

It should be remembered that brain lesions causing psychiatric disorder may do so without producing any significant disturbance of cognitive function. The extensive literature on frontal lobotomy demonstrates that, despite dramatic changes in behavior, changes in cognitive function are not prominent. Failure of psychological test batteries to show signs of "organicity" does not exclude organic causation for psychiatric symptomatology.

The reader may well decide that the differential diagnosis between organic and functional disorder can be extremely difficult; we would agree.

While organic causes of psychiatric complaints are less common than functional, their recognition is important both for theoretical reasons and because they open additional and specific therapeutic possibilities.

Certain general rules may be helpful. Hallucinations in childhood are far more suggestive of organic causation than when they occur in early adult life. A marked change in personality beyond the age of forty always suggests the possibility of organic disease. Meticulous history-taking may be needed to bring this out, however. If a change in personality occurs insidiously over several years, striking abnormalities may remain unnoticed by the family. Also, many people have a bias towards perceiving the personalities of their close associates as continuous. Thus the woman who has just been beaten by her husband may insist that there has been no change in his personality, yet admit, on direct questioning, that he had never struck her during the first ten years of their marriage.

One must be wary of sweeping statements such as: “Aggressiveness is characteristic of organic disease of the brain.” The symptom of aggressiveness, in fact, differs sharply in organic disorders. Temporal-lobe epileptics often give elaborate justifications for aggressive behavior based on strongly held moral positions; the same sort of justification would be extremely unusual in a patient with a frontal lesion who had been aggressive. Of course, much aggressive behavior, particularly in the young, has no

demonstrable pathology in the brain.

Finally, we must comment on the word “depression” which presents difficulties because of its ambiguous use. At present, it is used both as a diagnosis and as a description of behavior. Even when employed to describe behavior, the usage is ambiguous. The statement that a patient is “depressed” may mean that he is agitated, tearful, guilt-ridden, and suicidal. It may also mean that the patient is apathetic and suffers a psychomotor retardation. While psychomotor retardation may be the cardinal sign of a late-life depression, it is also a characteristic of many organic behavioral disorders. The apathetic, depressed appearance of many cases of organic brain disorder merely reflects a shallowness of emotional expression. Agitated depression, on the other hand, is rare as an organic symptom and a psychogenic depressive illness almost never occurs in patients with severe organic brain disorder. The term “depressed” should therefore be avoided as a description of behavior and replaced by more precise descriptive terms.

Etiologic Factors

In general, the symptomatology produced by focal pathology of the central nervous system (CNS) depends on the anatomic locus of the lesion as well as the specific variety of pathology. The most frequent forms of focal pathology underlying behavioral symptoms are intracerebral space-

occupying lesions, cerebrovascular disease, and trauma. Some manifestations of inflammatory disease, particularly abscess or granuloma, may produce focal symptomatology, but many varieties of central nervous system (CNS) disease (the degenerative, demyelinating, toxic-metabolic, developmental, and most inflammatory disorders) cause widespread CNS pathology (see chapter 3 for additional details). Even these disorders, however, often produce focal disturbances.

Most cerebrovascular and traumatic insults to the brain occur acutely, and the history suggests the probable source of behavioral changes. Many behavioral symptoms, however, do not occur immediately and differentiation from symptoms produced by a reaction to the diseased state may prove difficult. Premorbid personality structure is often retained but individual personality traits may become either exaggerated or damped. Changes in behavioral responses (mood, affect, interpersonal relations) which occur late after acute brain damage may not be manifestations of the original pathology. Additional organic disturbance or purely psychogenic reactions may underlie such changes. Both states are potentially correctable but all too frequently insufficient attention is given to these patients; the clinician tends to accept the original insult as the source of the late behavioral developments.

Space occupying lesions offer much greater diagnostic difficulty. The onset is usually insidious, often with vague behavioral changes, and the

classic signs of CNS involvement frequently appear late. As emphasized by Mayer-Gross et al., the absence of organic clinical features does not exclude the possibility of organic disease; indeed, apparently functional disturbances are often the earliest manifestations of brain tumor. Many psychiatrists have had the sobering experience of misdiagnosing and offering psychotherapy to a patient subsequently proved to have a brain tumor. Abnormal behavior patterns which appear unrelated to present stress, personal or familial background, genetic processes or other positive indications of functional disorder should always arouse suspicion, particularly in the older patient.

Several inflammatory diseases may appear only, or primarily, as focal processes and produce behavioral symptomatology. For instance rabies, a virus infection, involving the hypothalamus and hippocampus, usually produces depression, anxiety, and apprehension initially, then advances to terrifying delusions and ideas of reference. Only later do physical signs, such as pharyngeal spasms, occur. Encephalitis caused by herpes simplex infection usually centers in one or both temporal lobes. Early symptoms may include disorientation, memory loss, and aimless wandering. Bizarre ideation and inappropriate actions may occur before delirium, fever, severe headache, and diminished state of consciousness announce the presence of encephalitis. Most often the course of herpes encephalitis is fairly rapid and dramatic, but if slow and/or partial in development, it may offer a severe challenge for diagnosis. Intracerebral granulomas and abscesses often present the general

signs of space-occupying lesions but, if the onset is insidious, strongly resemble deteriorating functional disturbances. Hypochondriasis, psychomotor retardation including apathy, cognitive deterioration, and loose thought associations may be the presenting signs.

Subcortical Syndromes

One of the striking advances of the past two decades in the understanding of cerebral mechanisms concerns the effects, both facilitatory and inhibitory, of various subcortical structures. Most dramatic was the demonstration by Moruzzi and Magoun of the effect that lesions of the brainstem reticular substance had on sleep and consciousness. In the cat, destructive lesions of the midbrain reticular substance produced a state of somnolence, both clinical and electrical (EEG). The animal would respond to sensory stimuli (noise and touch) and could move all limbs but remained asleep. In contrast, an animal with lesions involving the major motor and sensory pathways of the midbrain, but sparing the reticular substance, retained a normal ability to awaken. It is suggested that this central reticular core, through connections with many subcortical neural structures, acts to modulate and modify cerebral activity. The functions attributed to this system include the initiation and maintenance of wakefulness, the orienting reflex and the focus of attention, many sensory control processes including habituation and external inhibition, conditional learning, memory functions,

and the management of internal inhibition including light and deep sleep.

Another system, essentially antagonistic to the reticular system, has been demonstrated. This is usually called the nonspecific thalamocortical projection system, but involves, anatomically, many subcortical centres including medullary and other brainstem centers, the caudate nucleus, hypothalamic and limbic structures, portions of the thalamus and selected areas of cortex. Identification of these two phylogenetically ancient and interlocking systems, both of which constantly modify cerebral activity, has allowed fuller understanding of complex mental symptomatology. In general, behavioral responses result from a composite of internal and external stimuli set against both of these internal regulating mechanisms. Many behavior modifiers, particularly drugs and the toxic states, are best understood as involving these subcortical systems. These behavior modifiers, while primarily affecting a single system, usually have a widespread (thus nonfocal or multifocal) distribution; there are, however, several focal disorders which produce major behavioral alterations by involvement of these subcortical systems and which will now be discussed.

Consciousness

Modifications of the state of consciousness may be produced by focal lesions involving specific subcortical areas. Thus the unconscious states,

coma, stupor, and lethargy, produced by head injury, are often ascribed to focal involvement of the mesencephalic reticular substance. At times gross pathology (i.e., hemorrhage, infarction, edema, or laceration) is present in the upper brainstem of patients who have suffered prolonged coma; other cases, however, show only minimal evidence of pathology, e.g., demyelination or lymphocytic clusters. Often there is no pathological remnant, even in cases which suffered moderately prolonged unconsciousness. Other varieties of focal pathology may also affect this area and produce changes in the state of consciousness. Thus tumors, aneurysms, hemorrhages, and infectious processes (encephalitis) which involve the upper brainstem often produce a diminution of alertness.

Attention

Two clinically distinguishable varieties of attention abnormality can be ascribed to disorders in the subcortical alerting system. One is the type occurring in drug-intoxication states, head injury, increased intracranial pressure, etc., associated with the diminished state of alertness described above. These patients can be alerted but their attention rapidly wanes and they drift back into a somnolent state (accurately described as “drifting attention”). In these cases the “background” state of alertness is impaired while “phasic” alerting is relatively unaffected. This disturbance of attention usually indicates pathological involvement of the midbrain or thalamic

portion of the reticular activating system.

In the second variety of attention disturbance the patient appears fully alert but has great difficulty maintaining attention on the immediate task (called “wandering attention”). While this patient is alert and attempts to be cooperative, his attention is distracted by almost any external stimulus. A coherent mental evaluation is extremely difficult because of this unpredictable irregularity of attention. Wandering attention may result from either focal or widespread CNS disease; if present, focal pathology is usually located higher in the neuraxis than that which produces the drifting attention state. Subfrontal tumors, CNS lues, and some tumors of the limbic system are characterized by this inability to inhibit external stimuli. Clinically similar defects of attention may be seen in the severely depressed patient (internal distracting stimuli) and the acute schizophrenic with grossly disturbed thought processing (distracted by either internal or external stimuli).

Thus when a patient with a behavioral problem manifests a disturbance of attention, the examiner should suspect pathology involving the two complex, subcortical neural systems which modify cerebral activity, the reticular activating system and the nonspecific thalamo-cortical projection system.

Akinetic Mutism

Akinetic mutism has been defined as a state of limited responsiveness to the environment in the absence of gross alteration of sensory-motor mechanisms. Two varieties are recognized, a hyperpathic type (coma vigil) and an apathetic type (somnolent mutism). In the former variety there is a state of alert or vigilant coma in which the patient lies immobile but appears alert since there is free movement of the eyes in following visual stimuli. If stimulated sufficiently, this patient may become restless or even agitated and may even say a few words but soon settles back to a state of extreme inertia. In the second variety the patient is also immobile but with eyes closed. Only strong stimuli produce movement, including eye opening; vertical gaze palsy or even total ophthalmoplegia, including loss of pupillary light reflex, is usually present. After stimulation, this patient rapidly subsides into the lethargic state, so that this is an apathetic or somnolent variety of akinetic mutism.

The two varieties of akinetic mutism reflect separate neuroanatomical loci. In the apathetic type the lesion, as would be expected, involves the junction area of the mesencephalon and diencephalon and produces both a state of lethargy and occulomotor disturbances. The vigilant variety occurs with pathology higher in the neuraxis, usually involving the postero-medial and inferior aspects of the frontal (septal area) or the hypothalamus. Both varieties involve portions of the reticular activating and/or the diffuse thalamic projection systems. Many varieties of pathology may produce

akinetic mutism, including tumor, granuloma, abscess, encephalitis, hemorrhage, trauma, vascular infarction, angioma, and occult hydrocephalus. Thus this specific behavioral disturbance is dependent on the location, not the nature of the pathology.

