

eMedicine Specialties > Clinical Procedures > none

Neurological History and Physical Examination

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Neurological History

"From the brain and the brain only arise our pleasures, joys, laughter and jests, as well as our sorrows, pains, griefs, and tears.... These things we suffer all come from the brain, when it is not healthy, but becomes abnormally hot, cold, moist or dry."

—Hippocrates

The Sacred Disease, Section XVII

Taking the patient's history is traditionally the first step in virtually every clinical encounter. A thorough neurologic history allows the clinician to define the patient's problem and, along with the result of physical examination, assists in formulating an etiologic and/or pathologic diagnosis in most cases.^[1]

Solid knowledge of the basic principles of the various disease processes is essential for obtaining a good history. As Goethe stated, "The eyes see what the mind knows." To this end, the reader is referred to the literature about the natural history of diseases. The purpose of this article is to highlight the process of the examination rather than to provide details about the clinical and pathologic features of specific diseases.

The history of the presenting illness or chief complaint should include the following information:^[2]

- Symptom onset (eg, acute, subacute, chronic, insidious)
- Duration
- Course of the condition (eg, static, progressive, or relapsing and remitting)
- Associated symptoms, such as pain, headache, nausea, vomiting weakness, and seizures

Pain should be further defined in terms of the following:

- Location (Ask the patient to point with one finger, if possible.)
- Radiation (Pay attention to any dermatomal relationship.)
- Quality (stabbing, stinging, lightninglike, pounding, etc)
- Severity or quantity (Estimate functional limitation.)
- Precipitating factors (stress, periods, allergens, sleep deprivation, etc)
- Relieving factors (sleep, stress management, etc)
- Diurnal or seasonal variation

Important miscellaneous factors of the history include the following:

- Results of previous attempts to diagnose the condition

- Any previous therapeutic intervention and the response to those treatments

A complete history often defines the clinical problem and allows the examiner to proceed with a complete but focused neurologic examination.

Neurologic Examination

The neurologic examination is one of the most unique exercises in all of clinical medicine. Whereas the history is the most important element in defining the clinical problem, neurologic examination is performed to localize a lesion in the central nervous system (CNS) or peripheral nervous system (PNS). The statement has been made, "History tells you what it is, and the examination tells you where it is." The history and examination allow the neurologist to arrive at the etiology and pathology of the condition, which are essential for treatment planning.^[3,4]

Unlike many other fields of medicine in which diseases are visible (eg, dermatology, ophthalmology) or palpable (eg, surgery), neurology is characterized by conditions that may be detected only by applying specific examination techniques and logical deduction, except when telltale cutaneous markers or other stigmata suggest the diagnosis. Considerable insight and intuition are required to interpret the symptoms and signs observed during neurologic examination. These features make the neurologic history and physical examination both challenging and rewarding.

A properly performed neurologic examination may take 90 minutes or even longer for the novice. Experienced neurologists take substantially less time and can frequently grasp the essential features of a clinical condition quickly. What might appear to be a complex problem of localization for the referring physician may turn out to have a simple explanation, and the neurologic consultation may help to avoid extensive testing.

Neurologic examination in the era of imaging

With the advent of CT scanning in the early 1970s, the future clinical role of the neurologist was questioned. During one of his visits to the United States, Dr. McDonald Critchley was asked what he thought would be the future of neurology in the era of CT. His answer was most enlightening: "CT scanning will take away the shadows of neurology, but the music will still remain." These prophetic words still ring true despite the advent of MRI, positron emission tomography (PET), and functional neuroimaging of all types.

It has been said that "neurology owes more to its disorders than those disorders owe to neurology." This is because much knowledge has come from previous observations of neurologic conditions, because the eponyms for the diagnoses were sometimes long, and because so little was previously offered in the terms of cures such that the specialty was ridiculed as one that was "long on diagnosis and short on treatment." Fortunately, technologic advances have changed that perception.

Steps in the neurologic examination

In examining a patient, abnormalities of function lead to localization and, eventually, to the pathophysiology.^[5] For the purpose of simplicity, the neurologic examination is divided into several steps. When mastered, these steps become second nature to the examiner, and the process of evaluating the patient proceeds smoothly, even though the steps are not always necessarily performed in the same order. These steps include the following:

- Higher functions
- Cranial nerves (CNs)
- Sensory system
- Motor system
- Reflexes
- Cerebellum
- Meninges

- System survey

Tools required

In addition to the stethoscope and the usual office supplies (eg, gloves, tongue depressors), the neurologist should have an ophthalmoscope, a reflex hammer, and a tuning fork.

