THE FUNDAMENTALS OF CLINICAL NEUROPSYCHIATRY

MICHAEL ALAN TAYLOR

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To Max Fink, who got this all started years ago;
to residents whose interest and questions keep it going;
and to patients and their good care, who, after all,
are the reason for it all

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Preface

In the past 20 years psychiatry and behavioral neurology have experienced an information explosion. Textbooks about general psychiatry, neuropsychiatry, and related topics have grown in number and size. Some of these books are excellent, and I often make reference to them. Their wealth of information, however, is also their limitation. Experienced and inexperienced clinicians alike are faced with almost daily new revelations about neurotransmitters. They are barraged with study after study reporting startling findings of brain dysfunctions in many psychiatric conditions, novel drugs of all sorts (atypical antipsychotics, specific reuptake-inhibiting antidepressants, autoreceptor enhancers), and new relationships (co-morbidities) found between disorders that make each new diagnostic system outdated in some areas before publication. Synthesis is needed. We need to make sense of it all.

Over the years I have been forced to try and do that in my lectures, seminars, and teaching rounds. Most students and residents are as well read as I and do not need a recitation of what is in reference textbooks. They need some guidance in how to put it all together and then to be taught the principles of diagnosis and treatment so that they can use the latest information in a reasoned way when taking care of their patients. I have tried to write that book.

Because students and residents, and most of us, learn best from plain speaking, I have also tried to make the writing less formal than the typical textbook, and I use language as I might on rounds or in a seminar. I begin each topic with the basics and then build the complexity upon that. I emphasize the fundamentals of how the brain is organized to generate behavior (its main *raison d'être*) and the principles of evaluation and treatment.

Because I am not a renaissance man, I have written about things I know something about and that reflect my clinical experience. If a reader finds some glaring omission (there is no chapter on child psychiatry, for example) it is not missing because I do not think the topic important, but rather because it is best left to those who have had to make sense of that topic in the care of their patients and in their teaching of students and residents.

Although I incorporate *Diagnostic and Statistical Manual of Mental Disorders* (DSM) terminology and classification into the discussion of many syndromes, the DSM is imperfect, and I organize some conditions from a different perspective. For example, I include some of the DSM impulse control disorders (e.g., trichotillomania, kleptomania) with obsessional syndromes because most research points to that association, and when treated as having a variant of obsessive compulsive

disorder patients with these conditions often get better. I have included dissociative disorders with epilepsy because these clinical features often co-occur, and many documented dissociative states result from a seizure disorder. In Chapter 16 I include conditions that do not have common pathophysioliogies, but that do have similar diagnostic challenges. Although each condition deserves a book of its own, I discuss them briefly because their omission would be glaring in a general book about neuropsychiatry.

The old saying "those who can, do; those who can't, teach" certainly does not apply to clinical medicine. The best teachers in my experience are those who have something to teach, have a passion for it, and are themselves learning daily from their patients. I have had the added advantage of learning from my colleagues, residents, and students. If this book even approximates that experience, I'll consider it a success.

Neuropsychiatry provides the perspective that has made most sense to me in my efforts to synthesize the neurosciences and clinical psychiatry. Neuropsychiatry is now a major theme in psychiatry, and there is now widespread recognition that many patients with major psychiatric disorders (e.g., the DSM's axis I conditions) have something wrong with their brains and that the necessary element of treatment for these patients is biological. It is also now widely recognized that behavioral change is a common expression of many general medical conditions (e.g., hyperthyroidism, hypertension) and of what is still called *neurologic disease* (to artificially separate it from mental illness). Indeed, the main premise of this book is that psychiatry and neurology are one field; that is, in its broadest sense, neuropsychiatry is the theoretical and practical approach to psychiatric patient care that recognizes the brain as the organ of the mind and that mental illness is, in fact, not "mental" at all, but the behavioral disturbances associated with brain dysfunction and disease.

Because of this broad view of neuropsychiatry, I include discussions of "classic psychiatric" disorders (e.g., depression, obsessive compulsive disorder) along with traditional "neurologic" conditions (e.g., stroke, headache). To me they all represent dysfunction of the brain. I have also included a long chapter on personality because there is a neurology underlying personality just as there is a neurology underlying all behavior, no matter how complex, and because understanding personality is important in providing good patient care.

This broad view of neuropsychiatry leads to the logical conclusion that psychiatry and neurology are parts of the same thing. No matter where you start, you end up at the same place; abnormal behavior results from brain dysfunction and brain dysfunction leads to abnormal behavior. For example, if you examine "neurologic " patients with brain diseases, you find that the most common expression of that disease is behavior change, not an abnormal reflex or other "neurologic" signs. This is true for disease almost anywhere in the brain, but it is particularly true when disease affects anterior brain circuits (prefrontal cortex, basal ganglia, thalamus) or the limbic system (particularly the amygdala and hippocampus). The accompanying table displays some conditions affecting anterior brain circuits and their commonly observed behavioral changes. The striking thing about these

Some Behavioral Syndromes Associated With Anterior Brain Lesions

Condition	*Personality*	*Mania*	*Depression*	*Obsessive Compulsive Disorder*
		FRONTAL CORTEX		
Dorsolateral prefrontal cortex trauma	Yes	No	Yes	No
Orbitolateral prefrontal cortex trauma	Yes	Yes	No	No
		BASAL GANGLIA		
Parkinson's disease	Yes	Yes	Yes	No
Huntington's disease	Yes	Yes	Yes	Yes
Stroke	Yes	Yes	Yes	Yes
Carbon monoxide poisoning	Yes	No	No	Yes
		THALAMUS		
Stroke	Yes	Yes	No	Yes

relationships is that it is not the pathophysiology that is most important, but rather the area of the brain that is involved. Anterior brain systems have more to do with behavior than with anything else. Disease in this system almost always produces behavioral change, and most commonly that behavioral change is a disturbance in mood and affect or in personality.

A reasonable alternative interpretation of the association between the diseases in the accompanying table and depression is that "who wouldn't feel depressed with a disease like that?!" However, of the 30% of Huntington's disease patients who become depressed, most do so *before* they know they have the condition. Among the 50%–70% of Parkinson's disease patients who become depressed, there is little correlation between the degree of neurologic impairment and the degree of the depression. Only convoluted theorizing could try to explain why the same group of illnesses can also cause an elevated mood as in mania or why obsessive compulsive disorder apperars more likely if the nondominant basal ganglia are involved and depression more likely when the dominant basal ganglia are involved.

In addition to brain lesions producing syndromes we usually think of as "psychiatric" (e.g., depression, obsessive compulsive disorder), brain lesions also can produce classic psychopathology. The following vignettes from Macdonald Critchley's famous monograph on the parietal lobe* illustrate this point:

*Macdonald Critchley, *The Parietal Lobes*, Hafner Press, New York, 1953.

> "I had a terrible shock this morning when I touched my left hand; I thought it was the head of a reptile." Asked where her left arm was, she said: "I don't know. Where is it? I don't feel it." When confronted with her left hand, she said: "That is someone else's hand." "Whose is it?" "It is not mine. It's a reptile."
>
> One patient insisted that a board had been inserted in place of the left side of the trunk and the left limbs. Of her left limbs, she said: ". . . that's an old man who stays in bed all the time." Asked if she had any objection, she replied: "Yes, I don't want any spirits in bed with me." A second patient thought her daughter was in bed with her because there was a strange arm across her chest.

In both vignettes, the patient expresses a delusional idea based on an experience that a body part actually belongs to another creature (a reptile) or to another person. These are *experiences of alienation* and in the psychiatric literature are termed *first rank symptoms* (see Chapter 3). They are common in several psychoses. Without specific evidence that the above patients had suffered nondominant parietal lobe strokes, they surely would have been hospitalized in a psychiatric hospital and labeled "psychotic." Only their other "neurologic" features led to the recognition that they suffered strokes and were "neurological."

The artificial nature of the separation of neurology and psychiatry is also illustrated by what follows in this book: Many patients with classic, primary psychiatric disorders, nevertheless, show evidence of brain disease or dysfunction. For example, schizophrenics have enlarged cerebral ventricles and cerebral cortical atrophy. They, and manics, and many depressed patients have decreased cerebral metabolism. Patients with obsessive compulsive disorder have visuospatial and visual memory problems. These and patients with other psychiatric conditions are as much "neurologic" as are Critchley's stroke patients. The neurology is just different.

Chicago, Ill. M.A.T.

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Brain Organization and Neurobehavioral and Neurocognitive Function

The functional organization of the brain has specific diagnostic and treatment implications. Chapters 3 and 4 describe specific behavioral and cognitive assessments of this organization. Three overlapping frameworks of brain functional organization are

1. *Left and right systems* motorically controlling and perceptually responding to the contralateral side of the rest of the body and two different information-processing styles and patterns of cognitive specializations of the dominant and nondominant cerebral hemispheres
2. *An action brain system* (anterior brain structures and the cerebellum-pons) and a *perceptual-integrating brain system* (temporo-parietal-occipital cortices and their related subcortical structures)
3. A brain stem to neocortical *tripartite phylogenetic system* (old vertebrate, social mammal and primate, and uniquely human structures)

Left and Right Brain Systems

Hemisphere Specialization

The cerebral hemispheres in humans have different specializations and information processing styles (Table 1.1). Knowing these different processing styles helps determine the side of a brain lesion because the different processing styles and specializations affect behavior differently. A helpful metaphor for hemisphere specialization is a train station. Think of the left hemisphere as a train station with one track, so that only one train at a time can enter the station. This forces the left hemisphere to look into each train car (in which metaphorical information is

TABLE 1.1. Information Processing Styles of the Left and Right Cerebral Hemispheres

Left	*Right*
1. Sequential processing	1. Parallel processing
2. Analytic, syllogistic	2. Intuitive, impulsive, emotional, rapid scanning, holistic
3. Focal and discreet	3. Diffuse
4. High frequency	4. Low frequency

being carried), one car at a time. Tedious and focused, the left hemisphere is very good at detail and is able to analyze and make inferences from that information. For example, if car one has 2 bits of information and car two 4 bits, the functioning left hemisphere will reason that car three is likely to have 6, 8, or 16 bits and, based on what that car actually has in it, the left hemisphere will then know the rule (adding twos, doubling or squaring the number). In contrast, the right hemisphere is like a train station into which several trains can enter simultaneously. Although the right hemisphere can be forced to look at each car in the sequential fashion of the left, it is not good at this. It is best at rapid scanning of information to detect broad patterns, or gestalts (for example: the trains are coming in fast or slowly or there are many trains, or only a few).

The train station metaphor suggests the two hemispheres are constructed ("hard wired") differently (Table 1.2), and there are left–right cerebral hemisphere differences consistent with the left being a discrete processor suited for language-related information (e.g., specific structures are larger) and the right hemisphere being a holistic processor of broad patterns of information (e.g., uniformly more white matter and dendritic communication).