Limbic System Syndromes

Since Papez's postulation that portions of the rhinencephalon (limbic system) were important for emotion, there has been an increasing emphasis on the role played by these phylogenetically ancient structures in behavior. For the present discussion the limbic system includes the hippocampus and hippocampal gyrus, the temporal pole, amygdala, fornix, hypothalamus, septal nuclei, certain thalamic nuclei (anterior, dorso-medial and intralaminar), cingulate gyrus, and parts of the orbital frontal cortex. The functions of these structures have been extensively reviewed and need not be listed here. Many behavioral syndromes may result from disease involving the limbic system. It should be noted that most disorders which affect the limbic system also affect other structures. Since, anatomically the limbic system is spread through much of the brain it is often involved along with contiguous, nonlimbic structures. This combination of behavioral features produces a complex clinical picture. Focal involvement of limbic function does occur, however, and can produce striking behavioral pictures. When one considers the "four F's" of the limbic system, feeding, fighting, fleeing, and the undertaking of

mating activity, it is obvious that disorders of the limbic system will often come to the attention of the psychiatrist.

Emotional Disturbance

There has long been awareness that structural brain disorder could cause emotional disturbance and, since Papez, many investigators have considered damage to the limbic system as particularly important. Others have noted that pathology outside the actual limbic system may produce severe behavior disorder which they attribute to limbic release or dis-inhibition. Many of the emotional disturbances produced by damage to the limbic system are similar to those seen in "functional" disorders.

In 1937 Kluver and Bucy demonstrated that bilateral anterior temporal-lobe resection produces a striking behavioral change in the monkey. Abnormalities included psychic blindness, abnormal tameness, hypersexuality, strong oral tendencies and hypermetamorphopsia, the tendency to shift attention frequently. Their work implied that bilateral limbic destruction (or release) could produce striking behavioral abnormality. Very few human cases which fit, this description have been reported. The authors once cared for a university professor, severely injured in an auto accident, who was found at operation to have severe bilateral anterior temporal contusions. Necrotic tissue was removed from the anterior

temporal regions on both sides. During recovery there was a phase, lasting over a month, in which he was fully conscious, and had no demonstrable paresis, primary sensory loss or visual field defect. He did not appear to understand language and spoke only a mumbled jargon. He apparently recognized no one (not even old friends) and could not, or would not, name objects placed in front of him or in his hand. He ate voraciously and showed a tendency to place everything in his mouth, even such things as the tea bags from his tray. He made sexual advances indiscriminately but otherwise had a flat affect. There was an almost constant shifting of attention and if restrained he would become agitated, only to calm down immediately if his attention was diverted. We believed he fulfilled the clinical criteria for the KIuver-Bucy syndrome at that time. He eventually made considerable recovery. Most reported cases of the human Kluver-Bucy syndrome show some, but not all, of the features reported in the original animal experiments.

Flattening of affect (placidity) has also been reported, in tumors and vascular disorders of the limbic system, particularly involving the anterior midline structures (septal area and hypothalamus). The affective change may vary from a mild indifference to a total akinetic mute state (see above). Malamud, in his description of psychiatric symptoms produced by tumors involving the limbic system, described seven cases with anterior midline pathology, who showed affective changes (flattening, depression, manic outbursts, or instability) and who were originally diagnosed as either

schizophrenic or neurotic. Severe depressive symptomatology including withdrawal, negativism, apathy, and even suicide attempts, has also been reported with limbic pathology. Many of the objective findings of depression, are, however, also produced by bilateral brain pathology (see below, frontal lobes). As already noted, psychomotor retardation can be produced in many ways and deserves careful evaluation.

Agitated, aggressive, impulsive, and assaultive behavior have also been ascribed to pathological involvement of the limbic system. Most of these reports suggest a relationship between violent behavior and temporal-lobe disorder, specifically seizures, and will be discussed under Temporal Lobe Syndromes.

Vegetative Disturbances

Pathology involving the limbic system can produce abnormalities in vegetative or endocrine function. When these symptoms are combined with the emotional disturbances described above, the resulting syndrome can readily be mistaken for psychosomatic or neurotic disturbance. Severe anorexia, clinically identical to anorexia nervosa, has been described frequently with hypothalamic disease including tumors. The opposite, i.e., hyperphagia, associated with outbursts of rage, polydipsia, polyuria, and slowly progressive dementia has been reported in a patient with a tiny tumor

of the hypothalamus. Diabetes insipidus is a well known disorder resulting from pathology involving the anterior portion of the pituitary; it can be differentiated from “compulsive water drinking” only by carefully performed water loading tests. Amenorrhea, impotence, sterility, and loss of libido are reported with limbic pathology, particularly that affecting the midline structures. Conversely, hypersexual behavior has been reported following midline encephalitis and abnormal sexual behavior has been reported in cases with seizures arising from temporal-lobe foci.

Memory Disturbances

Another major neuropsychiatric aspect of limbic-system disease concerns memory. In the past two decades a fairly clear-cut clinical-anatomical correlation has been demonstrated for memory defects. Excellent reviews of this work are available and only a synopsis will be presented here.

Many terms have been used to describe memory functions. Unfortunately, the usage of these terms in the literature has not been consistent. In general the functions called memory may be divided into three distinct categories: immediate recall (also called registration, short-term memory, immediate memory, minute memory, auditory attention); recent memory (consolidation, short-term memory, intermediate-term memory, ability to acquire new knowledge, learning, putting to memory); and remote

memory (retrieval, long-term memory, store of information, ability to retrieve old knowledge). Note that "short-term memory" has been and still is used to refer to two different types of memory function, an unfortunate source of additional confusion. Clinical and pathological findings clearly demonstrate that one variety, i.e., recent memory or the ability to acquire new knowledge, depends upon intactness of limbic structures. The classic example of disordered recent memory is Korsakoff's psychosis incidental to thiamine deficiency (see Chapter 15). Similar mental pictures may occur after head trauma (posttraumatic amnesia), cerebrovascular disease, cerebral surgery, and, in a modified state, electroconvulsive shock therapy.

Korsakoff's psychosis is often called the amnestic-confabulatory syndrome to emphasize its two most conspicuous features. The amnesia has distinct clinical characteristics. There is excellent (sometimes supernormal) ability to attend to auditory stimuli, and tests such as digit span are usually normal. In contrast, newly learned material cannot be retained for even a few minutes. Thus the doctor's name is forgotten within minutes and there is disorientation as to time and place because the patient cannot "remember." The ability to retrieve old learned material is relatively preserved, but evaluation reveals a period of retrograde amnesia. Most often this period is at least several years before onset; often there are gross lacunae in memories for many additional years. The best retained memories are either those that are oldest and most overlearned (language, toilet training, feeding, dressing

and grooming activities, and early life experiences) or those concerning emotionally significant occurrences (operations, death of a loved one, etc.).

Confabulation, often noted as the second major symptom of Korsakoff's psychosis, is not a constant feature and is not necessary for the diagnosis, but, when present, is remarkable. The patient answers all questions, often with bizarre responses. Thus, when asked where he is, he will offer an incorrect address or place name; if asked when he last saw the examiner he will give a response, again incorrect and often apparently bizarre; when asked the day and date he hesitates, then responds incorrectly; if asked what he did the previous day he often describes a job, a trip, a visit, or some other activity in considerable but incorrect detail. Careful study of confabulatory responses usually reveals that they represent material from the patient's past. Barbizet has described the confabulatory state as one in which the patient cannot remember that he cannot remember; when asked a question he will offer the best answer available to him, something from his store of old, overlearned memories. Confabulations, then, represent old information offered in answer to a new question.

Recovery from Korsakoff's psychosis is variable, but at least some degree of improvement is usual. The first sign of recovery is realization that something is wrong with the memory. As this concept grows, the amount of confabulation decreases and within two to three months of onset, almost all

patients with Korsakoff's psychosis cease confabulating. Some patients make considerable recovery, sufficient to allow them to return home and even to undertake a simple job. Others, however, fail to recover, and must remain in custodial care. In general, the disability in acquiring new knowledge, as characterized by Korsakoff's psychosis, is accompanied by a striking personality change which usually includes passivity, indifference, apathy, and contentment. In the later stages of the illness, although apathy usually persists, some of the patients do express concern about their disorder and may even show episodes of anger.

Nutritional Korsakoff's psychosis is based on thiamine deficiency secondary to alcoholism or severe malnutrition. The onset is usually acute, with restlessness, confusion, and often an occulomotor abnormality signifying Wernicke's encephalopathy. With prompt treatment (thiamine by injection) the occulomotor disturbance can be corrected. The stage of acute alcohol withdrawal is passed in a few more days. Only then (usually five to ten days after onset) can the presence of Korsakoff's psychosis be ascertained with certainty.

There are many reports on the neuropathology of Korsakoff's psychosis and all agree that bilateral pathology in the mammillary bodies (posterior hypothalamus) is almost always present. A recent study, based on fifty-three cases, claims that degenerative changes in the dorsal medial nucleus of the

thalamus were more important in the pathogenesis of memory disorder than those in the mammillary bodies.

Posttraumatic amnesia (PTA) is even more common than Korsakoff s psychosis but is usually accompanied by so many other signs and symptoms that the memory loss is often not clearly demonstrated. Pure posttraumatic amnesia and Korsakoff's psychosis present similar clinical pictures. Complete recovery, however, is much more likely after traumatic memory loss. During periods of PTA the patient exhibits a long retrograde amnesia amounting to at least several years; if the PTA clears this retrograde amnesia shrinks and becomes very short, often involving only a few seconds preceding the head trauma. From this it would appear that the same structures that are necessary for learning new material are also necessary for retrieval of recently learned material. As head injury produces widespread effects, it is difficult to localise precisely the pathology responsible for PTA. Most investigators agree, however, that the temporal lobes, particularly the hippocampal regions, are the most likely sites of pathology underlying traumatic memory disturbance.

Clinical pictures similar to Korsakoff's psychosis have been reported following cerebrovascular disease. In these cases bilateral posterior cerebral artery obstruction which produced infarction of the medial aspects of both the temporal and occipital lobes has been demonstrated. Unilateral infarction of the left hippocampal region may produce a memory loss similar to that of

Korsakoff s psychosis except that it clears in a few months.

Brain tumor with bilateral medial temporal involvement can cause an amnesic state. Similarly, colloid cysts of the third ventricle and other tumors involving the walls of the hypothalamus can produce an amnestic state. In these cases obstruction of CSF (cerebrospinal fluid) flow producing a hydrocephalus must be considered, but in some reported cases with memory loss this complication has been adequately ruled out.

Typical Korsakoff-like states may occur after temporal-lobe surgery. Scoville reports this finding after bilateral temporal-lobe resection, and a number of surgeons have reported memory loss after unilateral temporal-lobe resection. In the latter, subsequent study usually demonstrated significant pathology in the unoperated temporal lobe. There are reports of amnestic state following bilateral surgical sectioning of the fornix or bilateral fornix infarction but most neurosurgeons quote other works suggesting that there are no residua of bilateral fornix section.

Thus a severe memory disorder (inability to learn new material plus inability to retrieve recently learned material) appears after bilateral injury to central limbic-system structures (hippocampal region and mammillary bodies, possibly the dorsal-medial nucleus of the thalamus and the fornix). When this memory loss, with its strong element of disorientation, is

combined with the emotional and vegetative disorders mentioned earlier, the resulting clinical picture, presents many features commonly seen by the psychiatrist.