A pin (Wartenberg) wheel was once a favorite tool of many neurologists because it was easy to use for sensory (pinprick) testing. Unless it is disposable (commercially available), this wheel is no longer recommended because of the risk of transmitting infection. The use of sterile safety pins (to be discarded after each use) is recommended.

The final section of this article includes a Definition of Terms.

Examination of the Higher Functions

Components of Higher Functions

Higher functions include gait, speech, and mental status.^[6] These are referred to as higher functions because human bipedal gait, receptive and expressive speech, and cognitive function are more sophisticated than similar functions of any other member of the animal kingdom.

Gait

Gait is the attitude of a person in the upright position. Abnormal types are described below.

Hemiparetic gait

In hemiparesis, facial paresis may not be obvious. In mild cases, subtle features of facial paralysis (eg, flattening of the nasolabial fold on 1 side compared to the other, mild asymmetry of the palpebral fissures or of the face as the patient smiles) may be sought. The shoulder is adducted; the elbow is flexed; the forearm is pronated, and the wrist and fingers are flexed. In the lower extremities, the only indication of paresis may be that the ball of the patient's shoe may be worn more on the affected side.

In severe cases, the hand may be clenched; the knee is held in extension and the ankle is plantar flexed, making the paralyzed leg functionally longer than the other. The patient therefore has to circumduct the affected leg to ambulate.

In hemiplegic patients in whom all the paralysis is on the same side of the body, the lesion is of the contralateral upper motor neuron. In most cases, the lesion lies in the cortical, subcortical, or capsular region (therefore above the brainstem). In the alternating or crossed hemiplegias, CN paralysis is ipsilateral to the lesion, and body paralysis is contralateral. In such cases, CN paralysis is of the lower motor neuron type, and the location of the affected CN helps determine the level of the lesion in the brainstem. Therefore, paralysis of CN III on the right side and body paralysis on the left (Weber syndrome) indicates a midbrain lesion, whereas a lesion of CN VII with crossed hemiplegia (Millard-Gubler syndrome) indicates a pontine lesion, and CN XII paralysis with crossed hemiplegia (Jackson syndrome) indicates a lower medullary lesion.

Ataxic gait

In ataxia, the patient spreads his or her legs apart to widen the base of support to compensate for the imbalance while standing or walking. In severe cases, patients stagger as they walk. The heel-to-toe or tandem walking maneuvers and standing on 1 leg uncover subtle forms of ataxia.

Ataxia results from midline lesions of the cerebellum and may be isolated or associated with other cerebellar findings (see Cerebellar signs). When the lesion is unilateral, the patient may veer to the side of the lesion. With bilateral cerebellar involvement, the patient may fall to either side.

Shuffling gait

The individual takes short steps to the point of practically not moving forward or making little progress. In other words, the patient appears to shuffle his or her legs rather than put them forward. In some patients, the steps (albeit short) and pace may vary with a

tendency for the patient to accelerate (festinating gait) as he or she walks. Both types are seen in Parkinson disease and may be associated with other extrapyramidal signs.

Steppage gait

In steppage (high-stepping, slapping), the individual takes high steps as if climbing a flight of stairs while walking on a level surface. This peculiar gait pattern results from the patient trying to avoid injury to the feet (from dragging them) by stepping high. However, as the patient puts the feet down 1 by 1, they slap the ground, hence the description of a foot-slapping gait. This is 1 condition that can be diagnosed even before the patient enters the room because the sound is so characteristic.

Steppage gait is seen in chronic peripheral neuropathies and can be the result of the functional elongation of the legs due to bilateral drop foot.

Spastic or scissor gait

In this condition, the legs are held in adduction at the hip and the thighs rub against each other as the patient walks. Spasm of the inner thigh muscles also occurs. If the spasm is severe, with each advancing step the knees tend to slide over each other like the blades of a pair of scissors. This is typically seen in cerebral diplegia, a form of cerebral palsy.

Antalgic gait

Patient favors the affected painful (usually lower) extremity and walks, putting weight on the normal leg. The hand held over hip on the affected side is typical in patients with radicular pain.

Speech

Speech enables communication between individuals. Abnormalities include dysphonia, dysarthria, and dysphasia or aphasia.

Dysphonia or aphonia

Dysphonia is the impairment or inability to phonate. As a result, the voice becomes hoarse. In extreme cases, it is absent, and the patient is mute.

