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NEUROLOGIC CONDITIONS PRESENTING AS PSYCHIATRIC DISORDERS

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Human behavior is a complex interaction of responses to environmental stimuli and direct responses by our brains. When the brain is diseased or injured it often responds with alterations in human behavior. Occasionally, this behavior will resemble psychiatric diseases of depression, mania, paranoia, schizophrenia, and personality disorders. Thus, although patients may present with what seems a psychiatric diagnosis, neurologic disease must be considered (Table 1). A thorough neurologic examination of all psychiatric patients is essential to uncover unsuspected neurologic disease, and imaging procedures like CT and MR are invaluable in confirming suspected disease.

An understanding of the functional divisions of the brain and how diseases occur in specific areas to produce symptoms of psychiatric disease, can aid psychiatrists and other practitioners to localize defects and search for underlying treatable conditions.

FRONTAL LOBES

Functional areas within the frontal lobes include those areas related to motor activities: the primary motor cortex, frontal eye fields, and the center for motor speech. The cingulate and medial-orbital gyri are parts of the frontal limbic system that influence vegetative functions. The anterior frontal convexity or prefrontal area is concerned with initiation of planned action. Table 2 summarizes the effects of frontal lobe disease. Lesions of the frontal lobes cause three types of syndromes: alteration in motor activities, personality changes, and impairment of cognitive function.

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Table 1. NEUROLOGIC DISEASES THAT MAY PRESENT AS PSYCHIATRIC DIAGNOSES BY DSM-III-R CRITERIA

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- 1. Disorders First Evident in Infancy, Childhood, or Adolescence**
 - Gilles de la Tourette's syndrome
 - Subacute sclerosing panencephalitis
 - Vitamin deficiencies
 - Juvenile Huntington's disease
 - Acute intermittent porphyria
 - 2. Organic Mental Syndromes and Disorders**
 - Alzheimer's disease
 - Pick's disease
 - Dementia of frontal lobe
 - Vitamin B₁₂ deficiency
 - Normal pressure hydrocephalus
 - Metabolic (thyroid disease, Cushing's disease, hypopituitarism)
 - Neurosyphilis
 - Intracranial pathology
 - Wilson's disease
 - Leukodystrophies
 - Neurodegenerative disorders⁹
 - Chronic meningitis
 - Heavy metal poisoning (mercury, thallium, arsenic)
 - 3. Psychoactive Substance Use Disorder**
 - Acute intermittent porphyria
 - Epilepsy
 - Multiple sclerosis
 - Metabolic (thyroid disease, Cushing's disease, hypopituitarism, calcium abnormalities)
 - Infections (encephalitis, meningitis)
 - 4. Schizophrenic Disorders**
 - Complex visual disorders (Balint's syndrome)
 - Lupus cerebritis
 - Alcoholic hallucinosis
 - Temporal lobe epilepsy
 - Parietal lobe disease
 - Amphetamine overdose
 - Huntington's chorea
 - Inorganic mercury poisoning
 - Wilson's disease
 - Neurosyphilis
 - AIDS dementia
 - Neurodegenerative disorders⁹
 - Acute intermittent porphyria
 - Niemann-Pick disease type II-C
 - Aphasia (Wernicke's and transcortical)
 - 5. Delusional Disorder (Paranoia)***
 - Extrapyramidal disorders:* Parkinson's disease, Huntington's disease, Wilson's disease, Sydenham chorea, spinocerebellar degeneration, idiopathic basal ganglia calcification.
 - CNS infections:* viral encephalitis, Cruetzfeldt-Jacob disease, cerebral malaria, syphilis, trypanosomiasis.
 - Demyelinating disease:* multiple sclerosis, metachromatic leukodystrophy, adrenoleukodystrophy.
 - Epilepsy*
 - Neoplasm:* temporal lobe, deep white matter lesions
 - Cerebrovascular disease*
 - Post-traumatic encephalopathy*
 - Degenerative diseases:* Alzheimer's disease, Pick's disease
 - Miscellaneous:* hydrocephalus, cerebral anoxia, Marchiafava-Bignami disease, inborn

(Continued)

Table 1. NEUROLOGIC DISEASES THAT MAY PRESENT AS PSYCHIATRIC DIAGNOSES BY DSM-III-R CRITERIA (Continued).

errors of metabolism (GM2 gangliosidosis, Gaucher's type 1, Niemann-Pick type II-C), paraneoplastic limbic encephalitis.

6. Psychotic Disorder Not Elsewhere Classified

7. Mood Disorders

A. Major Depression

- Frontal lobe syndromes (trauma, postsurgical, tumors)
- Multiple sclerosis
- Huntington's chorea
- Subcortical dementias
- Organic solvent inhalation
- Vitamin B₁₂ deficiency
- AIDS dementia
- Paraneoplastic limbic encephalitis
- Neurosyphilis
- Cerebrovascular disease
- Parkinson's disease
- Progressive supranuclear palsy
- Calcification of basal ganglia
- Colloid cysts of third ventricle
- Pseudobulbar palsy
- Temporal lobe tumors
- Metabolic (Cushing's, thyroid disease, hypopituitarism)

B. Mania†

- Frontal lobe syndromes (trauma, postsurgical, tumors)
- Huntington's disease
- Multiple sclerosis
- Inorganic mercury poisoning (mad-hatter syndrome)
- AIDS dementia
- Neoplasms (diencephalic, hypothalamic, and medial frontal)
- Cerebrovascular lesions (temporal, deep hemispheric)
- Thalamotomy
- Hemidecortication
- Postencephalitic Parkinson's disease
- Wilson's disease
- Epilepsy
- Neurosyphilis
- Pick's disease
- Viral encephalitis
- Klinefelter's syndrome
- Klein-Levin syndrome
- Vitamin B₁₂ deficiency
- Cryptococcal meningitis
- Drugs

8. Anxiety Disorder (Generalized Anxiety Disorder)

- Vitamin B₁₂ deficiency
- Paraneoplastic limbic encephalitis
- Huntington's chorea
- Dementias, frontal lobe disease
- Neurosyphilis
- Toxins (mercury, heavy metals)
- Multiple sclerosis

9. Somatoform Disorder

- Multiple sclerosis
- Frontal lobe dementia
- Acute intermittent porphyria
- Neurosyphilis

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Table 1. NEUROLOGIC DISEASES THAT MAY PRESENT AS PSYCHIATRIC DIAGNOSES BY DSM-III-R CRITERIA (Continued).