Syndromes of Unilateral Hemispheric Involvement

The cerebral hemispheres, comparatively massive in size, are the most striking anatomical feature of the human nervous system. These structures comprise a vast area of enfolded cortex with underlying white matter pathways and subcortical nuclear centers. The two hemispheres have often been considered mirror images of each other, but recent investigations have demonstrated significant asymmetries. Certain behavioral tasks are dependent upon the function, solely or primarily, of only one of the two hemispheres. Language is the prime example of a strongly lateralized cerebral function (dominance), but some other behavioral tasks are, to a greater or lesser degree, also hemisphere-specific. While knowledge of these lateralized functions and of the significance of the interhemispheric connections remains incomplete, knowledge of the recognized hemisphere-specific syndromes is important to the student of behavior.

The dramatic loss of language following left hemisphere damage and the common preference for the right hand in skilled activities has emphasized the importance of the left hemisphere. This hemisphere is often called the

dominant or major hemisphere and some investigators have considered that the right hemisphere acts only as a reserve, an area of cortex which performs only elementary activities but has the potential to take over many of the "higher" functions subserved by the left hemisphere. The possibility that the right hemisphere is also dominant for specific functions has been promoted by the study of constructional disturbances following right hemisphere pathology. Recent studies of behavior following temporal lobectomy and callosal separation have further demonstrated the functional importance of each of the hemispheres and the great importance of interhemispheric cooperation in carrying out complex behavioral tasks.

Left Hemisphere

The loss of language (aphasia) following focal damage to the left hemisphere and the absence of language problems following a similarly placed lesion in the right hemisphere are the most striking manifestations of lateralization of cortical function. Language function is frequently correlated with handedness and the two have been carefully studied. Aphasia, the loss of language function, is the best studied of the lateralized hemisphere disabilities and will be reviewed below (and in Chapter 11). For most humans the left hemisphere appears essential for verbal tasks including speech, comprehension of speech, reading, and writing. The ability to name or describe a function is also specific for the left hemisphere. Studies of the

syndromes of corpus callosum separation, first described by Liepmann demonstrate this clearly. Patients who have suffered separation of the corpus callosum neither name nor describe the function of objects placed in their left hand (when blindfolded) but can select the correct objects from a group (using the left hand). Recognition and memory of the palpated object is performed by the right hemisphere but translation into words is not. In contrast, objects placed in the right hand are immediately named and fully described. Somewhat analogous results are present after left-temporal lobectomy; there is a drop in verbal memory, both in comparison to premorbid scores and to the abilities of individuals who have undergone right temporal lobectomy. Tests of callosally sectioned patients have suggested the presence of some reading capability in the right hemisphere, but the reading level is rudimentary when compared to the function performed by the left hemisphere. Writing is also a strongly lateralized left-hemisphere function. Callosally disconnected patients can write legible sentences with the right hand but either cannot write (except for copying) or write aphasically with the left hand.

Many skilled motor activities appear to be under left-hemisphere control. Actions performed to verbal command are directly dependent upon left-hemisphere function, but even imitation of movements is often difficult for patients who have suffered left-hemisphere damage. This asymmetry of motor function comprises most of the findings referred to as apraxia.

Thus we see that focal damage in the left hemisphere may result in many symptoms related to language or motor control; this includes many of the varieties of aphasia, alexia, agraphia and apraxia. Left-hemisphere damage may be present without any of these symptoms, but their presence strongly suggests left-hemisphere involvement.

Right Hemisphere

Several comparatively rare behavioral abnormalities, e.g., dressing difficulty and prosopagnosia are traditionally linked to right hemisphere damage and several other disturbances, such as constructional difficulty and unilateral neglect, are frequently reported after right-hemisphere damage. Some investigators suggest a single common factor, a disturbance of visual-spatial orientation and recent studies demonstrate that this function is more disturbed following right than left hemisphere damage; visual-motor difficulties are however not confined to rightsided damage.

Constructional disturbance (often called constructional apraxia) denotes a difficulty in drawing, copying, or manipulating spatial patterns or designs. Both right and left hemisphere focal damage are capable of producing constructional disturbance, but most authorities agree that the disturbance is greater in right hemisphere pathology. Problems in construction are seen in a high proportion of cases with structural brain

lesions, and studies show that the disability demonstrated in constructional tasks correlates with both the specific demands of a given test and the locus of pathology. In general the properties of constructional ability which deal with execution (e.g., drawing, manipulation) are more affected by left-hemisphere damage, while the properties dealing with visual-spatial orientation and recall are more affected by right-hemisphere damage. Unfortunately almost all of the standard tests (Bender-Gestalt, Benton three-dimensional figures, etc.) combine both types of task and therefore act as screening tests. The presence of significant constructional disturbance strongly suggests an organic basis for a behavioral problem, and if constructional disturbance is the only significant neurological abnormality noted, a right-hemisphere locus should be suspected.

Prosopagnosia is a state in which there is difficulty in recognizing faces of individuals who are well known to the sufferer. At the extreme, even the patient's own face cannot be recognized in a mirror and male-female distinction is dependent upon clothing or other nonphysiognomic features. Several investigators have suggested that facial recognition depends on subtle visual distinctions and consider that prosopagnosia represents a mild variety, almost a *forme fruste,* of visual agnosia. A number of recent studies have demonstrated that patients with right-hemisphere pathology have greater difficulty in distinguishing facial features than those with left-hemisphere damage. None of their subjects, however, had true

prosopagnosia. Prosopagnosia is rare but it is almost always reported in patients who have other evidence suggesting right-hemisphere pathology. Recently Tzavaras et al. reviewed all cases of true prosopagnosia with postmortem reports in the literature and found that each had bilateral pathology, suggesting that this disorder results, not from right-hemisphere damage alone, but from bilateral involvement of specific areas. This would explain the great rarity of prosopagnosia despite the common occurrence of right hemisphere pathology.

Dressing disturbance (apraxia) was first described in 1941 and has been described frequently since then. Two varieties have been distinguished. One is dependent upon unilateral neglect in which the patient adequately grooms one side of the face and body while totally ignoring the other. The second resembles a visual-spatial disturbance; when given an article of clothing the patient is unable to orient it correctly in space and, while attempting this, hopelessly tangles the article of clothing. The first variety, like other examples of unilateral neglect, is more commonly, but not exclusively, associated with right-hemisphere damage; the visual-spatial type occurs in patients with severe right or bilateral posterior cerebral involvement.

Musical ability, at least that portion dealing with melody, rhythm, and inflection, appears to be a function of the right hemisphere. There are a

number of cases of “amusia” recorded in the literature, some with right-hemisphere damage, others with left. A recent investigation by Bogen and Gordon showed that after injection of sodium amytal into the left carotid artery, the patients could hum a melody but could not sing the words. After right sided injection the opposite happened, i.e., good ability to recite the words but without a recognizable melody.

Unilateral neglect is a fairly common expression of cerebral damage and frequently manifests itself through rather complex behavioral symptomatology. While neglect can occur after damage in either hemisphere, it is most frequent when the site of pathology is on the right. All behavioral symptoms to be mentioned here can occur following left brain damage, but are more common or severe with right-hemisphere involvement.

A great number of variations of unilateral neglect are recognized (see Critchley for detailed classification), dependent on specific combinations of behavioral abnormality.

These variations can be considered points on a spectrum, not sharply distinct clinical entities, but to be understood they must be separated. For this purpose a simple classification will suffice:

1. Inattention to
2. Unconcern about illness, blindness, } illness, blindness, paralysis, etc.
3. Unawareness of paralysis, etc.

4. Denial of

The first and third categories indicate degrees of neglect, most often involving one side only, and are self-descriptive. Unawareness is a more severe degree of unilateral neglect and often indicates the presence of some degree of clouding of the sensorium. While the demonstration of inattention often demands some special examination technique (such as double simultaneous stimulation), simple observation of the patient with unawareness will show considerable decrease in use of the involved side or limb. When confronted, these patients admit that they have the difficulty demonstrated and also admit concern about the disabled state. They do not express concern, however, unless prompted, and attempt their routine activities as though they had no disability. The patient manifesting unconcern, on the other hand, shows evidence of inattention and/or unawareness coupled with an indifference for the disability. He also admits to the disability when confronted, but shows an inappropriate, flat, or facetious reaction. The final variety, in which there is overt denial of the illness, is the most severe. The patient will deny such obvious disabilities as unilateral paralysis or blindness, insisting that he can walk or run, or attempting to describe an unseen panorama in front of him. He often employs vague excuses for not performing the requested task (e.g., “I’ve been ill recently,” “I don’t have my spectacles here,” “The light in this room seems very poor,” etc.). The

symptoms are bizarre and these patients always show some disturbance of mental status. This denial of illness must be differentiated from psychogenic denial, the strongly motivated attempt to downgrade the severity of an illness or situation (see below).

Each variety of unilateral neglect may involve impairment of any sensory or motor function. Denial and unawareness may also involve purely mental tasks (i.e., denial or unawareness of aphasia, memory loss, intellectual deterioration, etc.). Denny-Brown et al. suggested an asymmetry of finely balanced hemispheric functions (*amorphosynthesis*) as the underlying factor in unilateral neglect. They postulated that a system attuned, through years of practice, to respond equally to balanced sensory stimuli reaching each hemisphere will respond most to the stronger signals arriving in the normal hemisphere and will neglect the weaker signals arriving in the damaged hemisphere. Amorphosynthesis appears to offer a basis (anatomically and functionally) for the common occurrence of inattention and unawareness after unilateral cortical damage. The greater prevalence of right- than left-hemisphere damage as the source of unilateral inattention, however, demands additional explanation. The possibility that there is a specific center for modulation (awareness) of this balance has been suggested. Damage to such a center, then, could weaken the ability to recognize unilateral inequalities. This intriguing hypothesis, however, remains totally unsubstantiated and additional factors must be considered.

Active denial of disease cannot be explained simply on the basis of amorphosynthesis. For instance, Anton's Syndrome, the denial of blindness, almost invariably occurs in the context of bilateral visual loss. Even the classic denial of hemiplegia (anosognosia of Babinski) appears to go beyond a mere inequality of stimuli reaching the cortex. This disorder occurs much more frequently in cases of left hemiplegia, and there is a tendency to treat anosognosia as a symptom of right-hemisphere (parietal) disorder. This doctrine has been questioned. Weinstein, who has made extensive studies of the disorder, suggests that anosognosia is motivated, i.e., the patient shows evidence of awareness of the disability which he is denying. As evidence he cites the dying patient, denying illness, but revealing that he recognizes the situation by describing the hospital as "a slaughter-house" or as a final rest home. He also cites the frequent occurrence of reduplication of place, where the patient names the hospital correctly but locates it much closer to his own home town. Weinstein records many patients who suffer anosognosia but do not have right hemisphere disease. While his own material did contain more left than right hemiplegias, we would agree that it is appropriate to consider other aspects of behavior when analyzing denial of illness.