Because of their structural differences, each hemisphere focuses on different physical attributes of the information being processed. For example, Figure 1.1 shows the letter H in high and low frequency forms. High frequency refers to dense information (or signal) per unit time or area; low frequency refers to less information per unit time or area. Although the letter H is a language symbol and should be processed better by the hemisphere organized for language (usually the left), this is only true when the H is presented as a high frequency stimulus. When presented as a low frequency stimulus, it is processed better by the right hemisphere because it is visually more ambiguous, and might represent several different things e.q. a goalpost or a street map. Figure 1.2 shows this even more dramatically. Information can be initially presented to only one hemisphere. This is done with an instrument termed a *tachistoscope*, which flashes stimuli into one or the other hemivisual fields. Because of the visual system's organization, left hemivisual field information goes to the right hemisphere first, and right hemivisual field information goes to the left hemisphere first. When information like that in Figure 1.2 is presented to each hemisphere, the left "sees" H, the high frequency stimulus, while the right "sees" S, the low frequency stimulus. Each hemisphere

TABLE 1.2. Anatomic Cerebral Hemisphere Asymmetries

Favoring Left	*Favoring Right*
Cerebral hemisphere larger and denser	Larger frontal area
Larger planum temporale (the posterior roof of the temporal lobe and an extension of Wernicke's area).	Greater dendritic arborization
Longer sylvian fissure and lateral ventricle	Relatively more white matter
Relatively more gray matter	Greater concentration of serotonergic neurons
Larger posterior temporal/parieto-occipital regions	
Greater concentration of dopaminergic neurons	

also differentially responds to other physical attributes of stimuli (e.g., luminescence, geometric shape, linearity). Because language and symbolic related stimuli are loaded with high frequency physical attributes, the left hemisphere, hard wired to handle the high frequency physical attributes of information, is activated when presented with such stimuli, and processes them, and so we think of it as "the language hemisphere."

In the real world, stimuli include both high and low frequency information, and the two hemispheres work together. As you read this book your nondominant hemisphere is processing the patterns. You can tell at a glance, even if you turn the book upside down, that the page has printed letters on it that form words and that the type of lettering is immediately recognizable as a Western European language. From the big picture provided by the nondominant hemisphere, the dominant hemisphere deals with the details, and you "read" what is written. The corpus callosum integrates these different forms of information processing, as well as being an interhemispheric communication bridge. For example, lesions in

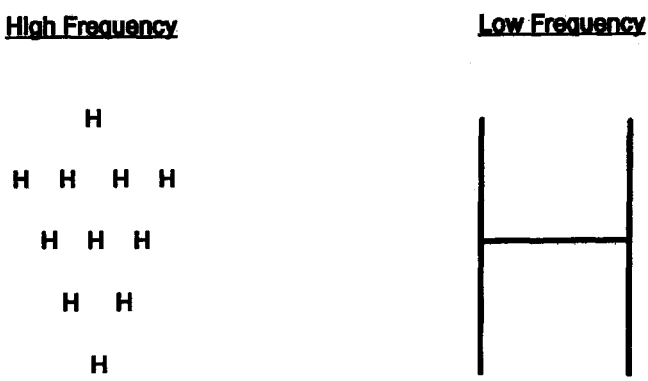

Figure 1.1. The frequency of the stimulus determines which hemisphere best processes it.

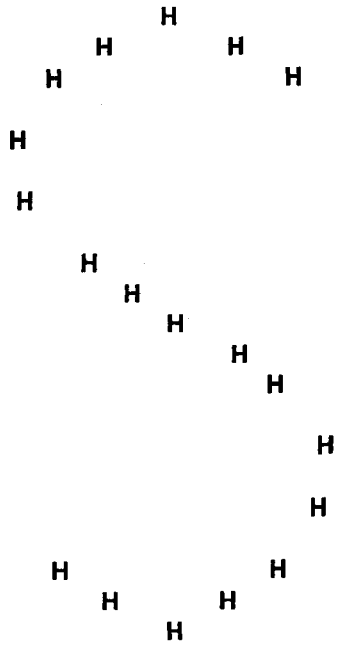

Figure 1.2. The left hemisphere processes the high frequency information and "sees" H. The right hemisphere processes the low frequency information and "sees" S.

the anterior corpus callosum can disconnect lexical language from the emotional content of language leading to a loss of emotional expression while reading aloud and speaking, but spontaneous emotional expression remains intact.

Although most people are left hemisphere dominant for language and right hemisphere specialized for visuo-spatial function (Table 1.3), specific language and nonlanguage tasks are variably lateralized in the same person. Thus, when trying to lateralize a lesion, always look for the pattern. Do not hang your diagnostic hat on one cognitive or behavioral peg. Other chapters describe the behaviors of dominant and nondominant cerebral hemisphere lesions in detail. Some classic dominant hemisphere syndromes are Broca's and Wernicke's aphasias and Gerstmann's syndrome. Some classic nondominant hemisphere syndromes are motor and receptive aprosodia, left spatial neglect, and misidentification syndromes (Capgras and Fregoli's).

Handedness

Primates prefer using one hand over the other for fine motor tasks. The preference distribution runs 50–50. Ninety percent of humans, however, primarily use the right hand for fine motor tasks. Hand preference roughly correlates with cerebral hemisphere organization for language. Assess each patient for handedness when looking for localized brain lesions.

TABLE 1.3. Some Specific Functions of the Left and Right Cerebral Hemispheres

Left	*Right*
1. Language	1. Visuospatial
2. Verbal learning and memory	2. Visual learning and memory
3. Verbal reasoning and planning	3. Nonverbal reasoning and planning
4. Complex fine motor function	4. Facial recognition

Determine handedness by asking patients to demonstrate the use of objects and by writing their name or a sentence. Patient self-report is not reliable (i.e., which hand they say they use for a task is not always the hand they actually use). Demonstrations include using a knife to cut bread, pouring liquid from a bottle, threading a needle, and brushing one's teeth. Pure right handers consistently do these things and write with the right hand. Their language hemisphere is the left. Pure right-handed women have more bilateral representation of cognitive functions than do men, but they are also primarily left hemisphere "dominant" for language. Among left handers and persons with mixed handedness (i.e., they do several things with each hand) there is less certainty which hemisphere is dominant for language. The odds, however, still favor the left. Combined with an assessment of behavior and cognition, knowing the handedness of a patient helps in lateralizing lesions to the right or left.

Because only 10% or so of the population is left handed, left handers are deviant. For some left handers this deviance is familial and may be associated with creativity and better performance on some cognitive tests. For example, there is a disproportionately high number of left handers among persons with PhDs. For other left handers, their deviance may reflect prenatal or perinatal neural developmental problems. Thus, left handedness is also associated with developmental disorder, alcoholism, bipolar mood disorders, and numerous other neurologic conditions.

Eye and foot preferences are not correlated with cerebral organization. Nevertheless, children with mixed eye, foot, and hand preferences (e.g., right or mixed handed, left footed, right eyed) are more likely to have behavioral and school problems.

THE PERCEPTUAL-INTEGRATING BRAIN

The temporal, parietal, and occipital lobes of the cerebral hemispheres and related subcortical structures organize and make sense of the world around us. The perceptual-integrating brain may also subserve consciousness because it recognizes the distinction between ourselves and the external world—literally where we end and where the rest of the world begins. The perceptual-integrating brain tells us where we are in three-dimensional space vis à vis other objects and then

stores this information as memory. Alzheimer's disease is a classic disease of the perceptual-integrating brain.

Parietal Lobes

The parietal lobes are roughly divided into two functional units: an anterior unit (the primary sensory cortex and its unimodal sensory associational cortex) *and a posterior heteromodal association unit (the inferior parietal lobule including the angular and supramarginal gyri, and the superior parietal lobule). The parietal lobes function as the primary and associational cortices of the thalamus.

Parietal lobe functions are (1) awareness of three-dimensional space and the relationship of the self to that space; (2) integrating semantic and visual sensory stimuli (e.g., vision to language); (3) linking somatosensory perception to motor performance and guidance of motor behavior in three-dimensional space; (4) manipulating spatial coordinates of abstract stimuli (e.g., mathematics); and (5) focused attention. Specifically, the parietal lobes are needed when we do calculations, simple motor and constructional tasks, read aloud, locate objects in space, pay attention to areas of the space surrounding us, and make abstract constructions.

Occipital Lobes

The occipital lobes process movement, color, and shape and begin organizing visual stimuli. The occipital lobes have no heteromodal cortex.

Temporal Lobes

Temporal lobe functions are (1) auditory sensory processing (recognizing and understanding speech and environmental sounds); (2) auditory and visual perception (facial recognition, reading); (3) providing affective tone (emotions) to sensory input; (4) new learning and long-term storage of sensory input (memory); and (5) triggering flight/fight behaviors and their physiologic changes. Specifically, the temporal lobes function in understanding speech, recognizing environmental sounds, generating emotion, learning new things, storing old perceptions and experiences for future reference, focusing our attention on sound, recognizing danger or potential danger, and initially and rapidly responding to danger.

Thalamus

The thalamus is the "pentium chip" of the brain. All somatosensory routes to the cerebral cortex pass through the thalamus. The thalamus (1) directly relays somatosensory information from the external world to the appropriate parietal primary sensory cortical homunculus so that body image is maintained; (2) through feedback projections, synchronizes visual and auditory information with the rest

*See below for details of cerebral cortex organization.

of the perceptual-integrating system; (3) organizes all sensory information so that, rather than experiencing the world in parts, we perceive it as an integrated whole; (4) integrates sensory information in conjunction with a special heteromodal cortical area in the temporal lobes (near the insula); and (5) provides tone to the frontal circuits and modulates attentiveness, wakefulness, and sleep.

Sensory experience is not a kaleidoscope but an integrated experience. The feel and weight of this book, the temperature of the room you are in, the ambient noise and light, the words on the page each are independently delivered to its respective primary sensory cortex. Because of integrating oscillations of the thalamus, and the further integration within the special heteromodal cortical area in the temporal lobes, you experience the entire integrated scene you are now in.

The thalamus is part of the perceptual-integrating brain *and* the action brain (see below). It links the posterior part of the action brain (the cerebellar-pontine unit) to the anterior part (the frontal loops). It is the feedback unit of the five left and five right functional frontal lobe circuits described below. Because of its connections to the reticular activating system it provides tone to the frontal circuits. It is one of the frontal circuitry connections to the limbic system.