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- 10. Dissociative Disorder (Hysterical Neurosis, Dissociative Type)**
 Amnesic syndromes
 Transient global amnesia
 Korsakoff's amnesia
 Parietal lobe disorder
- 11. Sexual Disorders (Hypoactive or Hyperactive Sexual Desire, Exhibitionism)**
 Tumors of the hypothalamus or third ventricle
 Medial temporal lobe diseases
 Klüver-Bucy syndrome
 Frontal lobe disorders
- 12. Sleep Disorders**
 Hypothalamic disorders
 Idiopathic narcolepsy
 Dream anxiety disorder due to apnea or medication
 Sleep apnea
 Restless leg syndrome
 Periodic limb movements of sleep
- 13. Factitious Disorders**
 Multiple sclerosis
 Acute intermittent porphyria
 Subacute heavy metal poisoning
 Epilepsy
- 14. Impulse Control Disorders Not Elsewhere Classified**
 Meige's syndrome
 Temporal lobe epilepsy
 Calcification of basal ganglia
 Gilles de la Tourette's syndrome
 Hypothalamic tumors
- 15. Adjustment Disorders**
 Frontal lobe disease
 Multiple sclerosis
- 16. Psychological Factors Affecting Physical Condition**
 Parkinson's disease
 Huntington's disease
 Gilles de la Tourette's syndrome
 Epilepsy

AXIS II:

- 17. Personality Disorder (Antisocial Personality)**
 Huntington's disease
 Frontal lobe syndromes
 Encephalitis lethargic affecting the brainstem
 Viral encephalitis
 Wilson's disease
 Gilles de la Tourette's syndrome
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*From Cummings JL: Organic psychosis: Delusional disorders and secondary mania. *Psychiatr Clin North Am* 9:293-311, 1986.

†Modified from Cummings JL: Organic psychosis: Delusional disorders and secondary mania. *Psychiatr Clin North Am* 9:293-311, 1986.

Alteration in Motor Function

Besides spastic hemiparesis, damage to the prefrontal cortex produces less well-defined alterations in motor activity including decreased psychomotor activ-

Table 2. EFFECTS OF FRONTAL LOBE DISORDERS

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1. Alterations of Motor Activities
 - a. Lack of spontaneity
 - b. Reduced rate and amount of mental/physical activity
 - c. Akinetic mutism
 2. Intellectual Impairment
 - a. Poor concentration
 - b. Inability to carry out plans
 - c. Attention deficit
 - d. Trouble sequencing tasks
 - e. Slowed mental processing
 3. Personality Change
 - a. Placidity
 - b. Lack of concern over consequences of action
 - c. Social indifference especially with bathing, dressing, bowel and bladder control
 - d. Childish excitement (moria)
 - e. Inappropriate joking, punning (Witzelsucht)
 - f. Instability and superficiality of emotion
 4. Language Dysfunction
 - a. Broca's aphasia
 - b. Mutism
-

ity, motor perseveration, and compulsive manipulation. Akinetic mutism, often caused by trauma to both frontal lobes or interruption of frontal-diencephalic connections leaves patients motionless but not paralyzed and speechless with wandering eye movements. Patients may eventually recover after years in this state or may remain affected indefinitely.²⁵

Personality Changes

It is not possible to correlate exactly anatomic disruption to personality changes but in general, pathology involving the inferior orbital frontal region causes euphoria, hyperactivity, and irritability, whereas involvement of the frontal convexity presents as apathy and slowness.⁵

Hyperactive patients who have orbital frontal lesions are characterized by social disinhibition as the following case demonstrates. A 21-year-old woman, who at age 11 suffered severe head trauma after she was struck by an automobile, presented 30 weeks pregnant by an unknown man. Her seizure disorder was poorly controlled because she frequently forgot to take her anticonvulsant medication. She subsequently became pregnant five more times by different men, each requiring termination. She was unable to manage her financial affairs and frequently used her mother's credit cards. Her mother complained that she had wide mood fluctuations even within minutes. She complained of being depressed, but was seen in euphoric states. Neuropsychological testing revealed her Verbal IQ (Wechsler Adult Intelligence Scale [WAIS]) to be 108 and her Performance IQ to be 106. An MR scan revealed extensive orbital frontal damage (Fig. 1).

Patients with frontal lobe disease exhibit poor judgement, inappropriate language and behaviors including antisocial acts or sexual promiscuity. Outbursts of anger or violence are short-lived and unmaintained. Blumer and Benson⁵ have termed this patient as "pseudopsychopathic." The euphoria is intermittent and intermingled with abulia, apathy, and depression. Formal mental status tests may be entirely normal.

The other frontal lobe personality disorder that occurs with conditions that



Figure 1. MR scan (T_1 -weighted and T_2 -weighted) of a woman with a large orbital frontal lesion causing sexual promiscuity and temper outbursts without loss of intelligence.

affect the frontal convexity presents as apathy, indifference, slowness, and "pseudodepression."⁵ These patients consistently lack initiative, spontaneity, or the interest to care for themselves. They remain intellectually intact and can perform motor tasks if motivation is provided. Although their apathy is often mistaken for depression, they have no sadness or vegetative symptoms. A feature that distinguishes them from true depression is that depressives have a preoccupation with worry whereas patients with frontal lobe disease remain indifferent.^{5,52}

Cognitive Changes

Disorders of cognitive function in frontal lobe disease are primarily problems with attention and memory. The memory deficit is an inability to integrate new experience. Patients may understand errors but are unable to correct them. They think and react directly to a stimulus and have lost the ability to abstract and reason. The memory deficit has been described as the lack of the initiative to remember.²⁵

A small proportion of patients with frontal lobe disease may present with transient disorientation, visual or olfactory hallucinations, or forced thinking about one thought. These may represent epileptic equivalents or auras.²⁵

Diseases that affect the frontal lobes include tumors (especially butterfly gliomas and meningiomas), trauma, stroke, Pick's disease, normal pressure hydrocephalus, and multiple sclerosis.

Dementia of the frontal lobe type accounts for about 20% of patients with dementia.⁴² Progressive personality change, socially inappropriate behavior, and a bland affect are the family's presenting complaint. Patients exhibit numerous somatic complaints and appear to be hypochondriacal. Extremes of behavior such as gluttony, especially for sweets, or total neglect of food; stereotypic behaviors such as hiding of objects; speech disorders like echolalia, perseveration, or mutism; and concrete thinking typify this dementia. The absence of neurologic signs (except for release signs) and a normal electroencephalogram (EEG) until late in the disease

make the diagnosis difficult in life. It frequently is identified only at autopsy with frontal and temporal lobe atrophy without neurofibrillary tangles and plaques of Alzheimer's disease or Pick bodies.⁴² Because of the prominent behavioral changes and absence of cognitive features, these patients can be misdiagnosed as simply eccentric or as suffering from psychiatric disease.