The most frequent cause of this problem is the common cold, which results in dysphonia due to inflammation of the larynx. Dysphonia may also occur in patients with hypothyroidism, as a result of thickening of the vocal cords from amyloid deposits. Neurologic causes include unilateral recurrent laryngeal nerve paralysis and lesions of the vagus nerve. Intermittent hoarseness may affect patients with vagus nerve stimulator implants, which are used for the treatment of certain medically intractable forms of epilepsy (MIE) and pharmaco-resistant depression (PRD).

Dysarthria or anarthria

Dysarthria is the inability to articulate spoken words. The quality of oration is impaired, but the content remains intact (eg, slurred speech). The patient's ability to understand and synthesize speech remains intact. It results from paralysis of pharyngeal, palatal, lingual, or facial musculature. It also is observed with cerebellar lesions and/or disease (eg, scanning or staccato speech).

Dysphasia or aphasia

In dysphasia, the ability to process language is impaired, resulting in an inability to understand (ie, receptive or sensory or Wernicke aphasia), transfer signals from the Wernicke to the Broca area (ie, conduction aphasia), or properly execute speech (ie, expressive, motor, or Broca aphasia). The combination of Broca and Wernicke aphasias is referred to as global aphasia.

Table 1 summarizes the essential features of common dysphasias (aphasias).

Table 1. Essential Features of Common Dysphasias

Type of Dysphasia	Fluency	Comprehension	Naming	Localization
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Broca	Nonfluent	Intact	Impaired	Broca area
Wernicke	Fluent	Impaired	Impaired	Wernicke area
Conduction	Fluent	Intact	Impaired	Arcuate fasciculus
Global	Nonfluent	Impaired	Impaired	Broca and Wernicke areas

Transcortical aphasia

Another function that is impaired in all 4 of the aphasias mentioned above is repetition. This finding is important in the diagnosis of transcortical aphasias. When repetition is preserved in a patient with Broca aphasia, it signifies transcortical motor aphasia, and the lesion is anterior to the Broca area. When repetition is preserved in Wernicke aphasia, it is called transcortical sensory aphasia, and the lesion is posterior to the Wernicke area. Transcortical mixed aphasia and global aphasia are similar except for the preservation of repetition, and results from combined lesions anterior to the Broca and Wernicke areas, respectively.

Mental Status

Mental status evaluation includes testing of memory, orientation, intelligence, and the other aspects of the patient's psychic state. Only the first 3 are discussed here. When overt symptoms or signs of a psychic disturbance are present, psychiatric evaluation should be considered.

Memory

Memory is the ability to register and recall prior sensory input. Recent and remote memory functions are differently affected depending on the disease process. Remote memory is relatively preserved in chronic dementing processes, with major disturbances in the attention span and recent memory. On the contrary, all aspects of memory are impaired in acute encephalopathies.

Orientation

Orientation is an individual's cognitive sense of his status in time, place, and person. These functions are affected in the same order as they are in organic disease. In other words, the sense of time is first to be impaired in organic dysfunction, and the sense of person is the last to be lost. However, the order may be disturbed in psychological dysfunction.

A patient who does not know who he or she is, but at the same time can tell the time and is oriented in place, is more likely to have a psychological disturbance than to have an organic etiology for the condition. Nonetheless, rare cases of isolated amnesia have been reported.

Intelligence

Intelligence is the ability to quickly and successfully apply previous knowledge to a new situation and to use reason in solving problems. Vocabulary, fund of knowledge, calculations (eg, serial-7 calculations), abstraction (eg, use of proverbs), and judgment (eg, what to do with a found wallet) are good indicators of intelligence.

Psychological disturbances

A brief survey of the other aspects of psychological function may be helpful in revealing abnormalities of thought process (eg, circumstantiality and tangentiality); of perception (eg, illusions and hallucination); or of thought content (eg, delusions of grandeur). Patients with these findings should be referred for appropriate evaluation.

Examination of the Cranial Nerves

Of the 12 CNs, some are named according to their function. Examples of these are the olfactory (smell), optic (vision), oculomotor (eye movements), abducens (abduction of the eye), facial (facial expression), and vestibulocochlear or statoacoustic (hearing and balance) nerves. Others are named for their relationship to neighboring structures (trochlear nerve), appearance (trigeminal nerve), extent of distribution (vagus nerve), composition (spinal accessory nerve), or location (hypoglossal nerve).^[7,8]

- Trochlear: Its midsection extends over a trochlea or pulley to reach its insertion on the inferior aspect of the globe.
- Trigeminal: The nerve divides into 3 divisions distal to the Gasserian ganglion.
- Vagus: The vagabond or wanderer, it travels long distances in the body.
- Spinal accessory: This nerve is composed of rootlets from the spinal cord in addition to its medullary component.
- Hypoglossal: Its course is sublingual in the neck.

