An Introduction to

Diagnosis and Management of Common Neurologic Disorders

Peritz Scheinberg, M.D.

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Peritz Scheinberg, M.D.

Professor and Chairman
Department of Neurology
University of Miami School of Medicine
Miami, Florida

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PREFACE

The objective of this book is to emphasize the diagnosis and management of the most common neurologic problems. Its intended audience is medical students, house officers, and physicians who encounter patients with neurologic disorders. Symptoms and signs are explained in the context of the presumed mechanisms of their causation, and details of management are included. Liberal use has been made of imaging techniques to illustrate specific points of anatomy and pathology.

This volume is the expanded third edition of the volume previously entitled *Modern Practical Neurology*. Chapters have been revised to include the latest advances in the clinical neurology. The chapter entitled "Disorders of the Spinal Cord and Adjacent Nerve Roots" has been completely rewritten; one chapter addresses intracranial tumors only; and an introductory chapter on pediatric neurology has been added.

The reader should be reminded that, like all single-authored texts, the opinions and views expressed herein may not be shared by all authorities or clinicians. They are my views, developed from personal experience, reading, and exposure to talented and informed colleagues, house officers, and students.

CONTENTS

CHAPTER 1

THE NEUROLOGIC EXAMINATION

The first prerequisite to the proper diagnosis and treatment of the patient with a neurologic disorder is the ability to perform a competent neurologic examination. To the many physicians who recall being intimidated by the vast array of nuclei and tracts that was their introduction to the anatomy of the nervous system, the task of evaluating some abnormality of these structures by a physical examination seems formidable indeed. In addition, some had a rather brief introduction to the neurologic examination during medical school without any subsequent instruction, so that the years have dimmed the original skills and knowledge. Whatever the reason, many physicians who feel perfectly comfortable when examining the heart, lungs, and abdomen feel completely inadequate about their ability to examine the nervous system and are therefore reluctant to deal with neurologic problems at all. Just as it is necessary to learn words to know a language, it is also essential to be able to appraise the nervous system intelligently before undertaking the management of a patient with a neurologic disturbance.

The fact is that the neurologic examination is not complicated. It is an application of rather simple and straightforward principles of the structure and function of the nervous system. The purpose of the neurologic examination is to localize the lesion. This does not preclude the possibility that a person may have neurologic disease in the presence of a normal neurologic examination. Indeed, rather significant CNS disease may be present despite a normal neurologic examination, and patients with a variety of neurologic complaints (e.g., headaches, dizziness, or pain in an extremity) may be entirely normal to examination. Before a decision can be made about the pathology of a lesion the physician must first localize it by history and examination. This localization is an orderly review of the function of the patient's central and peripheral nervous systems, including the various localizable functions of the cerebral hemispheres, brainstem, medulla, spinal cord, nerve roots, peripheral nerves, and muscles. Although it should be as complete as possible, the examination is not necessarily intricate and should always be modified to suit the situation. Obviously, a comatose patient cannot be examined in the same fashion as can an alert and cooperative patient, but with a little practice it is possible to adjust or modify the examination according to the circumstances and still obtain the data pertinent to the problem.

It goes without saying that a good history is essential to the interpretation of the neurologic examination. Often the experienced clinician can make the appropriate diagnosis from the history and is not often surprised by the findings on examination. A good history allows the examiner to focus on those aspects of the examination he considers to be most pertinent, thereby enabling him to detect ever more subtle pathology.