Thalamic lesions can cause (1) frontal lobe syndromes (particularly avolition and eye tracking problems); (2) disturbances in emotion; (3) perceptual disturbances of disintegration (including hallucinations); (4) speech and language problems; (5) coordination and balance problems; (6) analgesia, hyperesthesia, and pain syndromes; (7) disturbances in body image; and (8) parietal lobe features (e.g., Gerstmann's syndrome, astereognosia).

Thalamic stroke and its effect on speech and language is a model for formal thought disorder, a classic feature of schizophrenia. Because of its multiple and integrating functions, the thalamus has been implicated as a major site in the pathophysiology of schizophrenia.

The Action Brain

The perceptual-integrating brain makes sense of the world. The action brain responds. The action brain roughly includes the frontal lobes and their related subcortical structures, the cerebellum and pons, and the motor system between these anterior and posterior units. Inattention and *modest* altered arousal (low or high) have only moderate impact on the perceptual-integrating brain. Even small problems with attention and arousal can adversely affect action brain functioning. Structures of the limbic system involved in emotional expression and memory recall (e.g., amygdala, anterior hippocampus) can also be considered part of the action brain. The basic functions of the action brain are generating ideas, problem solving, emotional expression, and motor behavior (including speech).

The action brain also performs "executive" functions. These include planning, initiating actions, monitoring actions and self-correcting, verifying the planned action is being carried out correctly, and terminating the action when it is completed. Working memory (the brief holding of information in your mind's eye while you do something with that information, e.g., dial a phone number just

said to you), recall of memory, and willed action are also basic to action brain functions.

The anterior part of the action brain is organized into five left and five right parallel circuits that can operate independently of each other. Figure 1.3 illustrates a generic anterior circuit. Information in the circuit only flows in one direction. The limbic system has its own frontal circuit (the anterior cingulate circuit) and also connects to the other parallel circuits via the globus pallidus and striatum. Thus, the action brain is greatly influenced by emotion. The prefrontal cortex also interacts with other heteromodal cortices via cortico-cortical fibers. The five parallel circuits are

1. *The motor circuit*: The primary and secondary motor cortex (areas 4, M2-medial surface anterior to area 4), the supplementary motor area (area 6), and some somatosensory fibers send afferents to the putamen that in turn sends information to the globus pallidus and substantia nigra. The globus pallidus sends information to the thalamus, which sends feedback to the primary and secondary motor cortices. Deficits in this system cause problems with sequential movement, movement initiation, self-correction, and learning of new motor sequences (particularly when area 6 is involved). Schizophrenics have problems with these functions.
2. *Oculomotor circuit*: The frontal eye fields and prefrontal cortex project to the caudate, globus pallidus, and substantia nigra. The globus pallidus then sends information to the thalamus,which sends feedback to the cortical areas that began the circuit. Lesions in this circuit can result in deficits in eye-tracking of moving objects. Schizophrenics and some of their first-degree relatives have eye-tracking problems.
3. *Dorsolateral prefrontal circuit*: Brodmann areas 9 and 10 project to the head of the caudate. The caudate connects to the globus pallidus and

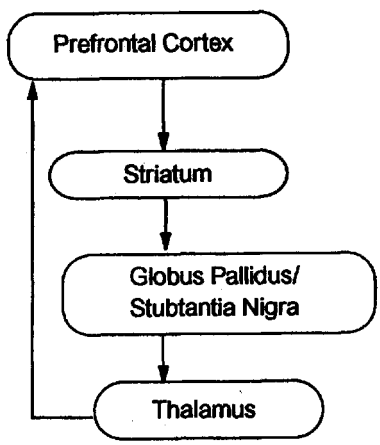

Figure 1.3. Anterior "action" brain parallel loop.

substantia nigra. The globus pallidus sends information to the thalamus, which sends feedback to the dorsolateral prefrontal cortex. Lesions in this circuit cause deficits in executive function. Patients cannot generate new ideas or shift cognitive set. They have no fluency or flexibility of thought. They have problems with organizational strategies for learning, poor construction strategies, and problems with motor programming (alternating and reciprocal motor tasks). The classic syndrome is termed the *dorsolateral frontal lobe syndrome,* and behaviorally these patients are avolitional (see Chapter 3). Chronic schizophrenics also have this syndrome. Pick's disease begins with this syndrome.

4. *Lateral orbitofrontal circuit*: The inferior lateral prefrontal cortex (Brodmann area 10) projects to the caudate. The caudate connects to the globus pallidus and substantia nigra with subloops to the globus pallidus externa and subthalamic nucleus. The globus pallidus and substantia nigra connect to the thalamus, which sends feedback to the lateral orbitofrontal cortex. Lesions in this circuit may lead to the classic *orbitofrontal (or disinhibition) syndrome* characterized by irritability, Witzelsucht (a silly, shallow mood), a coarsening of the personality with loss of social graces and tactlessness, echo phenomena,* and utilization behavior (they see an object and must touch it and use it). Chronic and atypical manics have this syndrome.
5. *Anterior cingulate circuit*: Brodmann area 24 projects to the striatum, nucleus accumbens, and the olfactory tubercle. The ventral striatum (or limbic striatum) also receives fibers from the hippocampus, amygdala, and entorhinal and perirhinal cortices. The ventral striatum then projects to the globus pallidus and substantia nigra. These connect with the subthalamic nucleus, ventral tegmental area, habenula, hypothalamus, amygdala, and thalamus. The thalamus then sends feedback to the anterior cingulate cortex. Lesions in this circuit can produce akinetic mutism, stupors with incontinence, and profound generalized analgesia. Catatonic patients can have these features.

Basal Ganglia

The basal ganglia form an integral unit of the frontal parallel circuits. They also have afferent and efferent connections with the limbic system. If the thalamus is the "pentium chip" of the brain, the basal ganglia is the crossroad of the brain. The frontal circuits and limbic system send information to it, and it sends feedback information to these systems. The brain's mesolimbic dopaminergic reward system (see Chapter 13) connects to it. The basal ganglia (1) fine-tune motor behavior and provide procedural memory programs of learned motor sequences (like riding a bike) to the frontal lobes; (2) help in self-monitoring of speech dur-

**Echolalia,* the patient repeating your words; *echopraxia,* the patient repeats your movements. These are also catatonic features.

ing conversation; (3) participate in hand and facial expression during conversation and spontaneous emotion; and (4) participate in cognitive tasks (e.g., attention, working memory, new learning; particularly sensorimotor and visuomotor sequencing).

Classic basal ganglia syndromes include Parkinson's and Huntington's diseases. Obsessive compulsive disorders and Gilles de la Tourette's syndrome also involve the basal ganglia. Disease of the basal ganglia typically leads to problems with *m*otor function, *m*ood, and *m*emory. The basal ganglia are the 3M Company of the brain.

Cerebellum

The cerebellum and pons are part of the motor system and, therefore, part of the "action" brain. This "unit" works in concert with the prefrontal cortex. There is direct functional reciprocal interaction between contralateral cerebellar and frontal neocortices. Anatomic connection is via corticopontine and cortico-olivary projections and lateral thalamic nuclei. Vermal connections to the thalamus are ipsilateral.

The cerebellum functions beyond motor coordination. It also modulates limbic system activation. This is an important consideration in diagnosing a person with what appears to be psychosensory (partial complex) epilepsy with irritability (see Chapter 10) because although the patient's symptoms may at first suggest a lesion in the temporal lobes, the pathology may actually be in the cerebellum. Your assessment of cerebellar motor signs helps differentiate these possibilities.

The cerebellum is also involved in (1) motor planning: trial and error planning with a motor output, (e.g., learning carpentry, solving a puzzle that requires moving objects into a pattern); (2) new learning: procedural motor responses that can be rapidly and automatically begun without having to "think" about it (e.g., learning martial arts, dance steps); and (3) speech and language: using action verbs, helping organizing motoric language

Cerebellar lesions can result in (1) loss of learned movement sequences (e.g., knitting, playing a musical instrument); (2) the inability to learn new movement sequences (practice does not help); (3) deficits in the ability to be classically conditioned (the conditioned stimulus—e.g., a tone is linked to an unconditioned stimulus such as a puff of air to the cornea that causes eyeblink, so that after a while the tone produces the blink); and (4) speech problems in which verbs are no longer used properly (incorrect tense, e.g., "I wanted to going to the store") and fluency is impaired (trouble linking action verbs and nouns).

THE TRIPARTITE PHYLOGENETIC SYSTEM

Almost 30 years ago Paul McLean described the triune brain: an old reptilian brain, a newer paleomammalian system, and the new primate neocortex. Each of these systems has its neuroanatomy and characteristic behaviors. Compared with our closest primates, the human brain differs most in the size of the prefrontal

cortex, the parietal lobe heteromodal cortex, and the neocortex of the cerebellum. Although oversimplified, McLean was making the following points:

1. Human behavior and the brain systems that subserve it are products of human evolution. A full understanding of human behavior is within an evolutionary context.
2. Evolution is a great conservator; it typically does not discard structures, it reuses them in modified ways.* Thus, human behavior includes aspects of our phylogeny.
3. Human behavior is not simply a function of the neocortex; subcortical structures and older cortex also play major roles in generating human behavior.

The left–right view of brain behavior relationships has heuristic value, but it is two-dimensional. When put together with the tripartite, phylogenetic three-dimensional view of the brain, the combined concepts tell us that lateralized cognitive function (e.g., the "left brain" does this, the "right brain" does that) is limited. The left and right sides of the oldest parts of our brains do not process information differently and do not have specialized "talents." Only the neocortex and specific nuclei in some subcortical structures (in the basal ganglia and thalamus) are lateralized this way. This understanding of the limits of lateralization helps when you try to localize lesions or hypothesize the pathophysiology of a behavioral syndrome.

The Reptilian Brain

Many parts of our brains have changed very little from those of reptiles. The anatomy of this system is brain stem, related vestibular system, thalamus, hypothalamus, basal ganglia, some forebrain nuclei, the amygdala, and the hippocampus.

This system is primarily responsible for arousal, activation, balance, information input and output, homeostatic and procreative drives, and flight/fight. Lesions in this system have generalized cognitive and behavioral effects, usually due to problems with arousal. Lesions in this system (usually bilateral) can result in stuporous states, catatonia, and anxiety disorders.

The Paleomammalian Brain and Limbic System

This system is primarily the limbic system, and it phylogenetically develops in earnest among social mammals where subtleties in emotion and good memory are helpful in maintaining the social structure of a relatively small group of individuals (as opposed to a herd of thousands). The anatomy of this system is the mammillary bodies, fornix, corona radiata, hippocampus, parahippocampus,

*For example, the basal ganglia participate in flight/fight motor actions (reptilian), fine tune hand and facial movement and expression (primates), and are involved in conversational speech (humans).

amygdala, hypothalamus, entorhinal cortex and pathways, septal nuclei, and the cingulate gyrus. The limbic system modifies appetitive drives and flight/fight. It generates emotion and provides emotional tone to perceptions. It is important in new learning and memory.