Pick's disease, multi-infarct dementia, and subcortical dementia can present with personality changes of the frontal lobe type in the absence of cognitive changes and may be misdiagnosed as depression in the elderly. These patients have disorders of activity and behavior, not mood. The personality change is usually more dramatic with impaired insight and judgement which are usually preserved in depressive illness. Failure of a middle-aged person to respond to antidepressant therapy should make one question the diagnosis of depression and consider other conditions that may involve the frontal lobes.

Normal pressure hydrocephalus causing dementia with frontal lobe symptoms presents with three primary features: gait disturbance, urinary incontinence, and dementia. The diagnosis is made by evidence of ventricular enlargement on CT scan. Recognition is important because symptoms may be reversible with early shunting, particularly if the dementia appeared after gait disturbance and incontinence.

PARIETAL LOBE

The parietal lobes integrate sensory, visual, tactile, and auditory data and perform intellectual processing. Preferential processing of verbal material occurs in the left hemisphere whereas the processing of primarily visual information takes place in the right hemisphere. Specific functions of the parietal lobe are listed in Table 3. Because clinical manifestations of parietal lobe dysfunction can be subtle and are not always tested on routine neurologic examination, lesions of the parietal lobes can be undetected or misinterpreted as functional or psychiatric disorders.

Denial or neglect, a fascinating syndrome of parietal lobe dysfunction, occurs with lesions of either parietal lobe but is seven times more common with right-sided lesions than with left-sided lesions.¹ Patients not only deny that a hemiplegic limb is abnormal and make excuses why they are not moving it, but also may deny that it is their limb, insisting that it belongs to someone else. This disorder termed

Table 3. EFFECTS OF PARIETAL LOBE DISEASE

I. Dominant (Usually Left) Parietal Lobe Disease	
A.	Alexia with agraphia, with or without anomia
B.	Constructional difficulty
C.	Gerstmann's syndrome (right-left disorientations, inability to localize fingers, agraphia, acalculia)
D.	Astereognosis (inability to recognize objects in the hand)
E.	Pain asymbolia
F.	Ideomotor apraxia
G.	Fluent aphasias
II. Nondominant (Right) Parietal Lobe Disease	
A.	Constructional apraxia
B.	Dressing apraxia
C.	Geographic disorientation
D.	Astereognosis of the left side
E.	Calculation or writing difficulties
F.	Denial or neglect of contralateral space (anosognosia)

anosognosia or denial of illness, is usually associated with other abnormalities including inattention, apathy, or confusion. Patients may experience visual or tactile illusions when sensing the paralyzed part, hallucinations of movement, or allocheiria (one-sided stimuli are felt on the other side). Although a lesion in the superior parietal lobule is responsible for this syndrome of neglect, frontal and right thalamic lesions also can cause neglect.

Because the parietal lobes integrate sensory input and awareness of the position of one's own body in space, parietal defects cause depersonalization. Parietal lobe patients *appear* indifferent to surroundings, making parietal lobe lesions difficult to recognize.

OCCIPITAL LOBE

The occipital lobe processes vision and is essential for visual perception and recognition. Effects of occipital lobe disease are summarized in Table 4.

Cortical Blindness

The most common disorder involving lesions of the occipital lobes are visual field defects. Large, destructive lesions involving both occipital lobes can produce cortical blindness with the usual cause being occlusion of both posterior cerebral arteries, (embolic or thrombotic) and other causes being hypoxia, encephalopathy, leukodystrophies, and trauma. Patients with Anton's syndrome or cortical blindness may actively deny any visual loss or they may seem indifferent to their loss of sight. The behavior of such a patient makes the casual observer unaware of the visual loss.

Balint Syndrome

In Balint syndrome, a complex visual syndrome caused by lesions in both occipital hemispheres, patients lose panoramic vision, have supranuclear "psychic" gaze paralysis, and optic ataxia (inaccurate visually guided limb movements). Patients report seeing only parts of objects as if looking through a keyhole. Such descriptions may lead to a misdiagnosis of tangential speech or hysteria.⁴⁶

Hallucinations

Visual hallucinations can occur in a number of neurologic, ophthalmologic, toxic, and metabolic diseases, and visual hallucinations caused by occipital lobe

Table 4. EFFECTS OF OCCIPITAL LOBE DISORDERS

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1. Anton's syndrome: denial of blindness
 2. Balint syndrome
 3. Visual agnosias: a normal percept stripped of meaning
 Prosopagnosia: inability to recognize faces
 Color agnosia: inability to distinguish color
 4. Alexia: inability to read
 5. Hallucinations
-

damage usually have geometric and colored nonformed quality. The sudden appearance of visual images may be extremely distressing to the patient and lead to psychiatric consultation. There are reports of hallucinations and palinopsia (persistence of visual image after stimulus is gone) caused by stroke presenting as schizophrenia⁴⁴ and psychotic depression.²¹ (For a more detailed description of hallucinations, see the article by Carter elsewhere in this issue.)

TEMPORAL LOBES

The temporal lobes are important for auditory and visual perception, learning and memory, and the emotional state of the individual (Table 5).

Aphasia

Speech and language function is represented in the dominant temporal and frontal lobes with connections to the parietal lobe. Wernicke's aphasia, a fluently articulated but paraphasic speech accompanied by a severe defect in auditory comprehension of all speech (the patient's own and that of others), occurs with a lesion in the posterior aspect of the superior temporal gyrus. Reading, repetition, and writing usually are impaired but other neurologic signs, such as a visual field cut or motor deficit, are often absent. The bizarre speech and absence of other neurologic signs may be confused with a psychotic illness, and careful testing of language function is necessary to differentiate between the two conditions. Language production alone can be misleading and the "word salad" of schizophrenia may be mistaken for an aphasia and vice versa. Schizophrenic patients almost always have intact comprehension, repetition, reading, writing, and naming but it may be difficult to elicit cooperation to test these functions. The neologisms in a

