American Handbook of Psychiatry

PSYCHIATRIC CONDITIONS ASSOCIATED WITH FOCAL LESIONS OF THE CENTRAL NERVOUS SYSTEM

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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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