The emotional behavior demonstrated in the milder disturbance, unconcern, is worthy of note. These patients exhibit shallow emotional reactions, often have a mildly euphoric affect, tend to flare up with outbursts of anger which recede rapidly. Often they are facetious and show

inappropriate social behavior. In short, they resemble patients with frontal-lobe pathology (see below) and their unconcern about a unilateral neurological disability could be considered a combination of "frontal lobishness" and amorphosynthesis.

Another possible consideration would be that a disorder of memory had been added to the specific neurological loss. Patients may deny disease because they cannot remember that they have any difficulty. Indeed, this mechanism does occur in some cases of denial and, when carefully sought, significant recent memory loss is present in many cases of denial associated with organic brain disease. There can be little doubt that disorder of memory is a frequent and important component of anosognosia, but there are cases in which it does not seem to be playing a role.

Another theory that has been advanced to explain the greater frequency of denial or unconcern with right rather than left hemisphere lesions is that right-hemisphere lesions produce a change in emotional responsiveness. Proponents of this view note the apathy, facetiousness, and mild euphoria of many left hemiplegics (right-hemisphere damage), contrasting this with the sadness and despair of many right-hemiplegic patients. If correct, this view would suggest right-hemisphere dominance for some aspects of emotion. The strongest evidence for this view is the very difference in the emotional responses of right and left hemiplegics. Evidence advanced from other types

of data to support this concept is still controversial. Some investigators assert that right unilateral ECT (electroconvulsive therapy) is more effective than left in the treatment of depression; this is not substantiated in other reports. Some observers report that after left-carotid amytal injection patients show a right hemiplegia, aphasia, and weeping, while after right-carotid injection they are said to show left hemiplegia and euphoria, but others deny finding these results in their material. The patient composition of the various series may have been different, and this issue must remain open. Certainly, if a unilateral difference in emotional response does exist it would play a significant role in the response to neurologic disability.

A final possibility to be considered is the presence of a purely psychological source of the denial. Certainly psychogenic denial does exist (the repressions, sublimations, etc., of psychodynamics) and sometimes produces a denial of perfectly obvious illness. There are significant differentiating points, however, and the type of denial should always be ascertained. The organic variety of denial occurs only in the face of brain disease complicated by impairment of mental functioning; the functional variety, in contrast, occurs without evidence of coarse brain disease and in a clear mental state. The psychogenic type of denial is usually intellectualized, rationalized, and presented in a quasi-logical manner; in contrast, the denial of the brain-injured patient tends to be crude and concrete.

In summary, organic brain disorders exist in which the presence of disease is neglected or denied. These cases frequently show an inequality of basic sensori-motor function and, in the more severe syndrome of denial of disease, this inequality is complicated by a change in the mental picture, either a facetious, euphoric "frontal-lobe" type of behavior or a loss of recent memory.

Language Syndromes—Aphasia

Much more is known about abnormal language function than any of the other disorders of behavior produced by focal brain damage. Generally termed "aphasia," this subject has been intensively studied for over a century; unfortunately there are still many unsolved problems and a great deal of disagreement exists. When fully developed, aphasia usually presents little diagnostic difficulty and indicates focal brain disease. In more subtle forms, however, aphasia may prove difficult to recognize and is easily mistaken for a functional psychiatric disease. Also, several of the varieties of aphasia produce specific behavior patterns which merit psychiatric concern. A short review of aphasia will help in recognition of these problems.

Aphasia can be defined most simply as a loss or impairment of language caused by brain damage. This definition presupposes that normal language had once been present and excludes pure speech disturbance (i.e., bulbar

palsy, Parkinsonian dysarthria, scanning speech, etc.). For the vast majority of people the function of language is performed almost exclusively by one hemisphere, the left, a factor of obvious significance in the study of focal brain disease. Specifically, over 99 percent of right-handed people have language dominance in the left hemisphere. The picture is not so clear for the left-handed but increasing evidence suggests that some language function in each hemisphere is most common. The presence of aphasia indicates left-hemisphere pathology in at least 95 percent of cases, regardless of handedness.

Further evaluation of the phenomenon of aphasia is dependent upon the classification of the varieties. Dozens of classifications of aphasia are currently in use, based on clinical, anatomical, neurological, psychological, linguistic, and even philosophical considerations. Careful evaluation demonstrates that the same or at least highly similar symptoms appear in many of the classifications, although under different names. The following list presents the classification developed at the Boston Veterans Administration Hospital, which is a modification of several of the nineteenth-century continental classifications and is based on personal evaluation, by the authors, of over 1500 aphasics; it presents this classification of the aphasias as well as some significant related disorders.

Clinical Varieties of Aphasia

A. Aphasia with repetition disturbance

1. Broca's aphasia

2. Wernicke's aphasia

3. Conduction aphasia

B. Aphasia without repetition disturbance

1. Isolation of the speech area

2. Transcortical motor aphasia

3. Transcortical sensory aphasia

4. Anomic aphasia

C. Disturbances primarily affecting reading and writing

1. Alexia with agraphia

D. Total aphasia

1. Global aphasia

E. Syndromes with disturbance of a single language modality

1. Aphemia

2. Pure word deafness

3. Alexia without agraphia

Two of the most popular simple classifications of aphasia deserve comment, if only to reject them. First, the popular expressive-receptive classification of Weisenburg and McBride has serious shortcomings. Almost all aphasics have difficulty with language expression but there are important differences in the type of expressive disorder which this classification overlooks. The equally popular motor-sensory dichotomy, originally proposed in 1874 by Wernicke, is unsuitable because, in great part, the original, specific meanings of these terms have been lost. Most often they are equated to the unsatisfactory expressive-receptive dichotomy. If these terms are to be used, the examiner must recognize that many forms of disordered expression are not "motor" disturbances.

Aphasia may be usefully subdivided into aphasia with normal repetition and aphasia with abnormal repetition. The latter category includes the classic varieties, Broca's and Wernicke's aphasias and a third distinct variety, conduction aphasia. Each variety will be described briefly and correlated with the usual anatomic locus of the causative lesion.

Broca's Aphasia

Originally described in 1861 by Broca and subsequently described under many different names (motor aphasia, cortical dysarthria, verbal

aphasia, efferent kinetic aphasia), this variety of language disturbance has a fairly consistent symptom picture. Conversational speech is nonfluent (dysarthric, sparse, dysprosodic, effortful, of short-phrase length, and consisting mainly of meaning-rich words); comprehension of spoken language is essentially normal; both repetition and naming ability are disturbed but are often better than spontaneous speech. Writing is almost always abnormal, whereas reading comprehension is often preserved. The causative lesion almost invariably involves the posterior-inferior portion of the third frontal convolution (Broca's area). Most patients with Broca's aphasia also have a right hemiplegia, and apraxia (the sympathetic dyspraxia of Liepmann) often affects the left limbs.

Wernicke's Aphasia

A second variety of aphasia, first described in 1874 by Wernicke presents a strikingly different clinical picture. Conversational speech is distinctly fluent (well articulated, presented rapidly with normal melody and phrase length) but is often contaminated by word-finding pauses and paraphasias. The term "paraphasia" designates substitutions within language. This may involve phonemes (literal paraphasia) or words (verbal paraphasia) or may consist of completely incorrect utterances (neologisms). When multiple paraphasias are combined with a rapid output, the production becomes incomprehensible and is sometimes called "jargon aphasia." Since

"jargon aphasia" may also be used to designate other types of disordered speech, one must be careful about its use. The verbal content of the output in Wernicke's aphasia is strongly biased toward relational, grammatical, and filler words and phrases; it is deficient in the meaningful nouns noted in Broca's aphasia. Other characteristics include severe disturbance of both comprehension and repetition of spoken language, inability to read or write, and usually a difficulty in word finding (naming). The causative lesion usually involves the posterior-superior portion of the first temporal gyrus. Often there are no overt neurologic signs (such as paralysis, sensory loss, or hemianopia) except for the language disturbance and, on the basis of his rapid, bizarre speech and poor comprehension, the patient may be misdiagnosed as confused or psychotic.

Conduction Aphasia

Originally characterized by Wernicke and eventually confirmed by a number of European neurologists, this variety still has not been accepted by many investigators. Much recent work including our own gives strong support to its existence and importance. Clinically these cases show fluent speech, often contaminated by paraphasia, with good comprehension but seriously disturbed repetition. Naming is disturbed, as is writing. Quite frequently reading comprehension is maintained, even though reading aloud is impossible. Paresis is minimal or absent, but cortical sensory loss on the

right side of the body is often present. Classically, the causative lesion was described as lying deep in the white matter of the supramarginal gyrus, thus involving the arcuate fasciculus and acting to separate the temporo-parietal language area from the frontal language area. Individual cases of conduction aphasia, however, have been reported in which the significant pathology was a total destruction of the left first temporal gyrus; in other words, in some cases the picture of conduction aphasia rather than Wernicke's aphasia occurs with lesions in this site. Two different anatomical locations of pathology apparently can produce the same clinical syndrome.

The second group of aphasias, those in which repetition is normal, are more difficult to diagnose, and patients with these disorders are frequently referred to the psychiatrist for behavioral investigation. While most often the result of focal brain disease, some of these syndromes can also derive from more widespread dysfunction, particularly degenerative or toxic-metabolic disorders, and, when seen in mild form, offer considerable diagnostic difficulty.

Isolation of the Speech Area

This striking clinical syndrome has only been reported a few times in pure form but is not infrequent in less complete form. The patient with the isolation syndrome does not speak unless spoken to, and then repeats almost

slavishly what has been said by the examiner (echolalia). There is no demonstrable comprehension of spoken or written language, no ability to write or name objects, but, in contrast, great ability to repeat even long and complex sentences, nonsense material, and foreign phrases. Usually these patients can complete stock phrases, e.g., grass is; red, white and. A severe degree of primary neurological disability is usually present and, in fact, almost the only useful function retained by these patients is the ability to repeat. The pathology has, in general, been caused by severe anoxia which has selectively involved the vascular border zone between the middle cerebral and anterior and posterior cerebral tributaries. This pathology spares the immediate perisylvian area but involves large areas of cortex in the frontal, temporal, and parietal lobes.

Transcortical Motor Aphasia

In this disorder the patient is nonfluent in conversational speech (except for a striking ability to echo), comprehends well, and repeats normally. Naming, reading, and writing are usually disturbed. Hemiplegia is present in most cases. The language output may resemble that of Broca's aphasia but is better described as a reluctance to speak. The comparative ease and clarity of repetition is all the more remarkable in contrast. The causative lesion involves the frontal association cortex anterior and/or superior to Broca's area, the frontal portion of the border zone.

Transcortical Sensory Aphasia

In pure from this disorder is rather uncommon, but it occurs fairly often in incomplete form and is often misdiagnosed. The patient speaks fluently but incoherently. He often repeats the examiner's questions (echolalia) but then produces totally unrelated answers. These answers, however, consist of real words, phrases, and sentences with proper intonation. Testing of comprehension shows remarkable disturbance, often a total inability to understand. Alexia and agraphia are present, in addition to a severe naming disturbance. Repetition is dramatically intact. These patients may have cortical sensory disturbance and/or homonymous hemianopsia but often have no paresis. The causative lesion involves the dominant parietal cortex, specifically the parietal border zone and/or the posterior temporal cortex.