Table 5. PSYCHIATRIC MANIFESTATIONS OF TEMPORAL LOBE DISEASE

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|--|-------|
| <p>A. Unilateral Temporal Lobe Lesions—Dominant Temporal Lobe</p> <ol style="list-style-type: none"> 1. Wernicke's aphasia: frequently mistaken for a psychotic break with neologisms 2. Dysfunctions in memory 3. Anusia: defect in ability to appreciate music <p>B. Nondominant Temporal Lobe</p> <ol style="list-style-type: none"> 1. Agnosia for sounds 2. Dysprosody: disturbed tonal lilt to spoken speech <p>C. Bilateral Temporal Lobe Lesion</p> <ol style="list-style-type: none"> 1. Korsakoff's amnesia 2. Klüver-Bucy syndrome with <ol style="list-style-type: none"> a. visual agnosia b. apathy and placidity c. disturbed sexual function d. dementia, aphasia, amnesia <p>D. Ictal Phenomenon</p> <ol style="list-style-type: none"> 1. Psychosensory <ol style="list-style-type: none"> a. hallucinations (visual, auditory, olfactory) b. illusions (visual, auditory) 2. Affective symptoms 3. Cognitive symptoms (deja vu, jamais vu, forced thinking) 4. Impaired consciousness 5. Automatism | <hr/> |
|--|-------|
-

patient with Wernicke's aphasia tend to be random, changeable, and frequent. In contrast, a schizophrenic uses fewer neologisms, and these are consistent, systematized, and have delusional significance.⁵² For example, Strub et al write "an aphasic patient may call a comb a 'clom', a 'come' or a 'hair thing' at various times while a schizophrenic considers all combs to be 'fleebles' that everyone carries around to monitor his thoughts".⁵² Other paraphasias, such as whole word or letter substitutions, are not heard in schizophrenia because their responses tend to be more elaborate and delusional. The age of the patient, previous psychiatric history, and the clinical time course is important. Wernicke's aphasia presents acutely in an older individual, but a slow growing temporal lobe tumor may be slowly progressive and mimic a psychosis.

Transcortical aphasias is caused by damage to the association cortex with sparing of the perisylvian fissure. A posterior or sensory transcortical aphasia results in impaired comprehension with fluent paraphasic speech and intact repetition. An extreme example of a sensory transcortical aphasia is *echolalia* in which the patient tends to echo everything he or she hears. He or she may be able to repeat everything with the correct grammatical response and in foreign languages never heard before. When asked "Is your name John Doe?", the response will be "Is my name John Doe?" This is most often due to ischemia of the border zone between vascular territories caused by transient hypotension or arrhythmia but can also be seen with thalamic and subcortical lesions, or dementias. Echolalia forms an important component of Tourette's syndrome, catatonic schizophrenia, and autism.

Memory

The anatomic structures important in memory function include the diencephalon (dorsomedial and midline nuclei of the thalamus), hippocampus, amygdala, and mammillary bodies. Diseases which preferentially affect the medial-temporal lobes include hypoxia, hypoglycemia, carbon monoxide poisoning, trauma, herpes simplex encephalitis, and stroke.

Korsakoff's amnesia is a diencephalic amnesia in which the subject is unable to acquire newly learned material rather than failing to retain information. As a result, the patient becomes disoriented in time and place but is able to retrieve distant memory. Patients frequently confabulate and answer all questions put to them with false or bizarre responses. This syndrome is often accompanied by personality changes of apathy and passivity. Korsakoff's syndrome is seen in all forms of nutritional thiamine deficiency and may be accompanied by signs of Wernicke's encephalopathy, including oculomotor signs, ataxia, and confusion.

Transient global amnesia is the sudden onset of severe anterograde and retrograde amnesia in a patient with clear sensorium.¹⁵ It occurs most often in middle-aged or older patients and is probably caused by transient ischemia of both medial-temporal lobes. Recovery usually is complete in a matter of hours and the patient has no memory of the event. The patient may appear confused and tends to be monotonously repetitive, asking the same questions over and over due to the loss of recent memory.

In contrast to the aforementioned causes of memory loss, amnesia caused by psychiatric conditions may require differentiation. Psychogenic amnesia is a disorder in which the patient forgets important personal information, including his or her name, home address, or job, while retaining normal vocabulary, social behavior, and knowledge of current events that are not part of the patient's personal history. Serious social problems, involving finances, the law, or marriage, from which the patient is trying to escape should be sought. Fugue states and multiple

personality disorder are dissociative disorders in which the patient adopts an entirely different identity. Patients in this group carry out natural and complex acts for which they claim no memory. Psychiatric patients are able to learn new information during their amnesia whereas patients with organic amnesic syndromes suffer from anterograde amnesia. This important feature distinguishes between organic amnesia and amnesia caused by dissociative anxiety states.¹⁵

Klüver-Bucy Syndrome

Klüver and Bucy first described a behavioral syndrome in monkeys following bilateral temporal lobectomy that consisted of psychic blindness (visual agnosia), tendency to examine objects orally, an irresistible urge to touch, loss of anger and fear responses, and increased sexual activity.³⁴ The human Klüver-Bucy syndrome resembles the above syndrome but, in addition, features the more complex behavioral features of amnesia, aphasia, dementia, and seizures.³⁴

Psychiatric manifestations of temporal lobe diseases are summarized in Table 5. The psychiatric presentations of temporal lobe epilepsy are covered elsewhere in this issue (see article by Dodrill).

THE LIMBIC SYSTEM INCLUDING THE HYPOTHALAMUS

The limbic system functionally includes the hippocampus, hippocampal gyrus, the temporal poles, amygdala, fornix, hypothalamus, cingulate gyrus, orbital-frontal cortex, septal nuclei, and thalamic nuclei. Disorders of many of these regions already have been discussed as they relate to focal hemispheric syndromes. Emotional disturbances brought on by diseases involving the limbic system include disinhibition of emotional expression, rage reaction and aggression, apathy and placidity, and altered sexuality (Table 6).