Good history-taking is a medical art form that is applicable to all organ systems. It implies the careful dissection of the patient's complaints by questions that spring from an understanding of the symptomatology of various disease states and the effects of pathology on different parts of the nervous system. A good history is never diffuse; it is pointed. The examiner finds out as much as possible about each system, calling on his knowledge of disease to formulate his questions. It is beyond the scope of this work to describe neurologic history-taking in detail, but the principle is always the same. Whether the patient's complaint is pain, weakness, dizziness, or difficulty in walking, the examiner finds out enough about that symptom to identify its anatomic origin and

obtain a clue as to its pathology. For example, if the patient's complaint is of weakness of an extremity, one must know:

Time of onset
Abrupt or gradual
Progressive, unchanged, or improved
Worse distally or proximally
Affected by activity or exercise
Association with other symptoms that give additional clues as to the location of the lesion, e.g., numbness or paresthesias, sphincter disorders, pain

In most instances the responses gradually fill in the spaces of the puzzle, giving a picture of the responsible pathology and allowing the examiner to approach the examination with greater confidence.

The remainder of the chapter outlines the neurologic examination. Each portion of the examination is treated separately and in some detail. It is important to get into the habit of following a pattern so as to avoid omissions. The following areas are considered:

Mental status	Motor function
Gait and station	Sensation
Head and spine	Reflexes
Cranial nerves	Autonomic and sphincter function

MENTAL STATUS

A great deal is learned about the patient's mental status during the initial interview and history-taking, before the formal testing is begun. It is necessary to take note of all of the following during the examination.

State of Consciousness

The state of consciousness may vary from alert to lethargic to stuporous to comatose. Depression of consciousness is a definite indication of organic brain disease, either structural, metabolic, or toxic. Interpretation of the significance of the altered state of consciousness is dependent on the remainder of the examination. A small brainstem lesion may result in coma, but there are practically always accompanying neurologic signs that aid in localizing the lesion. These and other distinguishing features of the evaluation of the comatose patient are discussed in Chapter 11.

Appearance, Behavior, Mood, Affect, Stream of Conversation, and Thought Content

Does the patient appear to be sick, in pain, or restless? Is his behavior appropriate to the occasion and to his complaints? A glaring disparity between the patient's behavior and his symptoms and signs is suggestive of a psychiatric disorder.

Is there evidence of depression or anxiety? Many patients compensate well, so that initial appearances are deceiving. It is therefore necessary to inquire about the symptoms of each, such as:

1. Bouts of gloominess and crying
2. Severe feelings of guilt and worthlessness
3. Reduced interest in surroundings, withdrawal, lack of motivation, early morning insomnia, lack of appetite, constipation

4. Restlessness, inner tension, tremor, irritability, swings of mood

Is the patient's conversation coherent, appropriate, and relevant? Is there evidence of delusions or hallucinations?

Intellectual Functions

Orientation (To Time, Place, and Person)

Disorientation is often associated with other evidence of confusion and may mean diffuse brain disease, intoxication, metabolic disturbance, or simple dementia.

Memory

Memory is defined by Webster as "the power or process of reproducing or recalling what has been learned and retained." It is a complex process which, for testing purposes, can be viewed as being composed of several functions, namely, the acquisition, retention, and recall of new information. It is now accepted that the areas of the brain specifically concerned with storing and retrieving information are the hippocampi and dorsal medial nuclei of the thalamus, and perhaps the fornices and mammillary bodies. Recent studies have implicated the median forebrain bundle as the neuronal structure involved in the dementia of Alzheimer's disease and offer strong evidence that memory is a cholinergic function. In Wernicke-Korsakoff syndrome, in which there is a defect in the ability to acquire new information, the prominent lesions are in the mammillary bodies and dorsal medial nuclei of the thalamus, and severe memory impairment may also occur as a result of diffuse brain disturbances, metabolic or structural.

Memory testing includes the following:

1. Digit span test. Most normal individuals can repeat five digits forwards and four digits backwards with no difficulty. This probably tests the patient's attention and may, in fact, be intact in individuals with significant dementia.

2. Three objects to remember for 5 min. The examiner tells the patient the names of three objects slowly and then has the patient repeat them to be sure he is attentive. The patient is then distracted for about 5 min by other tests and then asked to remember the objects. The complexity of this test can be varied.