Although there is some evidence that some social emotions (nostalgia, shame, guilt, disgust) are related more to left hemisphere activity, this may be an artifact of attribution verbalized in language systems (i.e., what we feel guilty or shameful about depends on what we have culturally learned), and this is embedded in our language. So-called primary emotions—anger, fear, happiness, sadness—are seen in other primates and are less lateralizeable. Not surprisingly, there are no consistent findings for lateralized dysfunction in patients with primary mood or anxiety disorders.

Disorders of the limbic system produce numerous forms of psychopathology. Disease of mesial structures (mesial orbitofrontal and diencephalon) can result in disinhibition, depression, mania, anxiety, and panic. Disease of the mesial amygdala and hypothalamus can cause rage; disease of the lateral amygdala and temporal lobe pole can lead to extreme placidity as part of the Kluver-Bucy syndrome.* Disease of the anterior cingulate can cause apathy and stupor. Psychotic features are also commonly seen with limbic system disease.

The Neomammalian Brain

The newest system of our brain includes the cerebral hemisphere heteromodal associational cortices, the cerebellar neocortex, and large parts of the corpus callosum. The neomammalian brain is part of the action brain *and* the perceptual-integrating brain. Lesions in this system can lead to specific behaviors that can be linked to the left or right.

Cerebral Hemisphere Cortex Functional Organization

In addition to left–right specializations, the cerebral cortex is divided into five zones: limbic cortex, paralimbic cortex, primary sensory and motor cortices, unimodal association areas, and heteromodal associational areas.

The *primary sensory cortices* receive afferentation (incoming information) from the external environment. This information is initially modality specific, that is, each primary sensory cortex can accept only one type of input (e.g., the primary occipital cortex can only process visual information; the primary auditory cortex in the temporal lobes can only process sounds). This "unimodal" information is then correlated with other sensory information.

Each primary sensory area is connected to a *unimodal sensory association area.* These secondary cortices organize sensory information into recognizable patterns (perceptions). For example, information of patterns of light and shadow from the primary visual cortex are further organized in the unimodal associational visual

*Bilateral temporal lobe disease causing placidity, increased oral behavior (compulsive chewing and biting; swallowing of foreign bodies), hypersexuality, and visual agnosia.

cortex to form an image so that the light and shadow on this page become recognizable as organized groups of black symbols representing words. Even if the symbols were in an unfamiliar language you would recognize the group of pages as a book.

Heteromodal association areas are in the inferior and superior parietal lobule, the temporal lobe neocortex, and the prefrontal cortex. The occipital lobe does not have heteromodal association cortex. These areas synthesize sensory information from different unimodal sensory association areas and with the limbic system produce integrated, language-relevant perceptions. Names are put to objects. Images on a page are given specific meaning.

The *primary motor cortex* in the frontal lobes subserves the brain's response to sensory information. It gives rise to the internal capsule, and the corticospinal and corticobulbar tracks, the former conveying motor impulses down the spinal cord to the skeletal musculature for movement. The primary motor cortex recruits motor neurons to perform movements.

Unimodal motor association areas contain motor programs for complex movements (e.g., typing, writing, speaking, playing golf). The motor program contains the central representation of all the combinations of pools of motor neurons needed to perform the task. A neuron may be in more than one pool, because there are many more complex motor movements (programs) than there are motor neurons. The motor programs guide the primary motor cortex in selecting the correct pools of motor neurons to do the task. The unimodal motor association areas in turn are guided by sensory information. For example, to hit a golf ball the motor neuron pools called upon for each sequence of the stance and swing must be guided by where the head and torso and the arms and legs are located through each phase of the swing. What each part of the body is doing, where it is precisely located in three-dimensional space at each moment in time, is needed sensory information to guide motor sequencing.

The *limbic cortex* modulates the internal milieu (homeostasis), provides emotional coloring to experience, modulates drives and instincts, and participates in memory and learning. The limbic cortex is in the temporal lobes, particularly the hippocampus.

The *paralimbic cortex* forms a rough link between the rest of the limbic system and cortical heteromodal association areas. It includes orbitofrontal cortex, insula, the temporal pole, parahippocampal gyrus of the temporal lobes, and the cingulate gyrus that curves over the corpus callosum and forms part of the mesial aspects of the frontal and parietal lobes.

Neurochemical Organization

Many behavioral disorders have been related to the brain's specific neurochemical systems. Some of these systems are distributed more or less evenly throughout the brain. Others have more anatomic and behavioral specificity. Understanding neurochemical neuroanatomy helps in understanding behavior, diagnosing and localizing brain disease, and guiding pharmacologic treatments. Several chapters in this book describe the neurochemical–neuroanatomic relationships of particu-

lar conditions (e.g., anxiety disorder, substance abuse), behaviors (e.g., violence), and treatments (e.g., the pharmacodynamics of psychotropic drugs). This section gives an overview of these relationships.

Table 1.4 displays the typical steps in pre- and post-synaptic neuron neurotransmitter activity. Different psychotropic drugs work at different steps in these processes. For example, specific serotonin reuptake-inhibiting antidepressants (SSRIs) such as fluoxetine work on presynaptic Step 5, keeping more neurotransmitter in the synaptic cleft. The increased neurotransmitter, serotonin in this case, is now more available to post-synaptic receptors *and* to presynaptic autoreceptors. Changes in these receptors, reduced (downregulation) or increased (upregulation) numbers, or sensitivity of receptors, is thought to account for the pharmacodynamic behavioral effects of these drugs. Pindolol, in contrast to reuptake inhibitors, is an antagonist of presynaptic serotonin autoreceptors (presynaptic Step 4) inhibiting the presynaptic terminal from slowing the production of serotonin. When given with an SSRI, pindolol permits continued serotonin synthesis and release despite the SSRI keeping more serotonin in the synaptic cleft, which would normally trigger autoreceptors to slow production. This neurochemical "enchancement" is the rationale for pindolol's use as an SSRI antidepressant enhancer (therapeutic effect is uncertain). A different example is lithium, which works on postsynaptic neurons affecting second-messenger systems (post-synaptic Step 2). Monoamine oxidase inhibitors work intracellularly to prevent the presynaptic neuron from breaking neurotransmitters so more is available (presynaptic Step 6).

Table 1.5 displays the different types of neurotransmitters. Neurotransmitters include monoamines, peptides, and amino acids. Receptors, ion channels, and second messengers respond to these transmitters. Hormones and other substances modulate the response. Table 1.6 displays the neuroanatomy and significance of the neurotransmitters presently considered most important to neuropsychiatry.

TABLE 1.4. Generic Steps in Neurotransmitter Action

Presynaptic Terminal	*Post-synaptic Terminal*
1. Transmitter synthesis from a *precursor*	1. Transmitter binding with receptors
2. Transmitter storage in a *vesicle*	a. *Ligand-binding receptor* opens *ion channel,* permitting ions through cell membrane
3. Transmitter release into *synaptic cleft*	b. *G protein–coupled receptor* initiates post-synaptic intracellular sequences via a *second-messenger* substance
4. Transmitter interaction (binding) with presynaptic *autoreceptors* to regulate synthesis and release (by inhibiting presynaptic membrane activity or through presynaptic *second-messenger* systems)	2. Ion exchanges through membrane and second-messenger cascades (e.g., cyclic AMP activity) lead to post-synaptic neuron inhibition or excitation
5. *Reuptake* of transmitter by presynaptic terminal	3. Enzymes degrade the transmitter or transmitter is transported back to presynaptic neuron for reuptake
6. Reuse of transmitter or breakdown of transmitter by *monoamine oxidase*	

TABLE 1.5. Neurotransmitters and Modulators

Monoamines	*Peptides*	*Amino Acids*	*Second Messengers*	*Hormones*
Dopamine	Endorphins	Excitatory Glutamic acid Aspartic acid	Cyclic AMP	Testosterone and other androgenic steroid hormones
Norepinephrine	Enkephalin (beta-, leu-, met-)	Inhibitory Glycine Gamma-aminobutyric acid (GABA Tacrine Proline	Phosphatidyl-linositol cycle	Estrogens
Serotonin	Dynorphin			Progesterone
Adenosine	Tachykinins			Cortisol and related steroid hormones
Acetylcholine	Substance P			
	Somatostatin			
	Cholecystokinin			
	Angiotensin			
	Oxytocin			
	Vasopressin			
	Prolactin			
	Interleukins			
	Neuropeptide Y			
	Neurotensin			

Dopamine is concentrated in the core of the central nervous system and mediates tone (but not arousal), the processes of the action brain, and the hedonistic reward system. Too much dopamine activity usually results in increased motor behavior, mood changes (euphoria and irritability), and disrupted frontal circuit function. Too little dopamine activity usually results in apathy, motor movement disorder, depressive-like syndromes, and poor working memory. Norepinephrine activity is fundamental to flight/fight. Its distribution begins in the pons and then bifurcates into each side of the limbic system and into the cerebral hemispheres. Too much norepinephrine activity usually results in anxiety and defensive or attack behavior. Serotonin distribution begins centrally in the brain stem. Its distribution then splits, one branch paralleling dopamine and the other paralleling norepinephrine. Becuse it interacts with several neurotransmitter systems, abnormalities in serotonin activity result in a wide variety of behavioral changes. Dopamine, norepinephrine, and serotonin are monoamine neurotransmitters. Monoamine oxidase degrades them all.