Disinhibition of Emotional Expression

Pathologic laughing or crying is characterized by outbursts of involuntary, uncontrollable laughter or crying without apparent cause and an inability to exhibit intermediate forms of emotion such as smiling or frowning. It has been reported to occur with a number of conditions including amyotrophic lateral sclerosis, multiple sclerosis, encephalitis and meningitis, hypoxic-hypotensive encephalopathy, trauma, lipid storage disease, multiple strokes, tumors in and around the third ventricle, and exposure to an insecticide.⁶⁰ The most common cause is lacunar vascular infarcts, which give rise to a symptom complex known as

Table 6. PSYCHIATRIC DISTURBANCES FROM DISEASES OF THE LIMBIC SYSTEM AND HYPOTHALAMUS

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1. Emotional lability: pathologic laughter and crying
 2. Rage and aggression
 3. Altered sexual behavior
 4. Limbic encephalitis associated with malignancy
 5. Delusions
 6. Anorexia nervosa, bulimia
 7. Sleep disorders
-

"pseudobulbar palsy" characterized by dysarthria, dysphagia, and bifacial weakness. The anatomic basis of pseudobulbar palsy is multiple bilateral lesions of the descending corticobulbar and frontopontine pathways and is postulated to be caused by the release of cortical control over lower brainstem centers.²⁶

Rage and Aggression

In 1928, Bard produced "sham" rage in cats by bilateral ablation of the amygdaloid nuclei. These animals exhibited an intense rage reaction with appropriate autonomic responses. The exact human anatomic correlate has not been defined fully but clearly points to the amygdaloid complex and its connections to the hypothalamus.¹ Outbreaks of agitated, aggressive behavior with violent assaultive acts have been reported in the following settings: (1) as a manifestation of temporal lobe epilepsy; (2) as an episodic reaction without identifiable neurologic abnormality; and (3) as part of an acute or chronic neurologic disease such as hemorrhagic leukoencephalitis, herpes simplex encephalitis, traumatic necrosis of the orbital and temporal lobes, ruptured aneurysm of the circle of Willis, gliomas of the anteromedial temporal lobe, and a hypophyseal adenoma.^{1,37,45}

Sexual Behavior

Alteration in sexual behavior has been reported with frontal lobe lesions with disinhibition of behavior and sexual promiscuity. It is also part of the Klüver-Bucy syndrome, indicating bilateral medial temporal lobe involvement. Hyposexuality or loss of libido may be seen with tumors of the hypothalamus.

Paraneoplastic Limbic Encephalitis

The syndrome of paraneoplastic limbic encephalitis deserves special attention as elderly patients frequently present to psychiatrists. A recent review showed that a majority of patients (10 of 19) underwent psychiatric evaluation and admission to a psychiatric hospital receiving psychotropic drugs or electroconvulsive therapy.⁴³ The clinical presentation of limbic encephalitis, includes a change in baseline mental status, difficulty with memory, and affective symptoms including depression, anxiety, personality changes, hallucinations, and catatonia. Other than memory impairment, cognitive functions may be well preserved or there may be associated confusion or delirium. The neurologic exam, CT scan, EEG, and cerebrospinal fluid tests may all be normal initially.

Paraneoplastic processes are caused by the remote effects of cancer on the central nervous system and not as a result of direct invasion. Small cell cancer of the lung is the most common associated malignancy but it also has been reported with breast, stomach, uterine, renal testicular thyroid, and colon cancers.

BASAL GANGLIA

Diseases of the basal ganglia are typically thought of as disorders of movement, but psychiatric and cognitive impairment is common in these diseases. Cummings¹² reviewed the psychiatric disorders of nine diseases with basal ganglia involvement—Parkinson's disease (idiopathic and postencephalitic), Huntington's disease, Wilson's disease, idiopathic basal ganglia calcification, Tourette's

syndrome, spinocerebellar degeneration, progressive supranuclear palsy, and Meige's syndrome. There is no distinct psychiatric disorder accompanying basal ganglia disease, but a spectrum of different psychiatric disorders including depression, mania, obsessive-compulsive disorder, and schizophrenia-like psychosis. Certain psychiatric symptoms are more prominent in certain neurologic diseases. For example, depression is seen commonly with Parkinson's disease, and obsessive-compulsive disorder with Tourette's syndrome. A feature common to all of these movement disorders is the aggravation of the abnormal movement with emotional stress and the alleviation with relaxation.¹²

An understanding of both anatomic and neurochemical connections provides insight into why movement abnormalities and behavioral symptoms commonly occur together and why extrapyramidal symptoms are influenced by emotional states. Major sources of input to the basal ganglia include the limbic system, the primary motor cortex, and motor association areas.¹² Also, four neurotransmitter systems have been implicated in the major extrapyramidal disorders and psychiatric diseases. These include dopamine, noradrenaline, serotonin, and gamma-aminobutyric acid (GABA). Table 7 shows these neurotransmitters and the diseases in which they have been implicated.

Huntington's Chorea

Huntington's chorea is a chronic, progressive hereditary disorder characterized by chorea and dementia. It is inherited in an autosomal dominant fashion with complete penetrance. Symptoms typically begin in midlife but there are variants that present atypically at different times. Psychiatric symptoms in Huntington's disease include personality changes, intermittent affective disorders, and progressive dementia. The mental symptoms may precede chorea by years.

Early changes in personality include decreased interest in work or personal appearance interrupted by periods of irritability and impulsive actions. Episodic mood disorders occur later in the course of the disease and depression is more common than mania.³⁹ Suicide is the cause of death in 7% of nonhospitalized patients with Huntington's disease.³⁹ The depression seen in Huntington's disease is not distinguishable from idiopathic depression, is treatable with antidepressants and electroconvulsive therapy, and suicides are preventable.

Schizophrenic-like psychoses can occur with Huntington's disease in which the patient suffers from hallucinations and delusions. A diagnosis of schizophre-

Table 7. NEUROTRANSMITTERS ASSOCIATED WITH DISEASES OF THE BASAL GANGLIA

Transmitter	State	Disease
Dopamine	decreased	idiopathic Parkinson's disease, postencephalitic Parkinson's disease, progressive supranuclear palsy (PSP)
	increased	Meige's syndrome, Gilles de la Tourette syndrome, schizophrenia
Noradrenaline	decreased	depression
	increased	anxiety
Serotonin	decreased	depression
GABA	decreased	Huntington's disease
	increased	anxiety

Modified from Cummings JL: Psychosomatic aspects movement disorders. Adv Psychosom Med 13:111-132, 1985; with permission of S. Karger AG, Basel.

nia or affective disorder was given initially in six of eight patients with Huntington's disease in one series.³⁹ The incorrect attribution of a movement disorder to neuroleptics and the absence of a family history suggesting Huntington's disease make the diagnosis especially difficult.

Juvenile onset Huntington's disease has less prominent chorea and more rigidity, extrapyramidal and cerebellar signs, and seizures. It may present with intellectual deterioration, poor performance in school, personality changes, or depression. Schizophreniform psychosis is seen more commonly in younger patients.⁷

Pathologically, there is wasting of the caudate and putamen and moderate atrophy of frontal and temporal lobes. Caudate atrophy is visible on CT scan in moderately advanced cases by the disappearance of the bulge created by the caudate into the frontal horns of the lateral ventricles (Fig. 2).