3. Current events and recent presidents. In many instances the patient may recall events from childhood or early adult life and yet have major defects in more recently acquired information. As dementia progresses, even more ancient memories become dimmed or obliterated, suggesting that different groups of cells may be responsible for the chronological "layering" of memory. Head injuries commonly produce both anterograde and retrograde amnestic defects. The former is a reflection of inability to acquire new information; the latter denotes the vulnerability of information acquired immediately prior to the lesion. The return of retrograde memory function occurs in a stepwise fashion, the older memories returning first. In some instances the events just prior to the onset of the lesion are never recalled. This complex process is not well understood, although some have attributed it to the reinforcement by repetition of older memories.

Calculations

The parameter of calculations may be difficult to evaluate because it is so dependent on prior education and experience; but significant abnormalities in the absence of other cognitive defects suggest a disturbance in the dominant parietal lobe. The patient is asked to subtract sevens serially from 100 or to calculate how much change one would receive from a dollar following purchases

of stated amounts. Such questions test the patient's ability to concentrate as well as perform simple calculations; hence a disorder of attention may also cause difficulty in calculating. Localized lesions in the dominant parietal lobe may produce dyscalculia after accompanied by deficits in language-related tasks.

General Information, Insight, and Judgment

The patient's store of general information as well as his insight and judgment are also dependent on his previous experiences and socioeconomic status, as well as on memory functions and motivation. Subtle alterations in a college professor would not be detectable if he were tested similarly to a poorly educated laborer. The examiner inquires about recent presidents, world or local events, and asks the patient to interpret simple proverbs to determine whether he is excessively literal or capable of deducing meanings. Proverbs frequently used are: "People who live in glass houses should not throw stones." "A bird in the hand is worth two in the bush." "A stitch in time saves nine." Comparisons such as the difference between a lie and a mistake or a baby and a midget are also helpful in evaluating the patient's reasoning capacities.

Language Function

Aside from the intellectual satisfaction of attempting to understand the nature of a patient's language disorder, it is of practical importance to establish if there is any evidence of aphasia for three reasons: (1) It helps in the localization of a lesion to a fairly predictable site in the dominant hemisphere. (2) Evaluation of mental status is difficult in an aphasic or partially aphasic (dysphasic) patient. (3) Aphasia decidedly influences the patient's potential for rehabilitation. It is evident that the various mental status functions described above cannot be properly evaluated if the patient cannot understand what is said to him or cannot express himself properly. Aphasic patients are occasionally misdiagnosed as being demented or depressed unless subtle alterations in their ability to understand or communicate are recognized.

Aphasia is an impairment of language function following a lesion in the dominant hemisphere, most frequently in the posterior frontal or temporal lobe, or both. The left cerebral hemisphere is dominant in all right-handed individuals and in about half of sinistrals. Although it is not necessary to be able to classify in detail the various types of aphasia in order to recognize its existence, an understanding of the type of language dysfunction is helpful in lesion localization and in the type of speech therapy to be utilized.

There are many classifications of aphasia, just as there are many schemes for aphasia testing. The classification utilized by the Boston Veterans Administration Aphasia Research Center is a useful one; it classifies aphasias according to whether repetition is disturbed (Table 1.1).

TABLE 1.1. *Classification of aphasia*

Aphasia with abnormal repetition
Broca's aphasia
Wernicke's aphasia
Conduction aphasia
Aphasia with preserved repetition
Mixed transcortical aphasia
Transcortical motor aphasia
Transcortical sensory aphasia
Anomic aphasia
Total aphasia
Global aphasia

Broca's aphasia is the result of a lesion of some type in the dominant (usually left) frontal operculum, the posterior inferior portion of the frontal lobe. There are many names applied to it, viz., motor, executive, and nonfluent. The patient shows a reduced to absent verbal output. Speech is sparse, performed with great effort, and characterized by the use of substantive words with a paucity of prepositions, articles, and modifiers. Articulation is poor, the length of phrases is short, and there is dysprosody. Comprehension is usually good but rarely intact. Repetition of words or phrases is difficult. Dysnomia is almost always present, but this can usually be helped by prompting. Writing is usually severely disturbed, as is reading comprehension. Most patients with Broca's aphasia have a right hemiparesis, but small lesions causing aphasia may not produce a motor abnormality.