Endogenous opiates are neuropeptides that usually work as a co-transmitter with some other neuroactive compound. Other neuropeptides are tachykinins,

Table 1.6. Neurotransmitters

Neuroanatomy	*Significance*
DOPAMINE (DA)	
1. DA receptors are G-protein linked; D_1 (post-synaptic) stimulate cyclic AMP, D_2 (presynaptic) and D_4 (post-synaptic) inhibit cyclic AMP 2. DA neurons are located in the brain stem, midbrain, and forebrain 3. Subsystems include a. Nigrostriatal (D_1, D_2) b. Tubulofundibular (D_1, D_2) c. Mesolimbic (D_2, D_4) d. Mesocortical (D_4, D_1, D_2 low) 4. The lateral hypothalamus, ventral tegmental area, and nucleus accumbens linked by the medial forebrain bundle is the brain's hedonistic reward system 5. Mediates some pain sensation via dorsal horn spinal cord projections	1. Provides tone to the brain core and via the thalamus to frontalcircuits 2. The most important transmitter in the action brain, involved in movement and cognition 3. The neurotransmitter of hedonistic reward (pleasure); all drugs of abuse affect DA 4. Underlies hypothalamic–pituitary–end organ axes 5. Underlies personality traits of behavioral activation (e.g., exploratory behavior, impulsivity)
NOREPINEPHRINE (NE)	
1. NE receptors are G-protein coupled; alpha receptors inhibit cyclic AMP; beta receptors stimulate cyclic AMP: $alpha_1$ (throughout the brain) are post-synaptic, $alpha_2$ (cortex and locus ceruleus) are pre- and post-synaptic; beta (throughout the brain) are post-synaptic 2. Major transmitter of the post-synaptic sympathetic nervous system 3. Spinal cord projections involved in muscle tone 4. Major source is the locus ceruleus in dorsolateral pons, projects to a. Cerebellum (few) b. Median forebrain bundle to the hypothalamus, thalamus, basal ganglia, amygdala, hippocampus, and cerebral cortex	1. Regulation of blood pressure, cardiovascular tone, lung airway dilation, intestinal motility 2. Mediates arousal 3. Underlies flight/fight system 4. Involved in new learning and mood (e.g., fear, anxiety, anger) 5. Underlies behavioral maintenance trait behaviors
SEROTONIN (5-HT)	
1. Origin in a series of midline brain stem nuclei: raphe nuclei 2. Multiple receptors: 5-HT_1, 5-HT_2, 5-HT_4 are G-protein linked; 5-HT_3, in periphery only, is ligand gated; some	1. Major homeostatic neurotransmitter; regulates gut, brain stem arousal, and sleep 2. Modulates NE and DA actions

Table 1.6. *(continued)*

Neuroanatomy	*Significance*
SEROTONIN (5-HT) *(continued)*	
receptors are post-synaptic, others are presynaptic 3. Projections include: a. Descending spinal cord in pain modulation b. Dorsal raphe nucleus projections parallel DA projections (forebrain–frontal circuits reward system) c. Median raphe nucleus projections parallel NE projections (limbic system, flight/fight system)	3. Low levels implicated in impulsiveness, violence; high levels implicated in anxiety 4. Underlies behavioral inhibition trait behaviors
OPIATES	
1. Distribution of opiate-related peptides (endorphin, enkephalin, dynorphin) and receptors (mu, delta, kappa) is widespread; some areas of concentration are a. Amygdala and hypothalamus b. Ventral tegmental area and n. accumbens c. Basal ganglia d. Hippocampus e. Cerebral cortex 2. Receptors are ligand binding 3. Receptors located at different sites than peptides (!?)	1. Pain, pleasure, and reward systems modulated by opiate system 2. Opiates modulate monoamine neurotransmitter systems
GAMMA-AMINOBUTERIC ACID (GABA)	
1. GABA widespread throughout the brain, released presynaptically 2. GABA is the only output neurotransmitter of the cerebellar Purkinje cell system, the caudate putamen, and n. accumbens 3. Other GABA concentrations include other basal ganglia structures, the thalamus, and the hippocampus 4. $GABA_A$ receptor, located with GABA neurons, is a ligand-gated chloride ion channel (hyperpolarizes post-synaptic neuron). Benzodiazepines modulate these channels even if GABA is absent 5. Benzodiazepine receptors: "peripheral" is on mitochondrial transporter and	1. The predominant fast-acting inhibitory neurotransmitter 2. The inhibitor of the action brain 3. Antikindling drugs work on GABA-mediated ion channels

Table 1.6. Neurotransmitters (*continued*)

Neuroanatomy	*Significance*
GAMMA-AMINOBUTERIC ACID (GABA) (*continued*)	
has nothing to do with GABA; "central" type 1 is anxiolytic, type 2 is sedative and antiseizure 6. $GABA_B$ receptor, located on presynaptic non-GABA neurons and on post-synaptic GABA neurons, is G-protein linked and inhibits cyclic AMP	
GLUTAMATE	
1. Widespread; particularly action brain 2. "Internal" triangle functional relaton-ship: presynaptic release, glial uptake and re-release, and presynaptic reuptake 3. Several types of ion channel–related receptors: *AMPA* (alpha-amino-3-hydroxy-5-methylisoxazole-4-propionic acid) works on sodium channels, particularly in the action brain; *NMDA* (*N*-methyl-D-aspartate) works on calcium channels, particularly in hippocampus and cerebral and cerebellar neocortices; *kainate* works on sodium channels in the neocortex; *metabotropic* G-protein–linked receptors in cerebellum, thalamus, basal ganglia, hippocampus, cortex	1. Brain's main excitatory amino acid neurotransmitter 2. Function appears to be CNS homeostasis and facilitating new learning 3. Too much release occurs in response to toxins and is implicated in seizure focus spread; phencyclidine binds to the NMDA receptor
GLYCINE	
1. Widespread on inhibitory spinal cord inter neurons and at NMDA sites 2. Post-synaptic inhibitory receptors and a second receptor that is part of the glutamate NMDA receptor required for that receptor to work	1. Inhibitory amino acid, needed for NMDA activation
ACETYLCHOLINE (ACH)	
1. Six types of Ach neurons in CNS with distribution in spinal cord motor ventral horn and brain stem motor nuclei 2. Projections to motor system and peripheral nervous system 3. Projections from pons to basal ganglia as part of the Reticular Activating System 4. The nucleus basalis of Meynert projects to other forebrain areas and then to the	1. Major transmitter of the peripheral motor system 2. Major transmitter of the presynaptic autonomic nervous system 3. Major post-synaptic transmitter in sweat glands and the entire parasympathetic nervous system 4. As part of Reticular Activating system (RAS) regulates sleep/wake cycles

Table 1.6. (*continued*)

Neuroanatomy	*Significance*
ACETYLCHOLINE (ACH) (*continued*)	
entire cerebral neocortex, hippocampus, and amygdala	5. Hippocampal and cortical projections involved in new learning; Ach neuronal degeneration in nucleus basilis of Meynert implicated in Alzheimer's disease; Ach neuronal degeneration in basal ganglia implicated in Parkinson's disease
5. *Nicotinic receptor* linked to ion channels (sodium and calcium) mostly in presynaptic neurons in CNS and sympathetic ganglia; *muscarinic receptors* mostly on post-synaptic parasympathetic neurons (influence heart, gut, sweat glands). A CNS muscarinic post-synaptic receptor (M_1) is G-protein linked, an M_2 version is an autoreceptor	

substance P, and somatostatin, concentrated in the medulla, hypothalamus, and hedonistic reward system. Amino acid neurotransmitters include GABA and glycine (both inhibitory), glutamate (excitatory), and acetycholine (the prime transmitter of the peripheral nervous system as well as a fundamental central nervous system transmitter).

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2

Neuropsychiatric Evaluation

Neuropsychiatric evaluation is the medical examination of the brain. The *mental status examination,* and the *psychiatric interview* are terms from the era of a mind–body dichotomy. There is no "mental" to it. The mind, in a neuroscience sense, is a shorthand metaphoric term for summing up what our brains do that reaches our awareness (e.g., internal speech subserved by the dominant hemisphere, emotional experience generated by the limbic system, body image and its physical limits subserved in part by the parietal lobes, and memory acquisition, storage, and recall subserved by several brain systems). When you assess "mental functions" and behavior you are assessing brain functions.

In examining for brain disease or dysfunction, behavior changes are the patient's signs and symptoms. Your questions and comments are your osculation and percussion. The evaluation, however, is of the brain, and the examination follows all the rules for the evaluation of any body organ. The evaluation includes the examination itself (the present state of the patient), the history, and laboratory studies. An analogy is the examination of the digestive tract with its different components (e.g., stomach, gallbladder, large intestine). Each component has its pattern of signs and symptoms, and each component is vulnerable to different diseases. The brain also has component parts or systems with different signs and symptoms (e.g., left, right, action vs. perceptual-integrating brain) and different disease vulnerabilities (e.g., herpes has an affinity for the temporal lobes). Knowing the brain's different components and their behavioral signs and systems helps to structure your examination.

Examination Goals

Neuropsychiatric examination requires skill in interacting with people. It is mostly done without touching the patient and begins when you first meet the patient. The goals are to establish a reasonable doctor–patient relationship (essential for correct diagnosis and treatment) and determine probable diagnosis.

Keep historical information separate from behavioral observations made dur-

ing the examination. Past information is needed for diagnosis, but behaviors can change as the illness changes: Bipolar patients can shift from stupor to excitement to depression within hours or minutes; suicidal feelings may fluctuate. You may need to examine an acutely ill patient several times daily, and confusion results if you mix historical with physical findings.

The examination also gathers information needed to plan laboratory testing (e.g., neuropsychological testing, brain imaging, electrophysiologic measures) and treatment. For example, knowing a patient's hobbies and interests may not be helpful for diagnosis, but it may provide clues about cognitive strengths and weaknesses that could affect treatment outcome. In such a situation neuropsychological testing might help vocational planning following recovery. Some personal information has no diagnostic importance (e.g., names and interests of the patient's children), but, because treatment is often long-term, this information helps you maintain an interest in the patient's life, reinforces the doctor–patient relationship, and increases compliance with treatment.

Examination Style

The style of the examination depends on your personality. You can facilitate the examination by establishing a conversational manner and interactive relationship with the patient (i.e., the tone and style of the examination) and reinforcing this interactive relationship with personal and supportive comments.

A metaphor for the examination style is a kitchen conversation with a neighbor who comes to you for advice. In that situation, many people would invite the neighbor into the kitchen (more homey and relaxing than the living room), ask the neighbor to sit, and perhaps share some coffee. There would be "small talk" while the coffee is brewing, and finally the reason for the visit would be raised. In the form of a medical evaluation, the patient is invited in and made to feel comfortable. You determine the setting and you initiate the "small talk." Doing this controls the situation and begins subtly establishing the doctor–patient relationship.

This process begins when you first see the patient. Greet the patient outside the office, and chat with him during the walk to the office and as he sits. This introduction often sets the tone for the rest of the examination. Put the patient at ease by commenting about the weather, or a recent inpatient activity, or the trip to the clinic or office. Patient uncooperativeness usually results from anxiety, suspiciousness, or irritability.

Additional examination style strategies are to

1. Sit kitty-corner to the patient, not behind a barricading desk (face to face is too aggressive and anxiety provoking for many patients).
2. Assume a relaxed posture (rather than sitting stiffly like a "judge").
3. Avoid jargon. Use colloquialisms and idioms whenever possible so that terms such as *episode, hospitalization,* and *diagnosis* become phrases such as "when you were sick before," "when you last stayed overnight in a hospital," and "did your doctors tell you what they thought was

the problem (did they give it a name)?" Specific words and phrases you use are important. "Hi" is less formal than "hello"; "chat" is more relaxing than "examine" or "talk."