Wilson's Disease

Progressive hepatolenticular degeneration or Wilson's disease is an autosomal recessive inherited deficiency of ceruloplasmin that results in progressive accumulation of copper in the liver, brain, cornea, and kidney. The copper deposition normally occurs first in the liver and can present with hepatomegaly, acute or chronic hepatitis, or cirrhosis anytime after the age of 5 years. However, 40% of patients present in the second or third decade with neurologic symptoms including tremor, dysarthria, mild clumsiness, ataxia, or rigidity. Cognitive function is preserved in the majority of patients, although many patients are mistaken as mentally retarded secondary to the dysarthria.

Ten percent to twenty-five percent of patients will present with psychiatric disturbances such as dementia, psychoneurosis, manic-depressive or schizophrenic psychosis, and frontal lobe type behavioral abnormalities.⁵⁰ Alcoholism is common due to the knowledge that alcohol decreases the tremors. In the absence of neurologic symptoms, patients may be diagnosed as psychopathic personality

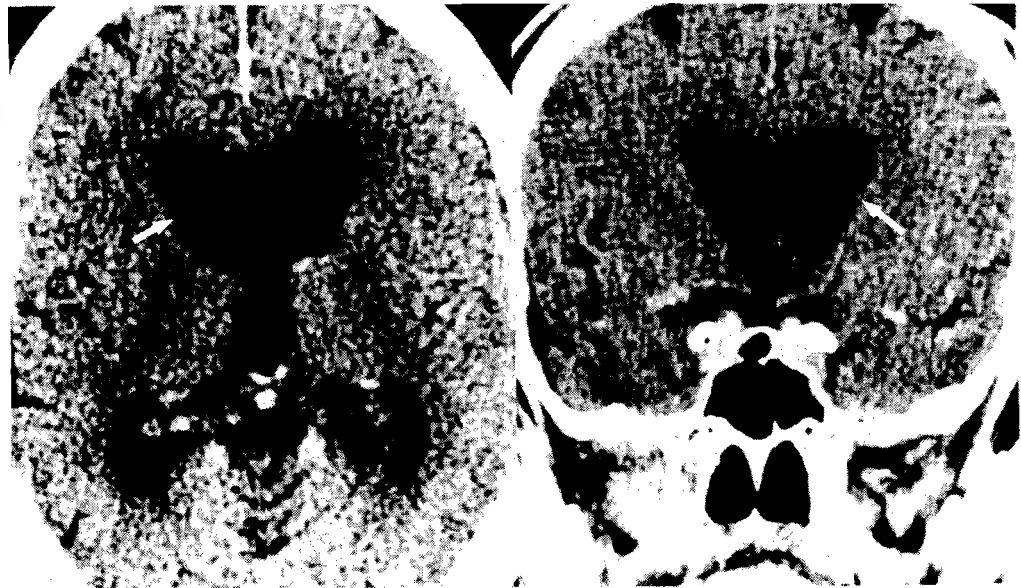


Figure 2. CT scan (axial and coronal) of a man with typical loss of the caudate nucleus (arrows) in Huntington's disease.

disorders. Treatment with penicillamine potentially can reverse the neurologic and psychiatric manifestations of the disease if started early but psychiatric symptoms are less responsive. Patients often require long-term antidepressants and psychotherapy in addition to decoppering therapy.

The diagnosis of Wilson's disease is made by demonstration of low serum ceruloplasmin, low serum copper, increased urinary excretion of copper, and the presence of Kayser-Fleischer rings in the cornea, which are golden or green-brown rings in the cornea that represent deposits of copper. Almost 100% of psychiatric presentations of Wilson's disease will show Kayser-Fleischer rings in the cornea.⁵⁰

Parkinson's Disease

Parkinson's disease is characterized by four cardinal features: akinesia, rigidity, rest tremor, and loss of normal postural reflexes. Studies show, however, that most patients eventually will develop depression¹² (40% to 60%) or dementia. Dementia occurs 10 times more frequently in parkinsonism than in age-matched controls, with the severity of dementia correlating with progression of the disease.⁵¹ As a complicating factor, all antiparkinsonian drugs can produce confusion, memory impairment, and psychosis, with anticholinergics, levodopa, bromocriptine, and amantadine being the worst offenders. Still⁵¹ gives the following guidelines for managing the patient with profound behavioral changes: (1) Avoid centrally acting anticholinergic agents; (2) Begin with lower doses of all drugs and more limited therapeutic goals; (3) Begin therapy with levodopa or amantadine; (4) Avoid bromocriptine; and (5) Avoid neuroleptics if possible; if not, use thioridazine.

Progressive Supranuclear Palsy

Progressive supranuclear palsy is a degenerative disease presenting in later life with features of parkinsonism (akinesia, poor postural reflexes, and fixed facial expression) plus ophthalmoplegia and dystonic rigidity of the neck and trunk. Changes in personality with a degree of apprehension may suggest an agitated depression.¹ Some degree of subcortical dementia is present but is usually mild. As with Parkinson's disease, depression is common.

Gilles de la Tourette Syndrome

Gilles de la Tourette syndrome (GTS) presents in childhood and is manifested by a waxing and waning course of multiple involuntary motor and vocal tics. Although not necessary for the diagnosis, it may include coprolalia (vocalizations of obscenities), copropraxia (inappropriate obscene gestures), echolalia (imitation of sounds or words), echopraxia (imitation of actions), or palilalia (repetition of the patient's own words). Stereotypic movements and obsessive-compulsive behaviors occur with increased frequency with reports varying from 11% to 90%.^{12,47} Other behaviors, which occur in a substantial number of patients, include hyperactivity, attention deficit disorder, learning disabilities, self injury, antisocial behavior, inappropriate sexual activity, and aggression.⁴⁷ It is felt that the initial manifestations of GTS may be behavioral and labeled as minimal brain dysfunction, hyperactivity, or attention deficit disorder.⁴⁷ The etiology of GTS is unknown but the response to haloperidol suggests hyperactivity in dopaminergic systems.

Meige's Syndrome

Meige's syndrome is an involuntary movement disorder characterized by blepharospasm, oral grimacing, and sustained neck contraction.^{46,56} It occurs in the sixth decade and affects women more frequently than men. Associated psychiatric disturbances include depression and obsessive-compulsive symptoms. Unlike hysterical dystonias, which are rare and typically postural rather than action,⁵¹ Meige's syndrome and facial dystonias are believed to be caused by neurotransmitter defects.^{27,55}

TUMORS

Focal expanding lesions, such as tumors, can produce a variety of syndromes including confusional behavior, focal symptoms, dementias, or psychiatric disorders. Psychiatric manifestations of intracranial tumors are reported to occur at some time in one half of patients with brain tumors.⁴⁹ Tumors located in the frontal lobes or limbic system are more likely than parietal or temporal lobe tumors to produce psychiatric symptoms. The type of tumor also will influence the presentation. A meningioma is more likely to produce focal symptoms as it grows extrinsic to the brain and compresses part of the cortex. A glioma, on the other hand, is likely to produce general mental deterioration as it grows slowly through the cortex. Tumors also differ from cerebral infarcts in that the magnitude of symptoms may be far less than what is seen with ischemic lesions of the same size. Confusional behavior is seen more often with rapidly growing tumors, large tumors, or metastatic lesions.