Wernicke's aphasia is caused by a lesion in the dominant temporal lobe, particularly in the superior-posterior part of the first temporal gyrus. The major abnormality is a severe disturbance of comprehension of spoken and written language along with an inability to repeat words. There is almost always a great deal of verbal output, but it is usually contextually meaningless, replete with substitutions of inexact or inappropriate words for the ones wanted. This substitution is termed paraphasia. Dysnomia is severe and not improved by prompting.

Conduction aphasia is thought to be the result of pathology in the white matter fasciculus that connects Broca's and Wernicke's areas. The patients have fluent paraphasic speech, fairly good comprehension, but severe difficulty with repetition. They can comprehend what they read but cannot read aloud because of paraphasic contamination. Writing is affected by insertion of inappropriate letters or reversal of words or letters, and dysnomia is common. The dysnomia is characterized by the use of incorrect phonemes so that the word produced may be unrecognizable to the listener (neologism).

The transcortical aphasias are characterized by either nonfluent (transcortical motor) or fluent (transcortical sensory) aphasia with preservation of repetition. These patients often exhibit echolalia, a tendency to repeat what the examiner has said, and may be able to complete overlearned phrases. The pathology of these lesions is not precisely established. In transcortical motor aphasia the pathologic site is usually anterior or superior to Broca's area. These aphasias have been described as an isolation or disconnection of the primary speech areas from other parts of the cortex.

Anomic aphasia, or variations thereof, may occur from a lesion in almost any part of the dominant hemisphere, particularly in the general region of the language centers. The patient manifests difficulty in finding the precise word with which to express himself and often substitutes a less appropriate one.

Global aphasia is said to exist when language loss is nearly complete. There is poor verbal output, and both comprehension and repetition are impaired. The pathology involves Broca's and Wernicke's areas and much of the brain between them.

Aphasia can be diagnosed with little difficulty by the trained observer, but detailed aphasia testing requires considerable time and often a great deal of patience on the part of the examiner. The Porch Index of Communicative Ability (PICA) is a popular, relatively brief (1 hr) test for aphasia that can be administered fairly easily, but it requires considerable experience for interpretation and scoring. Most clinicians rely on straightforward bedside observations performed as part of a routine neurologic examination to obtain a perspective of the patient's language disability and the presumed location of the responsible pathology.

Reading

Although reading ability may properly be considered a part of language function, its loss may occur without aphasia. Acquired dyslexia (i.e., impairment of reading function that occurs in a previously literate adult) usually denotes a lesion of the dominant parietal lobe and is often

accompanied by right homonymous hemianopsia. The anatomic localization of this lesion is classically in the splenium of the corpus callosum in the dominant hemisphere, thereby disconnecting the dominant angular and supramarginal gyri from the opposite visual cortex. Acquired dyslexia may take several forms, depending on the precise localization of the pathology. The patient may be unable to recognize either words or letters or may be able to read without understanding the significance of the words. The latter type of reading disorder is common in Alzheimer's disease. Primary dyslexia (i.e., difficulty in learning to read) is a more complex neurologic problem and is of little value as a sign of cerebral localization.

Praxis

Praxis is a term used to describe a variety of skilled, learned (noninstinctual) motor acts, e.g., drawing, copying figures, using a simple tool or pencil, putting on one's clothes, imitating another's movements. Apraxia means loss or impairment of such functions, and it may be caused by lesions in either parietal lobe and occasionally in the frontal lobes as well. The commonest form of apraxia can be described as ideomotor apraxia, in which the patient cannot perform an action to command, even though the command is understood and the patient has the ability to perform the action properly. One asks the patient how he would brush his teeth or comb his hair. The response will be to perform the act as if the toothbrush or comb were not present. Ideomotor apraxia usually occurs as a result of dominant hemisphere lesions. Dressing apraxia, inability to put on one's garments correctly, is classically a result of a lesion in the nondominant parietal lobe. Diffuse brain disturbances, metabolic or structural, may also cause apraxias.