Anomic Aphasia

Disturbances of word finding are certainly the most common finding in aphasia, and vary from mild to gross in degree. In the purest form, called anomic aphasia, conversational speech is fluent and somewhat paraphasic, and comprehension of spoken language may be slightly disturbed but is usually adequate, and repetition is perfectly normal. Testing demonstrates some difficulty in word finding and writing, and often (but not always) some disturbance in reading. Usually there is no evidence of elementary

neurological disorder. The clinical picture of anomic aphasia may be the result of focal vascular pathology in the posterior portion of the border zone area. A similar aphasic picture is often a prominent feature of biparietal degenerative disorders such as Alzheimer's Disease, and also appears in toxic or metabolic encephalopathy or raised intracranial pressure. Anomic aphasia also occurs in the recovery stage of many varieties of aphasia. Anomic aphasia, then, is seen frequently but, by itself, does not indicate a specific cerebral focus or particular etiology.

The clinical picture of any case of aphasia depends not only on the areas involved but also on the degree of involvement, the degree of language dominance, and differences in individual language development. Nevertheless, most cases of aphasia can readily be placed in this classification and the site of the underlying focal lesion can be localized with a high degree of accuracy. Table 9-1 gives an outline of the primary differentiating findings of the major types of aphasia.

Table 9-1. Clinical Aspects of Aphasia

	SPONTANEOUS SPEECH	COMPREHENSION	REPETITION	NAMING	READING	WRI
Broca's aphasia	NF	+	—	±	aloud — comp. +	-
Wernicke's aphasia	F	—	—	±	—	-
Conduction	F	+	—	±	aloud —	-

aphasia					comp. +	
Global aphasia	NF	—	—	—	—	-
Isolation syndrome	NF	—	+	—	—	-
Transcortical motor	NF	+	+	—	aloud — comp. +	-
Transcortical sensory	F	—	+	—	—	-
Anomic aphasia	F	+	+	—	+	-

Legend: NF = non fluent; F = fluent; + = normal or mildly affected; — = severely affected; ± = variable degree of involvement.

Several other varieties of aphasia deserve comment. Global aphasia refers to significant disturbance of comprehension with grossly nonfluent speech, usually caused by a large lesion in the sylvian region involving nearly all of the speech regions. Pure word deafness denotes a “pure” disturbance of auditory language comprehension; the patient understands written but not spoken language. Speaking and writing are essentially normal. Aphemia denotes a “pure” disturbance of spoken language, the patient retaining language comprehension and the ability to write. Alexia without agraphia denotes loss of the ability to read with no other language loss. These entities sometimes occur in “pure” form but, not infrequently, they are seen with only slight admixture of other disorders.

Psychiatric Features of Aphasia

Having considered briefly the clinical-anatomical outline of the aphasias recorded above, let us now turn to the behavioral features which may bring the patient to the attention of the psychiatrist. First we will note the problems that lead to diagnostic errors, then the specific reactions seen in aphasic individuals which demand psychiatric management.

Anomic Aphasia

Even a mild difficulty with word-finding (naming on visual or tactile confrontation, manufacturing word lists), suggests organic brain disease and warrants investigation of this possibility. In view of the large number of people using drugs, the possibility of toxic sources of anomia is important. It should be noted that a disproportionate degree of difficulty in writing (agraphia) is almost always present in patients with clinically significant toxic or metabolic disorders.

Word Salad

For many years psychiatrists have described a severe disorganization of spoken and written language which occurs in degenerated schizophrenics, called “word salad” at its most extreme. It is generally recognized that improved treatment of the schizophrenic has made this disorder uncommon,

but the diagnosis is still made occasionally. It has been our experience that every case of "word salad" which we have been asked to evaluate in ten years has had a demonstrable Wernicke's aphasia or, rarely, transcortical sensory aphasia with significant comprehension loss and marked neologistic paraphasia. A specific organic basis has always been demonstrable. Thus, before accepting the diagnosis of word salad on the basis of schizophrenia, patients should be carefully evaluated for evidence of aphasia. There should be little difficulty in making this distinction. Word salad is traditionally a disorder of the chronic, backward schizophrenic. Acute onset of fluent language output filled with paraphasic errors in a previously healthy individual in middle or late life almost invariably indicates the presence of aphasia. Even in the patient with well-established chronic schizophrenic disorder, the *acute* onset of "word salad" should suggest aphasia. The greatest problem occurs in the long-term patient who has been misusing language for many years. Even in this patient, language disorder should be considered and evaluated; there are instances of fluent aphasics misdiagnosed and treated for years as psychotic.

Paranoid Reaction

Among the many recognized sources of paranoid reaction, the psychiatrist should also be aware of the aphasias, particularly those with severe comprehension disturbance (Wernicke's aphasia, pure word deafness

and transcortical sensory aphasia). These patients often ask the examiner to speak more clearly (they hear but cannot understand) and do not realize that they are speaking gibberish. They may believe that those around them are discussing them, possibly in a special code which they hear but cannot understand. They develop severe frustrations and suspicions because their questions or statements go unheeded. A very large number of patients suffering auditory comprehension disability in marked degree show some degree of paranoia (similar to the paranoia which occurs in some cases of long-standing deafness). Their suspicion may be so extreme that the patient becomes a danger to the hospital staff, his family, other patients, acquaintances, or himself. The majority of patients from our aphasia section needing seclusion care have suffered a paranoid reaction, complicating an aphasia with severe comprehension difficulty.

Depression, Frustration, and the Catastrophic Reaction

In aphasia, as in all organic brain disease, the diagnosis of depressed affective state may be difficult. Many patients with organic brain disease, particularly those with anterior involvement, show a blunting of affect, psychomotor retardation, and diminished interest in their surroundings. Yet, when specifically questioned, they do not express depressive feelings. These objective signs of depression are commonly noted after frontal (see below) and certain subcortical (e.g., Parkinsonism) lesions but the appearance of

apathy is not accompanied by a depressed affective state. Nonetheless, true depression can occur in aphasic patients, and is particularly common in Broca's aphasia. In contrast, the patient with a severe aphasia from a posterior lesion rarely exhibits depression; in fact, these patients often fail to recognize their problem and appear euphoric or unconcerned. We believe that the depressive reaction seen in Broca's aphasia is usually a normal response to his disability. The lack of concern of the patient with the posterior lesion, on the other hand, is abnormal and depends on the specific clinical qualities produced by a specifically located brain lesion.

Frustration is seen most frequently in the aphasic with an anterior lesion. While frustration can be unpleasant for the patient and a hindrance to therapy, it is actually a favourable prognostic sign; the patient shows that he cares, is more likely to make an effort, and is therefore a better candidate for therapy. Goldstein described an extreme degree of frustration in aphasia under the term "catastrophic reaction." In this state the degree of frustration was overwhelming, leading to emotional breakdown with a combination of weeping, withdrawal, and anger. The catastrophic reaction is very rare and if the aphasic patient is handled with sympathy, frustration need not interfere with either evaluation or therapy.

In general, understanding, sympathy, and encouragement on the part of the examiner or therapist can overcome most of the complications of

frustration in aphasia. This level of "supportive psychotherapy" is an integral part of the management of almost all victims of aphasia. Affective illness, on the other hand, is often difficult to manage and demands considerable attention. In our experience the depressed aphasic patient has not responded well to treatment with antidepressant drugs and we have been reluctant to use ECT on individuals who have recently suffered a major brain injury. Intensive supportive measures, preferably by someone experienced in communicating with aphasic patients, is usually helpful. We have found that a trained speech therapist, working under the guidance of a psychiatrist, is more useful than either alone or both working with the patient independently. Additional support can be gained by including these patients in a small group with others receiving aphasia therapy. Group therapy not only offers support and a relationship with others suffering a similar disability, but also offers practice in communication in a less stressful environment.

Frontal-Lobe Syndromes

The frontal lobes are the largest divisions of the cortex, and with the Rolandic and sylvian fissures and the sagittal sulcus as boundaries, are also the best demarcated. The frontal lobes, however, are far from homogeneous. At least four distinct subdivisions, based on thalamic connections, can be specified, i.e., Rolandic, sylvian, limbic-temporal, and frontal proper, and

distinct neurological symptomatology has been suggested for each. In many cases, however, an admixture of symptoms referable to these four divisions is seen. In addition, frontal signs are often mixed with signs resulting from damage to other parts of the brain. Despite this common overlap of symptomatology, a clinical picture suggestive of frontal-lobe involvement has been recognized for many years.

Many of the changes produced by frontal-lobe pathology are neurological, (paralysis, aphasia, etc.). In addition, involvement of frontal association cortex can produce a transient total unresponsiveness to visual stimuli in the opposite field, a transient sensory loss (inattention), and occulomotor disturbance; some authors have even attributed a memory defect to frontal lesions. Frontal pathology produces distinct changes in behavior and personality, often referred to as the "frontal-lobe syndrome." The literature contains many descriptions of frontal-lobe syndromes, with variations based on the type of pathological material evaluated or on the orientation of the investigator. To evaluate this we will briefly review reports of frontal head injury, of psychosurgery, and of brain tumors, with several other neurological disorders which primarily affect the frontal lobes.

Head Injury

Behavioral changes following frontal-lobe injury have been reported for

over one hundred years. In 1868 Harlow described his patient Phineas Gage, a previously neat, upright, and capable foreman, who sustained an injury in which a crowbar traversed the left frontal lobe. Following injury the patient was described as irresponsible, vacillating, and incapable of carrying out sequential activities. Many similar case studies have followed, one of the most notable being the patient of Brickner who underwent bilateral frontal-lobe resection for treatment of a parasagittal meningioma. In addition to individual case studies, there are many group studies of patients with frontal-lobe war injuries. Feuchtwanger studied patients with frontal gunshot wounds and described changes in mood and attitude, including facetiousness, euphoria, irritability and apathy, defective attention, tactlessness and inability to plan ahead. Kleist" separated convexity lesions (motor and intellectual abnormalities) and orbital lesions (emotional disturbances), a division confirmed by others. A third locale, called the basal area but actually indicating midline inferior frontal structures, has also been suggested as the source of specific symptomatology. In general, these studies agreed that convexity lesions were characterized by a lack of drive, disinhibition, indifference, lack of productive thinking, euphoria, and incapacity to make a decision. Patients with orbital lesions were said to have normal intelligence on formal testing but severe personality changes; they were aggressive, disinhibited, demanding, interfering, and lacking in perseverance, with increased sexual libido and potency and proneness to criminal offences. With

involvement of the basal area (hypothalamus and orbital frontal region) marked sluggishness and apathy were described, along with a disturbance of the fundamental drives such as appetite, thirst, and sleep.