4. Use appropriate humor. Patients also say and do things that are humorous; do not be afraid to laugh or also say something humorous.
5. Be forthright. Even the awkwardness of having to complete clinical forms while examining the patient can be minimized by comments such as "I'll be taking a few notes to keep things straight in my mind" or "to be thorough to make sure I don't forget to check on things that may be important."
6. Use supportive comments to put the patient at ease, and give him the sense that you are interested in him as a person and not just as a clinical entity. Manic patients, for example, often become cooperative following positive comments about their colorful dress. Dysphoric depressed patients are more willing to relate their experiences following empathic comments about their obvious distress rather than "how are you feeling" type comments.

THE EXAMINATION STRUCTURE

The conversational tone of the examination is its style. Despite this seeming informality, the examination should not be haphazard. Structure is important. Questions and testing procedures should proceed logically while remaining responsive to the patient's behavior. Cover every area of the examination in a standardized manner. Use open-ended questioning at the beginning of the assessment of each topic area and then focus on the details. Maintain a sequence of topics and approach for each symptom area. The examination structure follows the logical steps underlying the diagnostic and treatment planning process.

Step 1: Determine the syndrome by establishing the patient's fundamental chief complaint. The fundamental chief complaint is not simply the first thing the patient tells you about why he is seeing you. It is the minimum amount of information needed to generate an initial differential diagnostic list. For example, the patient says he is "anxious." Such a statement usually becomes *the* chief complaint in a medical history. The fundamental chief complaint, however, puts this initial statement into some context. The fundamental complaint is a word picture suggesting diagnostic ideas to you. "Anxiety and headaches" has different but overlapping diagnostic implications from "anxiety and loss of interest." The first would generate a list of diagnostic choices that would include pheochromocytoma; the second would generate a list that included major depression. When looking for the pattern, focus on the basic big picture features of the signs and symptoms. When considering diagnostic choices also remember that the most important diagnostic information is age, gender, and then type of illness onset (e.g., acute vs. insidious). For example, if the patient is fe-

male you do not have to worry about testicular disease. The odds are also against antisocial personality disorder, Gilles de la Tourette's disease, and most alcohol-related conditions. These are not absolutes, but probabilities. The diagnostic choices are also different (although overlapping) for different age groups (e.g., under 40 or over 70 years). Complaints of anxiety and tremor in the latter would have Parkinson's disease high on the differential list, while in the former anxiety disorder would be high on the list.

An illustration combining age, gender, and onset is that of a 70-year-old man who became confused at a party and then fainted. Upon awakening he was frightened and did not recognize his neighbor. Put more precisely, a 70-year-old man had a sudden neurologic-like event. The same patient described a bit differently presents a clearer picture, and you think immediately of stroke or a TIA or RIND*.

Once you begin thinking of the differential diagnosis of the patient's fundamental chief complaint, you then ask a screening question for each of your choices. If the patient endorses a screening question, pursue the details to see if the patient in fact has that condition. As you proceed, you may think of additional diagnostic possibilities, and you also screen for these. At the end of this step you should reach your conclusion: the syndrome.

Step 2: decide if the syndrome is primary (idiopathic) or secondary to some classic neurologic (e.g., stroke) or general medical condition (e.g., hypothyroidism). This step is necessary for assessing prognosis and for determining specific treatments.

Step 3: Gather other information and identifying co-occurring conditions needed to shape acute and long-term management (e.g., other conditions the patient may have that increase drug side effect risks, premorbid personality traits that can hinder or help rehabilitation).

The above diagnostic steps apply to all branches of medicine. For example, a patient complains of a cough and feeling tired and feverish. You think of pneumonia, and the physical examination confirms it. That is Step 1. You next try to determine etiology: viral, bacterial, toxic. That is Step 2. Lastly, you gather additional information necessary for treatment, for example, is the patient asthmatic or immunologically compromised. That is Step 3.

Although generic to medical diagnosis, these steps are particularly important in diagnosing behavioral syndromes because the etiology and specific pathophysiology of these disorders remain elusive. Present-day classification is also a "best guess" approximation, and many patients do not clearly fit into the a diagnostic category. Within category heterogeneity is also likely. Knowing *how* to diagnose is more important than knowing every set of diagnostic criteria in the

*TIA, transient ischemic attack, usually lasts less than 24 hours; RIND, reversible ischemic neurologic deficit, usually lasts less than a week. Neurologic deficits related to a vascular event that last beyond 2–3 weeks define stroke.

Diagnostic and Statistical Manual of Mental Disorders (DSM). Criteria and categories change, but the logic of the diagnostic process does not.

Procedures for examining the brain need to be overlearned, just as procedures for examining the heart or lungs must be overlearned. Learn a flexible script or pattern of inquiry for each area of psychopathology or historical topic. Sample questions are:

> *Introducing topic areas:*
>
> "Sometimes when people feel the way you do now they also have (or experience). . . ." "I knew someone who had the same thing happen to him, and he also had. . . ." "You said you've had some difficulties with your memory. Is it the kind of difficulty that. . . ."
>
> *Screening questions:*
>
> "Has there ever been a time when for weeks or even longer you were feeling down, sad, depressed, or without energy or interest in things?" "Have you ever felt the opposite of being depressed, where you were full of energy, excited, or really hyper for more than a few minutes or hours?

"Occasionally, patients remain uncooperative or difficult to examine despite your efforts to put them at ease. Table 2.1 lists some of the more common examination problem situations and techniques that help resolve them.

Thus, to be a good examiner you must know the examination structure, know the script of questions, and have an examination style that puts patients at ease and gets them to talk. You also must understand how to generate a useful differential diagnostic list and know how to describe and organize examination observations so that they make sense.

THE DIAGNOSTIC PROCESS (FIGURING OUT THE SYNDROME)

Most clinicians diagnose by pattern recognition (interpretation of EEG and most x-rays are done this way). Pattern recognition is the hallmark of residency training: See a few, remember what they looked like and what treatments worked, and if you see it again reach the same conclusion and do the same thing again. All well and good, but consider the following:

It is Saturday night and a middle-aged man stumbles into your busy urban emergency room. His speech is slurred, and his eyes are blood shot. He is irritable and loud. Most clinicians are trained to recognize this word picture (this pattern) as possible alcohol intoxication because the odds are good that the patient is intoxicated. The man might even have had a drink, and so his breath smells of alcohol. But, of course, other conditions must be considered: He might have diabetic hypoglycemia, be an epileptic who just had a seizure outside the hospital and is now in a post-ictal state, or be suffering from a hit to the head while having a drink in a local bar. Only an understanding of the diagnostic process and how to use it will result in the correct diagnosis for this patient. Thus, as long as the pa-

tient has a typical syndrome or is well known to the physician, using pattern recognition to diagnose is helpful. Pattern recognition diagnosis can be very accurate, but it has only modest reliability (e.g., the degree to which a group of clinicians would agree on the diagnoses of a group of patients). Table 2.2 displays some neuropsychiatric patterns. However, additional methods are necessary for diagnosis because there are no pathognomonic laboratory tests for psychiatric illness, and many patients do not fit the DSM patterns. The method that is the basis for all medical diagnosis is the probabilistic process of exclusion and inclusion.

A patient's most likely diagnosis is selected from many possibilities. For example, Patient A arrives for evaluation and treatment. Without additional information (e.g., age, gender, chief complaint), every condition in the *International Classification of Disease* is equally probable. With additional information, however, the probability that Patient A is suffering from some conditions diminishes, eventually reaching zero, while the probability favoring other diagnoses increases. Eventually, this continuing process of exclusion and inclusion leaves you with the most likely diagnosis. This is the process you use in Step 1 of the examination structure. To illustrate further, if Patient A is female, the possibility of prostate disease is zero. If Patient A is a 23-year-old sexually active woman who has missed two periods, the possibility of pregnancy becomes a strong probability, while the probability of most other conditions decreases, many to virtually zero. More information is obviously needed for furthering the exclusion/inclusion process, but if you do it properly you will reach the most likely diagnosis.

The process of exclusion/inclusion by probability requires you to (1) know the possibilities (i.e., the available diagnostic choices); (2) know the information that discriminates patient groups (i.e., which clinical data change the probabilities and which do not); (3) be able to elicit signs and symptoms (the psychopathology that alters the probabilities); and (4) be able to identify and properly organize signs and symptoms.

Additional guidelines are

1. *The duck principle,* which states that "If it looks, walks and quacks like a duck, it most likely is a duck." This is pattern recognition. For example, a 68-year-old man complains of concentration and memory problems and his wife says he is not himself and he is losing his mind. He is also despondent and apprehensive, having trouble sleeping, is not eating and is losing weight, and says he wants to die. This "duck" is melancholia.
2. *Sutton's law,* which is based on the vignette that when Willy Sutton, a famous U.S. bank robber in the 1950s, was asked by a reporter why he robbed banks, he replied incredulously "Because that's where the money is!" Applied to diagnosis, Sutton's law tells you the most likely condition under the particular circumstances of the situation is probably the correct diagnosis. A corollary to this is, when you hear hoofbeats in the United States they are from horses, not zebras. The above melancholic man also had problems concentrating and with his memory. His wife said he was losing his mind. He is 68. Alzheimer's dis-

TABLE 2.1. Difficult Examination Situations and How To Resolve Them

Situation	*Resolving Techniques*
	DEPRESSION
A. Psychomotor retardation	1. Slow down rhythm of questions, reduce number of questions, ask more closed-ended, concrete questions. 2. Interview in several 10–15 minute segments rather than one long interview. 3. Interview in late afternoon when retardation may be less due to diurnal pattern of symptoms
B. Continuous ruminations	1. The approach is the same as for mania, C, but at a much reduced rate
	MANIA
A. Agitated, pacing patient	Walk with the patient and have an examination "conversation" "on the go"
B. Irritable, tendency to dismiss the question as stupid, to tell examiner to read the information in the chart	1. Address the least emotionally laden topics first. 2. If the patient has a constant theme, use it to introduce questions on other topics that must be assessed, even if the sequence strains logic. 3. Do not get insulted. 4. Remain firm but nonjudgmental and matter of fact
C. Overtalkative with press of speech; circumstantial speech or flight-of-ideas	1. Increase interview structure, use more closed-ended questions, and speed up rhythm of questions. 2. Repeatedly come back to a topic so that the interview content does not "get away." 3. If the patient is interruptible without producing unacceptable irritability, stop the flow of speech and say such things as. "I'd like to know more about that later, but right now. . . .

ease is a reasonable consideration. Sutton's law, however, also picks melancholia because in persons 65–70, depression is four times as common as Alzheimer's, which typically does not fully express itself until the mid-70s.