General Observations

In addition to the above functions evaluated in a mental status examination, there are a number of other clues to the location of the patient's lesion that can be obtained from simple observation or questioning. Patients with nondominant parietal lobe lesions (usually right) may actually deny that they are ill even though the opposite limbs are paralyzed (anosognosia). They may even deny that the paralyzed body parts belong to them (asomatognosia). Another clue to nondominant hemisphere disease is motor impersistence, characterized by inability of the patient to sustain a simple motor act (e.g., keeping his eyes closed or an arm outstretched) even though no actual motor weakness is present. Further evidence of nondominant parietal lobe disease can be obtained by having the patient copy a few simple figures or draw the face of a clock with the hands of the clock pointing to a designated time. Patients with such lesions cannot copy figures or objects accurately, and they often crowd all the figures on the clock into the right half of the face (Fig. 1.1).

In frontal lobe disease (e.g., meningioma) the usual cognitive testing may reveal no defect in memory, language, calculations, or reading, but subtle defects in judgment and associations can be detected. These patients behave inappropriately, show poor judgment in both business and social settings, seem to be poorly motivated and inappropriately jocular, become irritable and unpredictable, and may dress in a slovenly manner. On examination these individuals may show primitive reflexes, which will be described in a later section.

Dominant parietal lobe lesions, specifically in the left supramarginal and angular gyri, may cause all or part of the Gerstmann syndrome, viz., dysgraphia, dyscalculia, right/left disorientation, and finger agnosia.

Temporal lobe lesions, particularly those causing partial complex seizures, are said to be associated with an increased incidence of schizophrenia and other behavior characteristics, such as obsessive/compulsive traits and hyperreligiosity.

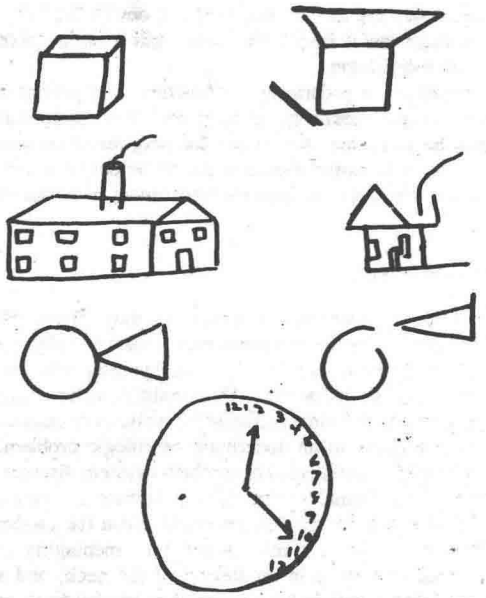

FIG. 1.1. Drawing and copying defects that occur particularly in right parietal lobe lesions but that may also occur in diffuse cortical disease. The patient has attempted to copy, on the right, figures drawn by the examiner on the left. He was also asked to draw the face of a clock with the hands pointing to 10 min after 2 o'clock. The crowding of the clock numbers to one side of the face is particularly characteristic of nondominant parietal lobe lesions. The distortions of the figures drawn by the patient are evident and denote a disorder of spatial perception.

Midline lesions, involving the genu of the corpus callosum or the mesial surfaces of the hemispheres, may be accompanied by a variety of mental status deficits including psychomotor retardation, lack of spontaneity, apathy, memory deficits, and abulia.