Frontal Lobe Tumors

As many as 88% of patients with primarily psychiatric manifestations of brain tumors had tumors localized to the frontal lobes or limbic system. Symptoms include those suggestive of frontal lobe disease. Bowel and bladder incontinence are present with frontal lobe tumors (35%) and are important clues to the presence of organic disease.

Temporal Lobe Tumors

Temporal lobe tumors frequently present with psychomotor seizures with hallucinations as an ictal manifestation. Both ictal and interictal behavioral changes are clues to the diagnosis of temporal lobe disease. Depression, schizophreniform psychosis and subtle personality changes have been reported in temporal lobe tumor patients.⁵² Localizing features include memory difficulty and aphasia, and when seen with hallucinations, seizures, and psychiatric symptoms, should alert the clinician to suspect tumor in the temporal lobe.

Colloid Cysts

Colloid cysts of the third ventricle may cause psychiatric symptoms presumably as a result of pressure on diencephalic structures and ventricular obstruction with raised intracranial pressure. The most common neurologic manifestations of colloid cysts are positional, intermittent headaches.¹ The range of psychiatric

symptoms reported include depression, emotional lability, hallucinations (olfactory and gustatory), and personality changes.^{16,57}

Although many patients with brain tumors will have some psychiatric symptoms, only a small percentage are hospitalized with a primary psychiatric diagnosis.⁵² It is important to look for organic causes in the psychiatric population but it is also important to do so in a cost-effective manner. Larson et al⁵¹ and Ron⁴⁹ argue that a detailed clinical history and careful neurologic exam are the best indicators of brain pathology. Patients who meet strict diagnostic criteria for psychiatric illness are unlikely to have tumors in the absence of neurologic signs and symptoms. Patients with intellectual deterioration, impaired consciousness, frontal lobe personality changes, incontinence, seizures, or signs of increased intracranial pressure, however, should be imaged.

INFECTIONS

Encephalitis

Encephalitis is a generalized infection of the central nervous system that typically presents as an acute febrile illness, with meningeal irritation, and a combination of delirium, stupor, or focal neurologic signs.

Herpes Simplex Encephalitis

Herpes simplex encephalitis is the most common focal encephalitis. Because of the propensity of the virus to involve the inferomedial portions of the frontal lobes and the temporal lobes, the behavioral manifestations are prominent. These include olfactory or gustatory hallucinations, anosmia, temporal lobe seizures, personality change, and bizarre or psychotic behavior.¹ The morbidity and mortality is high and survivors are frequently left with symptoms from damage to these areas—personality changes, lability, memory loss, or hallucinations.³

Rabies Encephalitis

Rabies virus infections present from 10 days to 1 year from inoculation with restlessness, overactivity, and agitation. Hydrophobia (in 50% of all cases), or the violent terror of water, is the result of severe laryngeal and diaphragm spasm as the patient tries to drink and can be precipitated at the mere mention of water. The disease is invariably fatal in a matter of days.³ Lyssiphobia is the extreme fear of rabies and is reported to occur in a number of individuals with a possible exposure to the disease. Hydrophobia and overactivity may be seen; however, these symptoms continue beyond the expected course of a true rabies infection.³

Neurosyphilis

The incidence of neurosyphilis has declined dramatically since the advent of penicillin; however, there has been a recent increase in the number of cases largely in the AIDS population. The symptoms of neurosyphilis or general paresis are manifestations of frontal lobe disease, which include subtle personality changes, poor judgment, irritability, and lack of interest in personal appearance. With time, patients become more irritable, and experience mood swings, apathy, and disori-

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entation. Delusions of grandeur are present in 10% to 20% of patients.⁵² The typical patient in historical medical literature is one who conceives grand schemes for amassing great power, wealth, or physical prowess, claiming to have powers equal that of a god or king. In reality, megalomania was less common and was present in only 30% of cases. A progressive dementia and physical disability with dysarthria, hypotonia, unsteadiness, and tremor of the tongue and hands will eventually leave the patient bedridden; hence, the term *paretic neurosyphilis*.

Neurologic findings that frequently occur with the behavioral changes should be sought and include Argyll Robertson pupils (pupils that are small, irregular, unequal, and exhibit light-near dissociation), tremor of the tongue and hands, dysarthria, and hyperreflexia. The diagnosis is made by examination of the cerebral spinal fluid, which shows a lymphocytic pleocytosis, elevated protein with a positive VDRL. The response to treatment with penicillin is fairly good if treated early in the course of the disease.

Chronic Meningitis

Organisms such as *M. tuberculosis*, *Cryptococcus*, *Coccidioides* and other fungi can produce indolent infections in the central nervous system with prominent behavioral symptoms. The patient is typically an immunosuppressed patient who develops a chronic headache, memory difficulties, and confusion. Fever is often present, but the presence of other neurologic signs is variable. At times these new behavioral symptoms may be attributed to the primary disease, and the possibility of a superinfection is overlooked. All are potentially treatable and should be considered.⁵²

Subacute Sclerosing Panencephalitis

Subacute sclerosing panencephalitis is a slow, progressive inflammatory disease of childhood or early adolescence that affects males three times more frequently than females, and usually follows measles infection or vaccination. It typically begins with behavior changes of disobedience, temper outbursts, distraction, social withdrawal, sleeplessness, and hallucinations. There are few neurologic signs at this stage and symptoms may be attributed to psychologic upset. Myoclonus, ataxia, seizures, and intellectual deterioration ensue and the eventual outcome is coma and death in 1 to 3 years.³

NEURODEGENERATIVE DISORDERS

Numerous degenerative disorders that ordinarily occur in childhood may rarely present as dementia or psychosis in adults. See Table 8 and an excellent review by Coker.⁹

METABOLIC CONDITIONS

Thyroid Disease

Confusional behavior or psychiatric symptoms of anxiety, or an agitated depression are common in hyperthyroidism.