While these studies suggest that differentiation of the psychiatric picture may be based on the site of focal injury, this division is somewhat artificial. Most head injuries are not well localized, and a broader definition of the frontal-lobe syndrome is needed. In a recent British review of head injury cases Lishman included under the term "frontal lobe syndrome" all patients with one or more of the following psychic symptoms in severe degree: (1) euphoria; (2) lack of judgment, reliability, or foresight; (3) disinhibition; and (4) facile or childish behavior. To this list many investigators would want to add apathy, the loss of drive.

Psychosurgery

Psychosurgery, the attempt to control abnormal behavior through surgical attack on the brain, has produced a great deal of information on the functions of the frontal lobes. While there has been some disagreement in reported results, due in part to variation in the surgical procedure, there has been much agreement on the behavioral outcome of frontal lobectomy and leucotomy, cingulotomy, and orbital undercutting. Greenblatt and Solomon reviewed much of the pertinent literature and their own extensive experience

up to 1956 and outlined four principal behavioral consequences of bilateral frontal lobotomy:

1. Reduced drive demonstrated by

    a. apathy, laziness, lack of initiative and spontaneity, and general contentment;

    b. decrease in suspicion, hostility, aggressiveness, violence, delusions, and fantasy.

2. Reduced self-concern demonstrated by

    a. decreased self-consciousness, less preoccupation with self, less sensitivity to criticism.

3. More immediate outward behavior demonstrated by

    a. less withdrawn, more notice of external activity;

    b. more outspoken, lack of tact, less concern for the future.

4. Superficial, shallow affective state

    a. quicker to become angry, but bear no grudge;

    b. general euphoria.

These behavioral changes are noticeably similar to the changes noted after severe frontal-lobe injury and can be said to characterize the “frontal-

lobe syndrome."

Cerebral Tumor

A third source of study material, of more concern to the practicing psychiatrist, consists of tumors involving the frontal lobes. Onset is usually insidious and behavioral abnormalities often appear first, prompting early psychiatric evaluation. At first behavior may be poorly restrained and tactless, with decreased concern for family members and a mood of fatuous jocularity (*Witzelsucht*). The patient may become boastful or grandiose, but initiative is decreased; work quality deteriorates along with decreasing attentiveness and concern, and finally a state of apathy and indifference replaces the previous euphoria. Socially unacceptable disinhibition such as urinating in public, carelessness in dressing, or inappropriate sexual approach may occur. By this late stage there is often other evidence to suggest neurological disorder such as seizures, unilateral paresis, visual disturbance, headache, or aphasia. These findings may, however, first suggest psychiatric disorder. In a review of 250 cases operated on for frontal-lobe seizure foci, Rasmussen noted six varieties of aura. Three were clearly neurological with combinations of unconsciousness, adversive turning, and generalized grand-mal seizures, but the other three consisted of behavioral symptoms. These included: (a) a vague epigastric sensation, i.e., a rising feeling beginning in the abdomen; (b) vague, poorly described sensations

which involved the entire body, usually called “restlessness,” “flush,” “heaviness,” etc.; and (c) sudden alterations in thought process, a forced thinking. The latter was described by the patients as “forced to think about something,” “my thoughts suddenly became fixed,” or “loss of thought control.” The first sign of aphasia in frontal tumor is almost always a loss of word-finding ability (anomia), producing a rambling, circumlocutory speech pattern, difficult to recognize as aphasic. Thus, tumors of the frontal lobe often mimic psychiatric disorder and offer a formidable diagnostic problem for the psychiatrist.

Syphilis

Before the advent of penicillin, general paralysis of the insane (GPI) was one of the commonest forms of organic brain disease, affecting first, and usually most severely, the frontal lobes (see Chapter 5). Now a rare disorder, GPI still deserves consideration in the differential diagnosis of dementia, especially in the middle-aged. A considerable variation in the onset and course has been reported; usually the onset is insidious with change of temperament and personality occurring before notable intellectual loss. Most often (in about two-thirds of cases) there is a gradual deterioration into a simple or euphoric dementia, characterized by impaired judgement, defective memory, and lack of insight. A much smaller group (about 10 percent) develop the classic expansiveness with a happy, exalted mood and delusions

of superb health, fabulous sexual prowess, masterful artistic capabilities, or superhuman strength. Differentiation from true mania is necessary but usually easy; the GPI victim is childlike and naive and the presence of underlying dementia is often readily demonstrated. The opposite state, serious depression, occurs almost as frequently in the early stages of GPI, with hypochondriacal and nihilistic delusions and even suicide attempts. Again the presence of dementia and a shallow, blunted affective state help to differentiate GPI from true depression. Once suspected, the diagnosis of GPI is readily confirmed by neurological examination and laboratory studies. The outstanding success and comparative safety of penicillin therapy makes it virtually mandatory that any case with reasonable suspicion of GPI receive a full therapeutic course (12-16 million units in divided doses). Many such patients are restored to full mental health (up to 80 percent of cases treated early;) others are left with a stable residual brain damage, usually a dementia with major frontal-lobe features.

Huntington's Chorea

A more generalized disorder of the CNS which often produces major frontal-lobe disturbance is Huntington's chorea (see Chapter 17). The earliest manifestations are usually psychiatric, with insidious but progressive personality deterioration, showing either of two pictures, i.e., irritability, morose discontent, and oversensitivity or apathy and social disinhibition. The

further progression to chorea and dementia is uneven; some patients have severe movement disorder and little dementia, while others show the opposite. Severe personality deterioration, however, invariably occurs and may precede either state by many years. A distinctive feature of the dementia of Huntington's chorea is the relative preservation in most cases of new learning ability in the face of severe intellectual dysfunction and marked distractibility.

Normal Pressure Hydrocephalus

Normal pressure hydrocephalus (NPH), a frequently reversible disorder of cerebrospinal fluid circulation, produces a dementia which features marked frontal-lobe symptomatology. Of the three cardinal symptoms of NPH, gait disturbance, incontinence, and dementia, the first two and at least part of the third appear to result from frontal-lobe dysfunction. In this disorder there is a tremendous overall increase in size of the ventricular system, but with the greatest enlargement demonstrable in the frontal horns. Diagnosis, by intrathecal radioisotope study, air encephalogram, or both can be followed by shunting, most often a ventriculoatrial bypass, which often produces a dramatic improvement in the entire picture.

Presenile Dementia

Of the presenile dementias (see Chapter 3), Pick's disease is characterized by early and marked changes in the frontal lobe in contrast to the early biparietal involvement of Alzheimer's disease. Pick's disease is well described as a "sloppy" dementia with crude, coarse social behavior, incontinence, and apathy. Alzheimer's disease can, in most cases, be termed a "neat" dementia featuring a remarkable preservation of social graces overlying a severe disturbance of cognitive functions. In late stages, with increased involvement of the frontal cortex, Alzheimer patients also develop a "sloppy" dementia. Occasionally, a patient with Alzheimer's disease shows early frontal signs and may be differentiated from patients with Pick's disease only by the greater intellectual disturbances.

Pseudobulbar States

A variable mixture of signs and symptoms is contained in the syndrome usually referred to as pseudobulbar palsy. The prefix "pseudo-" is used to indicate that bilateral upper motor neuron paresis is producing a false impression of lower brainstem (bulbar) pathology. Thus a flattened, expressionless face, lack of eye blinking, hoarseness, dysphagia, and drooling are common. In addition, there is often but not always evidence of bilateral upper motor neuron paresis of the limbs; incontinence, apathy, and disinterest are common but are not essential parts of the picture. The most characteristic finding is a disturbance in the control of behavior, correctly

termed a “lability of emotional expression.” These patients laugh or cry excessively, usually in response to an appropriate but trivial stimulus. In some cases an initial laughing expression can be seen to change slowly to unhappiness and then to agony. While the initial response may be appropriate to the stimulus, the degree of response is not; if asked, the patient will deny experiencing the degree of happiness or sadness that he is expressing and often feels distress because of his inability to control the response.

Recognition of the pseudobulbar state can help the psychiatrist avoid several misdiagnoses. The presence of an expressionless facies in a patient who manifests outbursts of severe weeping in response to appropriate but mild stimuli can easily lead to the diagnosis of a depressive reaction. The pseudobulbar state does not respond well to the present antidepressive drugs and ECT is quite likely to harm an already damaged brain further. The proper diagnosis can be made simply by noting the marked difference between the subjective and the objective expression of emotion, and the presence of bilateral motor involvement.

The drooling and expressionless patient who has outbursts of excessive laughing or crying is easily considered to be demented. It is true that many patients with pseudobulbar state do suffer intellectual deterioration but in some disorders, amyotrophic lateral sclerosis for instance, signs of the

pseudobulbar state may coexist with an entirely intact intellect. Even cases of psuedobulbar palsy secondary to bilateral vascular disease may have considerable retention of intellect. The lability of emotional expression seen in the pseudobulbar state should be considered an example of disinhibition, not of intellectual impairment.

In summary, the behavioral symptomatology of frontal-lobe pathology is varied but can be characterized by: (1) some degree of poor judgment or foresight; (2) superficial or shallow affective state; (3) disinhibition; and (4) reduced drive and self-concern. When some combination of these findings is noted in a behavioral evaluation, organic pathology involving the frontal lobes should be suspected.

Temporal-Lobe Syndromes

The temporal lobe, like the frontal lobe, has long been considered to have a symptomatology of its own but the anatomical demarcation of the temporal lobe is less exact. The sylvian fissure does separate the temporal lobe from the frontal and anterior parietal lobes, but the posterior boundary of the temporal lobe is indistinct. The supramarginal and angular gyri and the temporal-occipital junction are all transitional areas, both anatomically and functionally. Williams suggested three discrete functional areas for the temporal lobe: (1) special sensory, i.e., primarily auditory but also containing

cortical centers for taste, smell, and equilibrium; (2) association, i.e., not only auditory but also visual and possibly some somesthetic association areas which occupy much of the lateral surface of the temporal lobe; and (3) visceral, i.e., the medial and inferior aspects of the temporal lobe contain major structures of the phylogenetically ancient limbic lobe (hippocampus, amygdala, fornix, uncus, hippocampal gyrus). Pathology in the temporal lobe usually involves several of these areas simultaneously, producing a varied symptomatology. Many of the symptoms produced by temporal-lobe dysfunction have already been discussed (aphasia, memory loss, limbic disorders) but one aspect of great significance for the psychiatrist remains, the behavioral abnormalities associated with temporal-lobe seizures.

Temporal-Lobe Seizures

It is generally accepted that the temporal lobe contains the most epileptogenic tissue in the brain, but only in recent years has the full implication of temporal-lobe seizures been realized. Many varieties of motor seizures from short absences to full grand-mal convulsions are the result of temporal foci. Associated in some cases with seizures discharge, but often appearing to occur independently, are many varieties of aura, ictal manifestations and postictal activities which are behavioral acts. The following list outlines these symptoms.

Ictal Symptoms of Psychomotor Epilepsy

I. Sensory symptoms

A. External: Olfactory, auditory, visual, somesthetic sensations

B. Visceral: oropharyngeal, esophageal, abdominal sensations, etc. (i.e., nausea, palpitations, hunger, heat, cold, need to urinate, etc.)