GAIT AND STATION

A reasonably practiced and observant physician can learn a great deal about the localization of a patient's neurologic disorder by watching him walk. Indeed, there is probably no single performance in the neurologic examination so revealing as the observation of gait and station. The observer watches the patient carefully first during normal walking, observing the way in which he moves his trunk, legs, and arms, noting the rhythm of the patient's movements, observing whether there is decreased or absent arm swing (associated movements) and whether the trunk is rigid or moves naturally, checking for foot drop and the ability to turn sharply and to start and stop abruptly and smoothly. A widened base in walking means ataxia even before gross stumbling or staggering occurs. The patient should be made to walk on his toes and then his heels to accentuate mild weakness of dorsiflexion or plantar flexion of the feet. Disturbances of vestibular or cerebellar function can be accentuated by tandem walking, heel to toe in a straight line, and can cause the patient to step out of the pattern or stagger to one side. Proprioceptive abnormalities (decrease in joint sensibility or position sense) are suspected if the patient complains of difficulty walking in the dark, and if the gait is characterized by slapping of the feet, exaggerated lifting of the feet, or ataxia.

The postures of the head, arms, and trunk are important in the evaluation of gait, as they may be affected in extrapyramidal or cerebellar disorders as well as in weakness of the extremities. Patients with early parkinsonism walk with few associated arm movements and turn in a characteristically rigid fashion, like a toy soldier. As the disease progresses, these patients may demonstrate festination, wherein the steps are increasingly small and rapid, until they are actually running. Certain dementias (particularly normal-pressure hydrocephalus) and frontal lobe lesions may be

accompanied by difficulty in initiating coordinated walking movements, retropulsion (falling backwards), and a strange awkwardness of gait. Although this is frequently termed gait apraxia, there is doubt that this meets the requirements for such a definition.

The Romberg test is designed to detect abnormalities in proprioceptive function. The patient is asked to stand with feet together. If the patient sways excessively, falls, or cannot maintain that posture with open eyes, a cerebellar lesion must be suspected. A positive Romberg test is present if the patient has difficulty maintaining that posture with closed eyes; this should be confirmed by the sensory examination of the lower extremities, which should indicate impairment of position, vibration sensibility, or both.

HEAD AND SPINE

Examination of the head and spine requires little additional time and may pay huge dividends, particularly if one is examining a comatose patient, when the examiner must look carefully for any signs of head injury (e.g., abrasions or lacerations) or Battle's sign (ecchymoses over the mastoid region), which frequently accompanies basilar skull fracture. Abnormalities in head size or configuration may be helpful in diagnosis, particularly in children. Similarly, scoliosis or unusual spine configurations should be noted, for they may point to an underlying neurologic problem. Scoliosis is particularly common in muscular dystrophy, certain types of cerebellar system diseases, and slow-growing spinal cord tumors, e.g., astrocytoma. Spinal tenderness may indicate underlying bone disease (e.g., tumor or infection), and flexibility of the spine is important when the patient complains of neck, low back, or extremity pain. If there is any suspicion of meningitis or subarachnoid hemorrhage, one must look for neck rigidity, pain on flexion of the neck, and a positive Kernig or Brudzinski sign. When examining a patient with complaints of low back or lower extremity pain, a useful trick is to have the patient bend forward as far as possible without bending the knees, at which time percussion or finger pressure over the sciatic notch (the upper/outer quadrant of the gluteus muscle) may provoke local or radiating pain if the patient's problem is caused by compression of one of the roots of the sciatic nerve, as by a herniated disc.

CRANIAL NERVES

The cranial nerves are usually examined in sequence. Appreciation of normal or abnormal function is dependent on a few neuroanatomic principles.

Olfactory

It is helpful to test for appreciation, but not necessarily identification, of aromatic, nonirritating odors (e.g., coffee, peppermint, cloves). Most of the time it is sufficient to know if the patient can taste food flavors, for this faculty is dependent on intact olfaction. Olfaction should be evaluated in any patient suspected of having frontal lobe disease (viz., dementia, personality change, or unexplained gait disorders) because of the possibility of a tumor on the orbital surface of the frontal lobes in such an instance. Other causes for anosmia include local nasal or nasopharyngeal disease, head trauma with injury to the olfactory bulbs, psychogenic disturbance, pernicious anemia, multiple sclerosis, diabetes mellitus, and zinc deficiency.