Hypothyroidism, or myxedema, can present as a dementing illness. "Myx-

Table 8. CHILDHOOD DEGENERATIVE DISEASES THAT MAY PRESENT AS DEMENTIA OR PSYCHOSIS IN ADULTS

Neuronal intranuclear inclusion disease
Alexander's disease
Lafora's disease
Kuf's disease (neuronal ceroid lipofuscinosis)
Cerebrotendinous xanthomatosis
Polycystic lipomembranous osteodysplasia with sclerosing leukoencephalopathy
Adrenoleukodystrophy
Gangliosidosis 1 - type III
Gangliosidosis 2
Gaucher's type I
Niemann-Pick II-C
Mucopolysaccharidosis III-B (Sanfilippo's disease)
Mitochondrial disorders (MERRF, MELAS)
Metachromatic leukodystrophy
Wilson's disease
Fabry's disease
Krabbe's disease (globoid cell leukodystrophy)

From Coker SB: The diagnosis of neurodegenerative disorders presenting as dementia in adults. *Neurology* 41:749-790, 1991, with permission.

edema madness", was coined by Asher in 1949 to describe this psychiatric syndrome, which frequently includes paranoia and depression, but also hypomania, schizophreniform psychosis, and slow thinking.⁵² Acute confusional states with hallucinations also may be seen. Patients typically show characteristic signs of hypothyroidism (weight gain, deep, coarse voice, thin dry hair, loss of the lateral eye brow, facial puffiness, cold intolerance, and hearing difficulty). Ten percent of patients will have persistent intellectual deficits after treatment, and confusional and psychotic behavior has been reported as an adverse reaction to hormone replacement.⁵²

Pituitary Disease

Hyperfunctioning of the pituitary gland can lead to adrenal hyperplasia and Cushing's syndrome. Up to 70% of these patients will exhibit behavioral symptoms, including easy fatigability, irritability, depression, mental dullness and memory impairment. The characteristic signs of steroid excess, weight gain, moon facies, abdominal striae, and acne are usually present and aid in the diagnosis.

Hypopituitarism can present with a slowly progressive dementia with signs of mental slowness, depression, and emotional changes. The typical clinical setting for this disease is in the postpartum female, secondary to hemorrhage into the pituitary gland (Sheehan's syndrome).

Calcium Metabolism

Hypercalcemia typically presents as an acute confusional state but chronic behavioral changes also can be seen. Personality changes, loss of spontaneity and initiative occur in 60% of patients and cognitive difficulties occur in 20%.⁵² Normalization of serum calcium reverses the symptoms even in long-standing cases.

Hypocalcemia can present as a confusional state in the absence of tetany, depending on the rate of decline in serum calcium levels. Gradual decline in calcium produces a syndrome of more subtle personality changes. Cataracts, seizures, extrapyramidal symptoms, and signs of increased intracranial pressure may be seen with hypoparathyroidism, the most common cause of hypocalcemia. Patients may be left with permanent intellectual deficits even after normalization of serum calcium levels.

Porphyrias

The porphyrias are metabolic disorders of heme biosynthesis in which there is excessive accumulation of porphyrins. The triad of acute colicky abdominal pain, predominantly motor polyneuropathy, and psychosis constitute the disease known as acute intermittent porphyria (AIP). It is inherited as an autosomal dominant disease with predilection for women. The usual age of onset is from 20 to 50 years old; however, childhood cases have been reported.⁶

Psychiatric symptoms range from anxiety and insomnia to emotional lability, depression, or frank psychosis.^{38,58} The overall prevalence of AIP in a chronic psychiatric population was 0.21%.⁵⁴ Patients with AIP were diagnosed as having atypical psychosis or schizoaffective disorder. None had the classic triad of AIP, suggesting that chronic psychiatric disease may be more common in AIP than previously reported and may be the only manifestation of the disease.⁵⁴

VITAMIN DEFICIENCIES

Niacin Deficiency

Pellagra is rarely seen because breads are now enriched with niacin. It may still occur in alcoholics and vegetarians of underdeveloped countries. The earliest manifestations are cerebral and include apathy, irritability, insomnia, or fatigue and depression. An acute confusional psychosis may be rarely present. This psychosis is followed shortly by dermatitis, peripheral neuropathy, and diarrhea.

Vitamin B₁₂ Deficiency

Deficiency of vitamin B₁₂ causes megaloblastic anemia and central nervous system defects such as paresthesias, spinal cord syndrome, and optic neuropathy. Neuropsychiatric manifestations of cobalamin deficiency can be present in the absence of anemia or macrocytosis.³⁵

Mental signs includes irritability, and emotional instability progressing to depressive psychosis, and intellectual deterioration.¹ Patients at risk for developing a deficiency state are those with a history of gastric surgery, malabsorption, or strict vegetarians. Clinically suspected cases in which the vitamin B₁₂ level is normal should receive a therapeutic trial of vitamin B₁₂ or measurement of methylmalonic acid and total homocysteine (both metabolites) which will be increased.³⁵ Behavioral symptoms are reversible if treated early in the course of the disease.

Thiamine deficiency is covered in the article on neurologic complications of alcoholism elsewhere in this issue.

TOXINS

Mercury

Chronic exposure to mercury causes two distinct syndromes, depending on the type of mercury. Exposure to *inorganic mercury* at the turn of the century led to a distinct psychiatric syndrome characterized by the "Mad Hatter" in *Alice in Wonderland*. Psychiatric symptoms of severe depression, irritability, and florid psychosis are commonly seen.⁵² Less prominent symptoms are headache, tremor, and weakness. Current sources include exposure during the manufacture of thermometers, mirrors, incandescent lights, and x-ray machines.

Outbreaks of *organic mercury* poisoning have occurred from consumption of contaminated fish in Minimata, Japan, and by ingestion of grain contaminated with methylmercuric fungicide in farming communities in Iraq.³⁶ Methylmercury or organic mercury poisoning causes more of a neurologic syndrome with motor-sensory neuropathy, cerebellar ataxia, slurred speech, paresthesia, and visual field constriction. Depression, irritability, and mild dementia are the primary behavioral manifestations.

Thallium and Arsenic

These are known to cause delirious states. An update on delirium is covered elsewhere in this issue.

SUMMARY

Neurologic disease can present as a psychiatric disorder. Understanding underlying neuroanatomic function helps physicians to localize defects and search for treatable neurologic conditions.

Neurologic conditions such as Huntington's chorea, Wilson's disease, Gille de la Tourette syndrome, brain tumors, encephalitis and meningitis, neurodegenerative conditions and metabolic or toxic conditions can have psychiatric manifestations.

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