3. *The rule of parsimony* tells you to try to explain the patient's many complaints and clinical features by as few underlying pathophysiologic processes as possible—one being the best. Looking for parsimony is looking for the common underlying theme. In the melancholic man above, you find out he has some cortical atrophy and ventricular en-

TABLE 2.1. (*continued*)

Situation	*Resolving Techniques*
	4. If flight-of-ideas is uncontrollable, use the patient's distractibility by switching with great show to questions related to specific diagnostic criteria, or have a third party in the room to which you address your questions (e.g., "how old did you say he was?"), perhaps stimulating the patient to interrupt you with the answer (e.g., "48").
	5. Begin to ask questions in so soft a voice that the patient becomes distracted by it and asks you what you said. The examiner raising his voice rarely helps
	SCHIZOPHRENIA
A. Persecutory delusions and extreme suspiciousness	1. Start with the least emotionally laden topics.
	2. Orient the wording of all questions about present episode and psychopathology to the patient's viewpoint.
	3. Avoid all judgmental sounding phrases
B. Avolitional patient with paucity of speech and content	1. The approach is the same as for depression.
	2. Patient may be willing to do paper and pencil cognitive tests and cooperate with general medical examination.
	3. The latter can be used as a structure for assessing behavior
C. Patient with severe formal thought disorder	1. The approach is the same as with an avolitional patient.
	2. Some information may also be obtained by focusing on visual and pictorial tasks rather than verbal tasks

largement on magnetic resonance imaging (MRI) and diffuse cognitive impairment. You could conclude that he has both depression and Alzheimer's, but the MRI and cognitive findings are also consistent with depression and so all his problems can be explained by that single pathophysiologic process. Parsimony facilitates good treatment.

Diagnostic choices are discussed throughout the rest of this book. Discriminating data for each are presented. A good examination style and structure helps you to elicit important information. The phenomenologic clinical method (see below) helps you to organize psychopathology and other information that relates to specific conditions. This organizing helps you formulate a recognizable clinical pattern.

TABLE 2.2. Some Diagnostic Patterns Seen by the Neuropsychiatrist

Pattern	*Most Likely Diagnosis*
Hallucinations, delusions fluctuating language disturbance, loss of emotional expression, loss of drive and ambition, no history indicating traditional neurologic disease (e.g., stroke, tremor)	Schizophrenia
Typical depressive features (e.g., insomnia, anorexia, psychomotor retardation), but without profound unremitting sadness or dysphoria (the words, but not the "music" of depression)	Secondary depression A. With increased muscle tone and bradykinesia: parkinsonism B. With renal stones: parathyroid disease C. With paresis: stroke
Altered mood (irritability or euphoria), rapid/pressured speech, hyperactivity	
A. Broad affect, no traditional neurologic disease	Bipolar mood disorder, mania
B. Shallow mood, avolitional, chronic course	Frontal lobe disinhibited syndrome
Transient and episodic perceptual disturbances and delusions with altered responsivity, but no affective blunting at other times	Seizure disorder
Anxiety or obsessive compulsive features beginning after age 35 years in a male patient	General medical condition (e.g., endocrinopathy, hypertension), traditional neurologic disease
Altered or fluctuating level of arousal, agitation, diffuse cognitive impairment, rambling speech	Delirium
Clear consciousness; diffuse cognitive impairment, particularly affecting memory; organizational and problem-solving difficulties	Dementia
Dementia with ataxia and urinary incontinence	Normal-pressure hydrocephaly

THE PHENOMENOLOGIC CLINICAL METHOD

The phenomenologic clinical method incorporates (1) objective observation of signs and symptoms; (2) description using precise terminology; and (3) assessment of the form of behavior separately from its content.

Objective observation of signs and symptoms means first observe, then interpret. For example, if a patient is loudly mumbling to himself, you might interpret this as hallucinating, and some patients do subvocalize while they are experiencing auditory hallucinations. But what if this patient is not hallucinating and instead has frontal lobe disease and is no longer able to monitor his own behavior? Most of us talk to ourselves, but we do it in private. We have learned to monitor our behavior to keep it socially acceptable. A hallucinating patient is psychotic,

and that suggests a number of diagnostic possibilities. A patient who cannot self-monitor from frontal lobe disease reflects a different group of possibilities. Treatments for these two groups often differ. Because no single behavior is pathognomonic, the best approach is to describe each behavior accurately and see if the descriptions fit into a recognizable pattern.

A corollary of observation before interpretation is observation before treatment. Treating a patient means you have already made several important decisions: The patient is ill and needs treatment (a very big decision indeed), and the patient has a particular condition likely to respond to the treatment choice.

Sometimes treating immediately (e.g., the patient is hallucinating, give him an antipsychotic) works, but many times it masks important clinical features (e.g., are those tremors from the neuroleptic drug, or does the patient have basal ganglia disease that caused the behavioral symptoms?). Rushing to treatment may also make some patients worse. For example, some antipsychotics can lower seizure thresholds so that some patients with post-ictal psychosis can be made more irritable (and possibly dangerous) by giving them an antipsychotic for their psychosis rather than an anticonvulsant.

Precise terminology is important because imprecise terms fail to discriminate disorders. For example, the term *confusion,* could refer to an altered state of consciousness (as in delirium), disorientation in clear consciousness (as in dementia), unintelligible speech (as with a dominant hemisphere stroke), or it could describe a patient who gets easily lost or who cannot find his way about the hospital unit (as with some nondominant hemisphere strokes). Each of these conditions has a different diagnostic implication that goes unrecognized when each is subsumed under the vague term *confusion.*

Terms such as *incoherent or irrelevant speech* or *looseness of associations* are also imprecise. Does looseness of associations mean jumping from topic to topic, uttering nonsequiturs, speaking in word salad, having fluent paraphasic speech, or rambling? The speech of some aphasic patients is incoherent and at times irrelevant to the topic, as is the speech of some schizophrenics and some manics. If you think in and use imprecise terms, you will not discriminate aphasia from schizophrenic formal thought disorder or from manic flight-of-ideas, and misdiagnosis may occur. *Paranoid* is imprecise: Does it mean delusional, having persecutory delusions, being suspicious, being uptight, being crazy? The precise term helps in the diagnostic process. The imprecise term usually leads down a diagnostic dead end.

Form separated from content is difficult. The form of psychopathology reflects the illness process (what you are trying to identify). The content of psychopathology most often reflects the person and his experience. What a patient is talking about, the words spoken by a hallucinated voice, and the specifics of a delusional idea are content. The linkage of speech and word usage, the clarity and duration of a hallucination, and whether the delusional idea derives from other psychopathology or appears suddenly and fully formed are form. The content of psychopathology varies dramatically across cultures; the form of psychopathology does not. For example, melancholic patients from rural African villages and from industrialized western cities share the characteristic profound apprehensiveness, gloominess, insomnia, anorexia, psychomotor retardation, and feelings of guilt. What varies is the content of the guilty ideas and ruminations.

USING THE DSM SYSTEM

The DSM system is easy. The criteria for individual syndromes, however, are complicated, and very few clinicians know all the criteria for all the syndromes. The system fits with the examination structure previously described: (1) diagnose the syndrome; (2) decide if the syndrome is primary (idiopathic) or secondary (to a knowable neurologic pathophysiology or a general medical condition); and (3) identify other co-morbid conditions. The rest of the evaluation is done either to confirm these three steps or to gather additional information necessary for treatment and management.

However, knowing some diagnostic criteria is important, because you use diagnostic criteria at the beginning *and* at the end of the diagnostic process. At the beginning, the DSM provides the list of choices (i.e., the possibilities). In the DSM system these possibilities are axis I (the primary and secondary states of illness) and axis II (the personality disorders). A working knowledge of specific sets of diagnostic criteria is also needed, so when the patient endorses a screening question, or you strongly suspect a condition and need to know the details, you know what to ask and look for. For example, if the chief complaint suggests depression, you ask a screening question for depression. If the patient endorses this, you must know the basic diagnostic criteria for depression to ask further questions (e.g., about sleep, appetite, changes in libido). Criteria help shape the structure of the evaluation from its beginning. Once you have collected all the information you need, knowledge of diagnostic criteria is again used to evaluate what has been collected. You do this to determine if the patient's signs and symptoms and historical information meet criteria.

The DSM system's complexity of criteria does not ensure their specificity. Many criteria, are not operationally defined. For example, the diagnosis of dysthymia requires the vaguely defined criteria "low energy" or "fatigue," "poor concentration," and "difficulty making decisions"; the criteria for generalized anxiety disorders include "feeling keyed-up or on edge" and "having difficulty concentrating." The DSM does not define these terms. There are specific tests, however, of concentration and more precise ways of describing various other criteria, but these procedures cannot all be incorporated into the DSM system. You must have your definitions to become a precise diagnostician. The scientific literature provides some data suggesting what constitutes an abnormal "this" or a deviant "that" and how to go about measuring them. Succeeding chapters will focus on these suggested operational definitions.

THE NEUROPSYCHIATRIC HISTORY

A good psychiatric history is the basis of a good neuropsychiatric history. If there is a difference, it is of emphasis. For example, a good psychiatric history must determine if the behavioral syndrome is secondary to some classic neurologic or general medical condition. The neuropsychiatric history does this too, but also focuses on psychopathology as indicators of specific brain dysfunction (e.g., action

vs. perceptual-integrating brain disease) that may respond to more specific treatments rather than just information to determine if DSM criteria are met. For example, you assess a patient's avolition to see if he meets criteria for schizophrenia or depression. The neuropsychiatrist also wants to know if this avolition is due to action brain dysfunction that might respond to a dopamine agonist such as methylphenidate. The neurologic implications of historical information about psychopathology are covered throughout this book. Historical areas to assess when determining if a behavioral syndrome is primary or secondary are displayed in Table 2.3.

The Traditional Neurologic Screening Examination

No psychiatric evaluation is complete without a traditional neurologic examination. The traditional neurologic examination includes an assessment of gait, balance, coordination, motor strength, reflexes, and cranial nerve and sensory function. Table 2.4 describes a screening neurologic examination that takes about 20 minutes to complete.

Many patients with behavioral syndromes have normal traditional neurologic examination findings, but nevertheless, have *soft neurologic signs.* These are expressions of brain dysfunction, but with less localizing power than the neurologic signs described in Table 2.4. Some soft neurologic signs are advantitious motor overflow, Gegenhalten, and stereotypes (see Chapter 3—motor behavior). Also included is soft signs are palmomental reflex, and double simultaneous discrimination)*. When a patient has a behavioral syndrome and specific neurologic features, assume the behavioral syndrome is secondary until proven otherwise. Chapters on syndromes describe specific relationships. The presence of soft signs, however, is consistant with a primary syndrome.