Optic

Tests for visual function include evaluation of visual acuity and fields of vision. A Snellen eye chart or a J. G. Rosenbaum Pocket Vision Screener, which is inexpensive and commercially available, may be used. The card is held in good light, and best corrected visual acuity is measured in each eye, allowing the patient to hold the card at the most comfortable distance. If the patient's

best corrected visual acuity is faulty, it means that there is a defect in the macula or the optic nerve, provided there is no abnormality in the ocular media (lens, vitreous, etc.). Such a finding always demands explanation, which should be sought from an ophthalmologist if it is not apparent to the examiner.

A person's visual fields include all that he can see when he fixes his gaze on one target. The examination should therefore include the area around the fixation points of both eyes as well as the periphery. Defects in the visual fields may be caused by lesions of the retina, optic nerve, chiasm, optic tracts, or optic radiations. The abnormalities caused by lesions in each of these areas are fairly stereotyped, so that the location of the lesion can usually be predicted from the pattern of the field deficit. Lesions of the optic nerve cause central scotomas or complete blindness in the affected eye. The field deficit caused by lesions of the optic chiasm depends on the portion of the chiasm involved, but the usual and "characteristic" chiasmatic deficit is bitemporal hemianopsia, caused by interruption of the crossing fibers in the center of the chiasm by a pituitary or other perichiasmal tumor. Lesions involving the visual pathways posterior to the chiasm, namely, the optic tract, lateral geniculate body, optic radiations, or visual cortex, cause homonymous hemianopsia (i.e., a visual defect in corresponding or homonymous half-fields of both eyes). The congruity of the visual field defect refers to the similarity of size and shape of the paired defects. The congruity of the defect may give a clue as to its location. A complete anatomic lesion will cause a complete homonymous field defect, which, therefore, gives little clue as to the anatomic location of the lesion. Optic tract lesions are rarely complete and the resultant hemianopsia is therefore rarely congruous. Lesions of the anterior temporal lobe may characteristically cause a superior quadrantanopsia, which is incongruous because of the anatomic disposition of Meyer's loop. More posteriorly the fibers in the geniculocalcarine radiations become increasingly contiguous so that lesions are somewhat likelier to cause congruous field defects or complete homonymous hemianopsias. The absence of any other neurologic deficit in the presence of a congruous field defect or complete hemianopsia points to the occipital lobe as the location of the lesion. Macular sparing is said to be characteristic of occipital lobe lesions, presumably due to a double blood supply to the area of macular representation in the cortex. In some instances macular sparing may be an artifact of poor ocular fixation during the testing.

There are several methods of examining the visual fields quickly and accurately by confrontation. All depend on the patient's ability to fixate vision reasonably well on one object (examiner's nose). The four quadrants of vision of each eye should be tested.

One way is to compare the patient's fields with those of the examiner by using a small object, remembering that the fields include the central 30° of vision as well as the periphery. Other methods include asking the patient to compare the appearance of the examiner's hands when they are held up simultaneously, on either side of the vertical meridian of each eye. One hand will appear darker or less clear than the other if there is a defect in that field of vision. In this way the nasal and temporal fields of the patient's eye can be compared to each other. Another similar method is to use a red object rather than one's hand as the target. The red color will appear brighter in a normal than in a defective field.

There are other even simpler methods that must occasionally be resorted to, depending on the patient's state of awareness or cooperation. On occasion the examiner must rely on the patient's blinking or flinching to a threatening hand movement aimed at his face from each field of vision. Persistence and resourcefulness are important assets when adapting the testing procedure to the patient's capabilities.

The central 30° of the visual field is readily examined with a tangent screen. With the patient seated 1 m in front of the screen and fixating the central point with the eye being examined, the examiner uses a light projected on the screen or light objects to plot the fields. The exam gives exact information of the central margins of a field defect or the size and location of a central or paracentral scotoma.