II. Mental symptoms

A. Consciousness: varies from fully normal to totally lost

B. Perceptual:

1) Illusions: micropsia, macropsia, metamorphasia, deja vu, jamais vu, depersonalization, etc.

2) Hallucinations: complex, dynamic, dreamlike

C. Ideational: thought-blocking or interfering thoughts

D. Temporal: time stands still (or rushes by)

E. Affective: fear, depression, pleasant, unpleasant, anger

III. Motor signs

A. Somatic:

1) Simple: clonic contractions, unilateral or bilateral hypertonic: primarily axial, posturings

2) Complex: orienting and investigatory actions; ambulation or flight; response to stimuli (scratching, putting hand to face, clearing throat, etc.); confusional state gestures: palpation of body part, rearrangement of clothes, manipulation of objects, occupational activities

B. Vegetative signs:

1) Respiratory: apnea, polypnea

2) Digestive: mastication, salivation, borborygmi

3) Vasomotor: paleness, flushing

4) Pupillary: usually mydriasis

C. Speech disorders:

1) Aphasia (indicative of left-temporal focus)

2) Speech automatism (indicative of right temporal focus)

This classification was originally presented by Gastaut from a study of several thousand temporal-lobe seizure patients. Several modifications have been made based on subsequent studies. Unfortunately, this list can only offer an outline of the many behavioral disturbances that occur; it cannot provide

the detailed clinical description that each variety deserves. For this the reader is referred to clinical studies. Some aspects of temporal-lobe-seizure behavior have, however, received considerable attention in recent years and deserve to be discussed here.

The motor manifestations of temporal-lobe discharge may be extremely limited, often consisting of only a few seconds of absence, and are easily mistaken for the thought-blocking of a schizophrenic or a neurotic. Motor activity, such as movement of the jaw, mastication, licking of the lips, eye blinking, or the rhythmic jerking of a finger may be observed. A glassy-eyed, vacant stare and a total amnesia for the period of absence are common. Often, however, the patient resumes activity or conversation immediately after the short episode and continues as though nothing had happened. Thus, even an experienced observer may be unaware that he has witnessed an epileptic seizure.

Abnormalities of perception are frequent manifestations of temporal-lobe seizure. Micropsia or macropsia (changes in the size of objects seen) should always suggest temporal or temporal-occipital pathology. “Deja vu” is a feeling that an episode occurring now has occurred in exactly the same fashion in the past (re-experience, familiarity). Efron has suggested that this phenomenon is due to a delay in the callosal transfer of sensory impulses from the nondominant hemisphere to the dominant hemisphere. The delay

could produce a repeated conscious experiencing of the single stimulus and thus a strong sense of familiarity. Deja vu is experienced by almost everyone at some time but, if a frequent complaint, temporal-lobe pathology should be suspected. It is more common with right-hemisphere than left-hemisphere disease.

The presence of an emotion, mood, or feeling tone as part of a psychomotor seizure has received attention. Williams studied all descriptions of the emotional content reported as part of a convulsive episode by several thousand patients. Only one hundred of them described emotional experiences and only four states were noted (fear, depression, pleasantness, unpleasantness). Other observers have confirmed this limited variety of ictal emotionality. Fear is reported most often (well over half in several series) and a report of paroxysmal unexplained feelings of fear should suggest the possibility of psychomotor seizures.

Serafetinides and Falconer studied speech disturbances reported by one hundred patients treated surgically for temporal-lobe seizures, and found significant disturbance in sixty-seven. Dysphasic manifestations (inability to produce or comprehend speech) were associated with left-temporal lesions almost exclusively. Speech automatisms (recurrent utterances), on the other hand, occurred most often in cases with right-temporal-lobe pathology. The patients producing speech automatisms were always unaware (amnesic),

while those with dysphasia were usually aware of their language difficulties.

Aggressive, violent behavior either ictal, postictal or interictal has recently received emphasis as part of the temporal-lobe seizure pattern. Mark et al. speak of a "dyscontrol syndrome" and outline four major symptoms: (1) unrestrained and senseless brutality (particularly wife- or child-beating); (2) manic behavior after limited alcoholic intake (pathological intoxication); (3) sexual assault; and (4) repeated serious traffic accidents. In addition to these symptoms they look for speech or reading defects, visual field defects, memory impairment, seizures, hallucinations "or other indications of schizophrenia," gross sleep disturbances, and episodic mood disturbances. Any combination warrants investigation by EEG and pneumoencephalogram; demonstration of a focal abnormality in the temporal lobe in either would be considered confirmation of psychomotor seizures as the source of behavioral dyscontrol. Mark et al. have recorded a number of carefully investigated cases and their hypothesis has received additional support from other cases. Other investigators, however, have disagreed; the role of temporal lobe seizures in violent behavior remains unsettled (see below).

A number of careful studies have demonstrated that at least one type of serious interictal behavior disturbance may occur in patients with temporal-lobe-seizure disorder. This has been called a schizophrenia-like state and, indeed, is often indistinguishable from schizophrenia. In this condition there

are frequent delusions—both primary and secondary—and hallucinations, mainly auditory, but occasionally mixed with visual, gustatory, or olfactory references. Paranoid states are common, as well as catatonic states and repetitive, stereotyped, ritualistic activities. Affective responses, however, are usually preserved; this preservation of affect and the ability to establish rapport are the major clinical points which differentiate the schizophrenia states from "true" schizophrenia. Pond found no deterioration to a hebephrenic state in the schizophrenia-like group, although partial mental and social incapacity was the longterm outlook. The quasi-schizophrenia state often appears at a time when the seizures decrease or are brought under control, usually many years after the onset of seizures. In the majority (80 percent in Slater's series) there is evidence of temporal-lobe pathology as the source of the seizure focus.

In addition to the schizophrenic-like state, many authors suggest that other aspects of interictal behavior may be altered in patients with temporal-lobe seizures. Personality deterioration, dementing states, and paroxysmal mood changes are frequently reported. Many investigators state that psychomotor epilepsy produces behavior changes which are "clinically indistinguishable from purely psychiatric disorders" (Gibbs). Some feel that impulsiveness and aggressive behavior are common interictal phenomena and use this point to urge earlier and more radical treatment of temporal-lobe epilepsy.

The presence of psychiatric abnormalities in the interictal phase of psychomotor epilepsy, however, is not universally accepted. Guerrant et al. reviewed the literature comparing the behavior of psychomotor and other seizure patients and found an absence of careful documentation. They then analyzed the psychiatric status of thirty-two psychomotor epileptics, twenty-six idiopathic grand-mal patients and twenty-six patients with chronic medical illness not involving the brain, utilizing both psychiatric and psychological evaluations. They found no differences in the incidence of psychiatric abnormality in any of the three groups, and concluded that psychomotor epilepsy did not produce a specific personality derangement. Their conclusion is seriously weakened, however, by the fact that over 90 percent of all three groups, including their "normal" control group, had psychiatric abnormality, and by their own finding that "psychotic" abnormalities were more common in the temporal-lobe group, while "neurotic" abnormalities were more frequent in the medical controls. Stevens performed a similar study comparing psychomotor and grand-mal-seizure patients and found the incidence of psychiatric abnormality approximately equal in the two types, with a much lower incidence in focal nontemporal epileptics. She noted, however, that the prevalent psychiatric disabilities in the psychomotor group included "schizophrenia, mood disturbance, anxiety, and withdrawal" while the grand-mal group showed apathy and mental slowing. Also, the psychomotor patients decompensated psychiatrically when

they became seizure free whereas the grand-mal group decompensated in the face of more frequent seizures. Most recently Mignone et al. analyzed the results of psychological tests given to seizure patients at the NIH and found no significant difference in Minnesota Multiphasic Personality Inventory (MMPI) profiles between psychomotor and nonpsychomotor epileptics. The profiles of both groups, however, were different from normal controls. There would appear to be an increased incidence of behavioral abnormality in patients with psychomotor seizures when compared to normal subjects; whether this behavioral abnormality differs either quantitatively or qualitatively from that of grand-mal epileptics remains unsettled.

The diagnosis of temporal-lobe disorder as the cause of bizarre or paroxysmal behavior depends on a healthy degree of suspicion on the part of the examiner. History of a major seizure occurring at any time of life in a patient with bizarre behavioral problems should arouse suspicion. Confirmation by laboratory studies is not always easy to obtain. Not only routine EEGs but one or more specialized studies such as sleep- or metrazol-activated tracings utilizing special leads (sphenoidal or nasopharyngeal) should be used. The presence of a temporal-spike focus, either unilateral or bilateral, would confirm a suspected temporal-lobe-seizure diagnosis. Air encephalography is often abnormal in patients with temporal-lobe-seizure disorder. This is a hospital procedure with distinct though transient morbidity, and is usually reserved for patients considered for surgery or

where the presence of a tumor is suspected.

Treatment of temporal-lobe-seizure disorder is neither easy nor certain. Anticonvulsants, usually in large doses, are sometimes effective. Mysoline, Dilantin, and phenobarbital are most frequently recommended. Control of interictal symptoms may be aided by use of tranquilizers such as the phenothiazines, Valium or Librium. Successful control has occasionally been reported with other anticonvulsants; bromides, Phenurone, or Mesantoin have all been used but toxicity limits their use to exceptional cases under the closest supervision.

Surgery has proved beneficial in carefully selected cases of temporal-lobe seizures. If the focus for the seizure discharge is localized in one temporal lobe, removal of that lobe often produces improved seizure control, improved personality, and even improved intelligence. Temporal-lobe amputation, however, is known to affect memory; verbal memory is disturbed if the left side is removed, and nonverbal memory by right-temporal amputation. The degree of memory loss, however, is mild and usually not significant to the patient. Bilateral temporal-lobe amputation, on the other hand, produces a severe memory disturbance resembling Korsakoff's psychosis. Similar memory loss has occasionally been reported after unilateral amputation. In this situation, pathology involving the other temporal lobe has been either demonstrated or conjectured. Temporal-lobe

amputation does not appear to alter the schizophrenic-like behavior in most cases. Most investigators agree that surgery has a limited place at present in the treatment of seizures but has been successful in selected cases, and with improving techniques—particularly specific stereotaxic procedures—may play an important role in the future.

Parietal- and Occipital-Lobe Syndromes

The primary function of these posterior hemispheric areas is the reception and integration of extrinsic sensory stimuli. Somesthetic information first reaches the cortex in the post-Rolandic area, and visual stimuli are initially channelled to the calcarine cortex of the occipital lobe. Surrounding both of these areas of primary sensory cortex are large areas of sensory association cortex. In addition, a fairly large area of cortex at the temporo parieto-occipital junction, the angular gyrus, appears to act as a secondary association area, receiving and processing stimuli from visual, somesthetic, and auditory association areas. It is in this area that cross-modal associations (from one sensory sphere to another) are thought to occur. Much of the clinical symptomatology of these two areas consists of demonstrable sensory deficit (e.g., decreased position sense, astereognosis, visual field defect) but some of the symptomatology can mimic psychiatric disturbance.