Laboratory Assessment

To use laboratory tests well, several principles and strategies are followed. Other than tests required by hospital or clinic policy, there are no routine laboratory tests. Every test costs time and money and may inconvenience or discomfort the patient. Have a specific reason for ordering every test. Some basic reasons are as follows:

To confirm or eliminate a suspected etiology for the patient's behavioral syndrome is the main reason for ordering laboratory tests. To get the highest yield

*The *palmomental reflex* is induced by scratching the fleshy base of the thumb causing a downward movement at the corners of the mouth. The reflex indicates dysfunction only when repeated stimulation continues to produce a response. The frontal lobes are usually involved when the reflex fails to extinguish. *Double simultaneous discrimination* (the face–hand test) is tested by having the patient close his eyes and then lightly stroking him on the cheek and dorsal surface of the hand ipsilaterally and contralaterally. Failure to perceive the hand being stimulated or one side of the face or hand (left or right) indicates dysfunction usually in the frontal or parietal lobes.

TABLE 2.3. Historical Antecedents of Secondary Behavioral Syndromes

Antecedent	*Most Likely Secondary Syndrome(s)*
Cardiovascular disease	
Hypertension	Generalized anxiety disorder, subcortical dementia
Atherosclerosis	Focal stroke syndrome, vascular dementia
Acute heart failure	Delirium with psychosis
Chronic heart failure	Personality change, frontal lobe dementia, atypical depression
Lung disease	
Acute infection	Delirium with psychosis
Chronic obstructive pulmonary disease	Personality change, frontal lobe dementia, atypical depression
Kidney disease	
Infection or acute renal failure	Delirium
Chronic renal failure	Personality change, frontal lobe dementia, atypical depression
Diabetes	Vascular dementia, hypoglycemic delirium, stroke syndromes
Other neurologic disease	
Viral encephalitis	Dementia, generalized anxiety disorder, adult-onset attention deficit disorder
Head injury	Many syndromes, including personality change, dementia, focal brain syndromes
Epilepsy	Many syndromes, including depression, psychosis, and panic disorder
Multiple sclerosis	Anxiety disorder, nonmelancholic depression
Other conditions	
Lupus erythematosis	Mood disorders
AIDS	Nonmelancholic depression, subcortical (white matter) dementia
Carcinoma	Depression, subcortical (white matter) dementia
Gestational, labor, and delivery problems	Developmental disorders, schizophrenia
Drug Abuse	
Acute	Delirium, psychosis
Chronic	Psychosis, dementia
Endocrinopathies	
Hyperthyroidism	Anxiety disorders, delirium
Hypothyroidism	Depression, dementia
Cushing's disease	Depression, bipolar mood disorders, generalized anxiety disorder, delusional disorder

TABLE 2.4. Screening Neurologic Examination

Examination Area	*Techniques*	*Some Important Findings*
Inspection	Looking with educated eyes	1. Dysplasia* 2. The triangular-shaped head and face with large ears, lips, and nose of fragile-X syndrome 3. Short stature, webbed neck of Tuner and Noonan (male Turner) syndromes 4. Short stature and facial features of Down syndrome 5. Asymmetry of facial features, trunk, and limb muscle groups from atrophy
Gait	Free walk and tandom walking (about 12 feet). Standing with feet together, eyes closed for 1 minute (Romberg sign if the patient is unsteady)	1. Loss of secondary arm movements in Parkinson's disease 2. Broad-based gait in cerebellar disease 3. Shuffling gait of anterior brain disease 4. Hemiplegias 5. Ataxia
Motor strength (rest of motor examination is covered elsewhere)	Flexion, extension, rotation of head; shrugging shoulders, flexion, extension abduction of limbs and fingers all to resistance; sitting up from a prone position standing on one leg and then the other for 10 seconds each; simplest routine is to do motor examination in rostral to caudal order	1. Weakness in both legs, or both legs and arms, indicates spinal cord problem 2. Weakness in lower face, arm, and leg on one side indicates damage in contralateral cerebral hemisphere or brain stem. 3. Weakness in distal part of limb indicates peripheral neuropathy
Deep tendon stretch reflexes (DTRs)	Dorsum of arms, triceps, knee "jerk," ankle, and plantar muscle tendons tested; must be relaxed and under maximum gravity effect	1. Asymmetry is more important than absolute rating of reflex, intensity can be altered by many factors (e.g., anxiety, drugs) 2. DTRs are hyperactive with corticospinal damage, and are hypoactive with peripheral nerve damage 3. If patient has an atypical depression always check for sluggish reflexes of hypothyroidism 4. Extension of the big toe to plantar stimulation (Babinski sign) seen with brain or spinal cord damage (upper motor neuron)

TABLE 2.4. Screening Neurologic Examination (*continued*)

Examination Area	*Techniques*	*Some Important Findings*
Somatosensory function (excluding the face)	Pain and touch: Stimulate the skin with the sharp and blunt ends of a safety pin in random order covering dorsum and soles of hands and feet	The main purpose of somatosensory testing in a neuropsychiatric setting is to assess for peripheral neuropathy due to alcoholism, thiamine deficiency, and diabetes
	Vibratory sense: Apply vibrating tuning fork to bony eminences (the malleoli, distal ends of radii) of each limb	
Cranial Nerves		
Olfactory (I)	Any strong odor-producing substance will do. Test one nostril at a time. Most overlooked area of examination.	1. Unilateral and bilateral anosmia not due to pharyngeal disease is ominous. Look for olfactory groove meningioma (unilateral), optic atrophy and optic nerve tumor (unilateral), and frontal lobe masses
		2. Frontal head trauma shearing olfactory nerves is most common cause of anosmia. Change in olfaction can cause poor appetite and changes in food preferences and sexual behavior. Associated with frontal lobe dysinhibition syndrome
Optic (II)	Always look at the optic nerve and test for visual acuity; carry a pocket eye chart or have one in the examining room.	1. Normal fundus is yellow, flat, and clearly demarcated from surrounding red retina. Vessels are clear and not tortuous
		2. "Salt and pepper" pigmentation seen in thioridazine overexposure and complaints of night vision problems
		3. Poor light reflex and pale fundus seen in optic neuritis (think of multiple sclerosis)
		4. Blurred and raised optic disc with retinal vessel pulses visible indicate increased intracranial pressure.
	Visual fields tested just before testing range of ocular movement. Face the	Patterns of visual field loss
		1. Unilateral central: Migraine, optic neurotics, nerve damage

TABLE 2.4. (*continued*)

Examination Area	*Techniques*	*Some Important Findings*
	patient and stand 50 cms apart. Have him stare at your nose. Have the patient cover one eye with his hand. Hold your index finger just outside your peripheral field in the inferior quadrant equidistant between you and the patient. Wiggle finger slowly and move it toward center field until the patient sees it. Repeat in all quadrants, and then repeat with other eye	2. Bitemporal upper quadrant: Lesion in the optic chiasm, such as pituitary adenoma or craniopharyngioma 3. Bitemporal hemianopsia: Advanced optic chiasm lesion 4. Homonymous hemianopsia (same sides of visual fields): Contralateral cerebral lesion such as stroke 5. Homonymous upper quadrant anopsia: Contralateral temporal lobe lesion
Oculomotor (III), trochlear (IV), and abducens (VI)	Range of ocular movement: Tell the patient to follow your finger with his eyes without moving his head. Then, holding your finger 50 cms in front of the patient, move it from the center of the patient's presumed visual field to the right, left, up, and down positions. At the extremes of these check for nystagmus. Then have the patient follow your finger to the tip of his nose	1. Impairment: Dilated pupil, ptosis, outward deviation of eye (III) 2. Eye deviated inward (superior oblique). Look for midbrain lesion (IV) 3. Inturning of the eye (lateral rectus muscle) but no ptosis or pupil changes. Look for pontine lesion. Classic sign of Wernicke-Korsakoff constellation (VI) 4. Diplopia results from dysconjugate gaze from III or VI lesion. 5. Basilar artery syndrome: Brain stem lesion causing diplopia, contralateral hemiparesis, or ataxia
Trigeminal (V)	Touch and sharp–dull testing of three divisions: forehead, cheek, jaw Corneal reflex tested with a wisp of cotton on cornea producing a blink While feeling the muscle, have patient clench jaw, protrude jaw, and push jaw left and right against your hand pressure Jaw jerk: While patient's jaw is relaxed, place your index finger under lower lip and tap it with wide end of reflex hammer	1. Damage causes facial analgesia, poor corneal reflex, hypoactive jaw jerk, and jaw deviation to side of lesion 2. Nasopharyngeal tumor, gun shot wounds, acoustic neuroma (unilateral hearing loss) 3. Hyporeflexic in bulbar palsy, 4. Hyperreflexic in pseudobulbar palsy (associated with dysarthria, dysphagia, and emotional lability) 5. Upper motor neuron lesions

TABLE 2.4. Screening Neurologic Examination (*continued*)

Examination Area	*Techniques*	*Some Important Findings*
Facial (VII)	Observe expressions; test taste with diluted salt or sugar solution if patient complains or other signs indicate need	1. Poor facial expression (muscles of the face are paretic) 2. Can chew (V) but cannot taste (glossopharyngeal posterior $1/3$ of tongue for taste, facial anterior $2/3$) 3. Upper facial muscles bilaterally innervated; lower facial muscles contralaterally innervated 4. Lyme disease can mimic Bell's palsy (idiopathic inflammatory disease), with unexpected crying or laughing.
Acoustic (VIII)	Whisper into each of the patient's ears while covering the other, or use tuning fork beginning at same distance you can hear it. Weber test: Tuning fork on vertex, mastoid	Unilateral deficit in men usually hereditary and benign. Can be due to sudden noise, virus, Meniere's disease, or acoustic neuroma (rare) Nerve problem, air and bone conduction of sound poor and from vertex, sound louder on less impaired side. Conduction problem, sound louder on more impaired side
Glossopharyngeal (IX), vagus (X), spinal accessory (XI), Hypoglossal (XII)	Listen to the patient's speech, look for uvula deviation, induce gag reflex with tongue depressor, feel patient's throat as he swallows, have patient stick out tongue, swallow, cough	1. Bulbar group nuclei in brain stem caudal to III, IV, and VI 2. Look for pontine and medulla lesions 3. Dysarthria, dysphagia, hypoactive jaw, and gag reflexes in pseudobulbar palsy

*Dysplasia refers to body parts disproportionate in size or shape and deviant in location (e.g., low-set or malformed ears, eyes too widely spaced apart, arms or legs too short for torso).

from these tests, be specific. For example, if you order an EEG, the patient should have a suspected condition likely to produce an abnormal EEG. EEG is helpful if epilepsy, delirium, or a focal brain injury is suspected. EEG is usually not helpful in distinguishing the pseudodementia of depression from Alzheimer's disease. Other laboratory tests are more discriminating. Specific laboratory testing is described in chapter sections on differential diagnosis. The principle, however, is to be specific in what tests you order and link them to specific choices on your differential diagnostic list or to determine whether a syndrome is secondary to a sus-