Intelligence

Damage to the parietal lobe, particularly the angular gyrus, which disrupts second-order associations, may affect certain aspects of intelligence. Involvement of the left angular gyrus usually produces a severe aphasia with constructional disturbance, right-left disturbance, acalculia, and other disturbances to be discussed; but despite these specific disturbances other aspects of intelligence may not be affected. With bilateral parietal involvement, however, severe intellectual deterioration is noted. Alzheimer's disease usually starts with biparietal deterioration; depression of intelligence is an early clinical feature. Analysis of findings, however, demonstrates that specific abnormalities are notable (i.e., anomia, constructional disability, memory disturbance); it does not appear appropriate to consider the parietal lobes as centers for some overall faculty of "intelligence."

Body Image

Through the sensory channels entering the brain (vision, cutaneous sensibility, proprioceptive impulses, labyrinthine inputs, etc.) we are consciously aware of our own bodies, their component parts, and their constantly changing position in space. This complex function may be referred to as "body image" or "body scheme" and is subject to a number of disorders (see Chapter 33). The most prominent disorders of body image are those producing neglect, unawareness, or even denial of a part of one's body, and have been discussed in the section on right-hemisphere disorders. While

many reports link these disorders to parietal defects, other studies suggest that lesions elsewhere may also be implicated. Amorphosynthesis, the inequality of perceptual rivalry discussed earlier, usually indicates parietal or occipital locus of causative lesion.

The Gerstmann syndrome is often cited as an example of disturbance of body image. As originally defined, this syndrome consisted of four components: finger agnosia, right-left disorientation, acalculia, and agraphia. To this complex Schilder added a fifth component, constructional disturbance. There was general agreement that the Gerstmann syndrome indicated dominant (usually left) parietal pathology. Recent studies have questioned the syndrome as lacking in intersymptom correlation, but there is still general agreement that the combination of all four of the originally listed components strongly suggests dominant parietal dysfunction. The fifth component, constructional disturbance (see earlier discussion), while not soley produced by parietal dysfunction is very severe, with biparietal pathology; this is often one of the earliest signs of a dementing process beginning with parietal degeneration.

Neuropsychological investigation of brain-injured individuals has demonstrated that parietal damage, far more than damage in other areas, produces a disturbance of topographical orientation. Both route-finding and maze-learning were abnormal in many cases with parietal damage, but

neither difficulty was related to defect in one hemisphere preferentially. The patient who tells of getting lost on the streets or is unable to find his way about the ward should be suspected of parietal disturbance. This deficit has been termed topographagnosia and may be investigated clinically by asking the patient to draw or locate significant features on a map of his state, country, home, or the hospital ward.

Visual Hallucinations

Most of the signs and symptoms produced by focal pathology in the occipital lobe are obviously neurological or ophthalmologic and are rarely considered functional. An exception, however, must be made for visual hallucinations. There are many varieties of visual hallucination, some associated with psychiatric disorders (e.g., schizophrenia) and some with obvious organic pathology (e.g., temporal or occipital tumor). Some occur in special stress situations without obvious alteration of nervous tissue (e.g., black-patch psychosis, the hallucinations of sensory deprivation) and some are the product of a transient functional alteration (e.g., a migraine aura). Visual hallucinations may occur in many nonfocal brain diseases such as delirium tremens, drug intoxications, febrile states, and encephalitis (see chapters 1 and 2). Often, however, the etiologic causation is not clear when hallucinosis is first investigated and a short review of some focal CNS lesion-producing hallucinations is indicated.

Tumors are well known as a source of visual hallucinations. In 1889 Jackson and Beevor reported well-formed visual hallucinations in a case with a tumor involving the tip of the right temporal lobe. The next year Henschen reported a case of visual hallucinosis in a patient with an irritative lesion of the occipital lobe. Most subsequent reports have confirmed the importance of the temporo-occipital axis in cases of visual hallucinations caused by tumor. There is also a relationship between the nature of the hallucinatory experience and the location of the tumor. With occipital involvement the visual imagery is often brightly colored, diffuse, and formless, usually involving only one half of the visual field. The images are described as floating stars, zig-zags, spots, or fire. When the tumor is more anterior, the hallucinatory images tend to be well formed and are sometimes accompanied by auditory hallucinations. Familiar individuals or objects, often in meaningful activity, have been reported in the visual hallucinations of temporal-lobe tumor cases. Formed hallucinations may also occur in occipital lesions, particularly if the right hemisphere is involved.

Not all visual hallucinations due to structural lesions involve cortical structures, however. There are reports of visual hallucinations occurring in patients subsequently proved to have pathology which involves the subcortical visual pathways. In some the hallucinations consisted of poorly formed images, colored and in motion; in others the images were complex, with recognizable figures and faces. The latter occurred almost exclusively in

patients who became recently blind and persisted after the onset of blindness.

Another type of visual hallucination, reported only rarely but likely to cause diagnostic confusion, is peduncular hallucinosis. Most patients with this disorder are elderly and are usually described as being mildly confused; some complain of giddiness or vertigo, and blindness or severe diminution of vision is usually reported. The hallucinations tend to be persistent and well formed, frequently Lilliputian (little people, miniature animals, etc.), often brightly colored, and usually in rapid movement. The affective response to these hallucinations is often one of pleasure; the patients are interested in and amused by the hallucinatory experience. Only rarely does the hallucination produce distress or alarm. In the cases first reported, vascular pathology involving the mesencephalon was reported. More recently "peduncular hallucinosis" has been reported with mass lesions in the interpeduncular fossa (pituitary or hypothalamic tumors). The disturbance of vision may be due to pressure on the optic tracts, but may also be secondary to bilateral obstruction of the posterior cerebral arteries producing ischemia in the calcarine region, or to other as yet unexplained mesencephalic mechanisms.

Focal causes of hallucinosis are exceptional; most individuals suffering hallucinations have a demonstrable toxic-metabolic or functional source. If the patient reports depression of visual acuity along with the hallucinosis, however, a focal disturbance should be sought.

Brain Tumors

Of all focal neurological disorders producing psychiatric symptomatology, the most perturbing to psychiatrists is the brain tumor. As so cogently stated by Pool and Correll: "There is a pathetic, poignant ineffectiveness about doing psychotherapy in the hope of exorcising an expanding brain tumor. We have become so enchanted with emotional factors in the production of symptoms that we sometimes forget organic components."

Brain tumor is not common in psychiatric practice and its rarity allows the physician to overlook this possibility when seeing a patient with clear-cut behavioral symptomatology. Hard statistics on the frequency of brain tumor in psychiatric practice are not available; several studies report the occurrence of brain tumor as ranging between 0.3 and 0.6 percent of new patients in general psychiatric practices. Mental hospitals report that brain tumor is present in between 1.5 and 4.0 percent of their autopsies.

The classic signs and symptoms of brain tumor, i.e., headache, vomiting, and papilledema often occur too late to be helpful. Most earlier abnormalities such as seizures, hemiparesis, visual field defect, etc., indicate neurological disorder and patients with these findings are usually seen by neurologists or neurosurgeons. Many brain tumors do not produce elementary neurological findings initially, however, and may produce psychiatric symptomatology. In

fact, most individuals with tumors seen by the psychiatrist have no elementary neurological signs or symptoms. The question of why the patient with a brain tumor is so often seen by the psychiatrist has been explored and the following suggested:

1. Behavioral changes may be the only initial finding, but the organic nature of these symptoms may not be obvious.

2. A brain tumor may occur in a functionally psychotic individual.

3. The patient may develop functional symptoms secondary to a misdiagnosis and/or mismanagement of the unrecognized brain tumor.

4. The patient may develop functional symptoms secondary to subjective awareness of decreased function caused by brain tumor.

As the psychiatric symptomatology of the brain-tumor patient may be identical to that arising from psychogenic causes, psychiatrists must remain alert for other suspicious symptomatology. A persistent and increasing headache should always be considered suspicious. Most signs of increased intracranial pressure, however, occur late, often too late for optimal treatment. The most helpful symptomatology depends upon focal disturbances produced by the tumor, a subject already discussed in this chapter. While tumors producing motor, sensory, visual, or extraocular

symptoms, seizures, etc., eventually become obvious, tumors occupying a so-called “silent area”—e.g., the anterior frontal, or posterior parietal regions of either hemisphere, or the right temporal lobe—do not. Psychiatric symptomatology may be the major abnormality.

While a high level of suspicion is the one indispensable tool for diagnosing brain tumors, one simple test may help. Reproduction (copying) of line drawings, including both two dimensional figures (square, daisy, clock, etc.) and three-dimensional figures (cube, house, etc.), are requested, and judgment is made concerning the quality of the reproduction. Poor reproductions may result from unilateral neglect, messiness of lines, alteration of angles, loss of the third dimension, disturbance of either internal or external configuration, etc. Normal adults copy line drawings adequately as do persons with psychogenic disorders, but pathology in either hemisphere involving frontal, parietal, or occipital tissue usually causes difficulty in producing copies. If there is uncertainty about the drawing ability, standardized psychological tests such as the Bender-Gestalt may be employed for confirmation. Almost any type of organic brain disorder including degenerative dementia, head injury, meningitis, etc., will produce abnormality. In contrast, most psychogenic disorders do not cause abnormalities and the tests are valuable as screening measures. While abnormal drawings do not specify location or type of pathology, poor ability to reproduce drawings should be looked upon with considerable suspicion.

Note, however, that significant lesions of the temporal lobes may not cause any drawing problems.

Laboratory studies can be of help in diagnosing brain tumor, but they are only of value when the presence of brain tumor is suspected.

At present, both the electroencephalogram and the radioisotope brain scan offer non-traumatic evaluation for brain tumor and the new computerized axial tomograph (CAT) appears to perform this function even better. If these tests give equivocal or nondiagnostic results, additional testing may be necessary. Lumbar puncture is useful, elevated pressure or elevated protein being suggestive of brain tumor. Arteriography and pneumoencephalography are used frequently and are often mandatory in the full investigation for brain tumor. Each of the last three tests carries a small but real risk for the patient and should be performed under the supervision of a neurologist or neurosurgeon. Negative results are not necessarily useful. There are many reports of negative diagnostic tests in patients subsequently proved to have a tumor.

As the brain is contained in a fixed structure, anything that takes up space acts as a tumor. The list of brain tumors, therefore, is extensive. The tumor most likely to produce psychtetric symptomatology is the meningioma because it grows slowly, often originates in silent areas and can become very

large before producing recognizable neurological symptomatology. Similarly, slow growing members of the glioma family (oligodendroglioma and low-grade astrocytoma) often cause difficulty for the psychiatrist. Subfrontal tumors such as craniopharyngioma and supracellar cyst frequently present with psychiatric symptomatology. Rapidly growing gliomas, dependent upon their location, can also lead to behavioral changes. Hematoma, particularly chronic subdural hematoma of the elderly, and abscess often produce psychiatric symptomatology. Actually, almost anything which occupies space inside the skull can produce psychiatric findings and be mistaken for psychogenic disease. A strong level of suspicion remains the most valuable clinical tool available for this treacherous diagnostic problem.

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Notes

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