Knowing the names of the CNs makes it easy to remember their function, thereby making their examination self-evident. The following mnemonic is helpful in recalling the names of the CNs: Oh, oh, oh; to trek and feel a great valley; ah! ha! Another is this: On old Olympus towering tops, a Finn and German viewed some hops.

Olfactory nerve - CN I

The olfactory nerves consist of small unmyelinated axons that originate in the olfactory epithelium in the roof of the nasal cavity; they pierce the cribriform plate of the ethmoid and terminate in the olfactory bulb. Lesions of the nerve result in parosmia (altered sense of smell) or anosmia (loss of smell).

The common cold is the most frequent cause of dysfunction. Dysfunction can be associated with fractures of the cribriform plate of the ethmoid bone. Frontal lobe tumors may compress the olfactory bulb and/or tracts and cause anosmia, but this is rare occurrence.

Olfactory function is tested easily by having the patient smell common objects such as coffee or perfume. Commercially available scented scratch papers may also be used.

Optic nerve - CN II

The optic nerve is a collection of axons that relay information from the rods and cones of the retina. The temporal derivations reach the ipsilateral and the nasal derivations the contralateral superior colliculi and the lateral geniculate bodies. From there, axons extend to the calcarine cortex by means of the optic radiation, traversing the temporal (Myer loop) and parietal lobes. Fibers responsible for the pupillary light reflex bypass the geniculate body and reach the pretectal area, from where they innervate the parasympathetic (midline) portion of the third-nerve nucleus, enabling the consensual pupillary reflex.^[9]

The following testing is appropriate:

- Acuity, by using the Snellen chart (near and distant vision)
- Visual fields, by means of confrontation or perimetry if indicated
- Color, with use of an Ishihara chart or by using common objects, such as a multicolored tie or color accent markers
- Funduscopy

Lesions of the visual pathways result in blindness and pupillary abnormalities, such as the Marcus-Gunn pupil (retinal or optic nerve disease), scotomata, quadrant or hemianopsias (optic tract and radiation), and hemianopsias with macular sparing (calcarine cortex).

Oculomotor nerve - CN III

The oculomotor nucleus of the nerve is located in the midbrain and innervates the pupillary constrictors; the levator palpebrae superioris; the superior, inferior, and medial recti; and the inferior oblique muscles. Lesions of CN III result in paralysis of the ipsilateral upper eyelid and pupil, leaving the patient unable to adduct and look up or down. The eye is frequently turned out (exotropia). In subtle cases, patients complain of only diplopia or blurred vision. Lesions at the nucleus of the third nerve cause bilateral ptosis, in addition to the findings mentioned above. The exotropia seen in CN III paralysis can be distinguished from that in internuclear ophthalmoplegia because in the latter convergence is preserved.

Paralysis of CN III is the only ocular motor nerve lesion that results in diplopia in more than 1 direction, distinguishing itself from CN IV paralysis (which also can result in exotropia). Pupillary involvement is an additional clue to involvement of CN III. Pupil-sparing CN

III paralysis occurs in diabetes mellitus, vasculitides of various etiologies, and certain brainstem lesions such as due to multiple sclerosis.

Trochlear nerve - CN IV

The nucleus of the nerve is located in the midbrain. It innervates the superior oblique muscle, which incycloducts and infraducts the eye. Trochlear nerve typically allows a person to view the tip of his or her nose.

An isolated right superior oblique paralysis results in exotropia to the right (R), double vision that increases on looking to the (L), and head tilt to the right (R). The mnemonic is R, L, R (ie, the marching rule). The rule is L, R, L for left superior oblique paralysis. This rule and the lack of ptosis and/or pupillary involvement allow easy distinction of the exotropia of CN IV paralysis from that seen in CN III paralysis.

Trigeminal nerve - CN V

The nucleus of the nerve stretches from the midbrain (ie, mesencephalic nerve) through the pons (ie, main sensory nucleus and motor nucleus) to the cervical region (ie, spinal tract of the trigeminal nerve). It provides sensory innervation for the face and supplies the muscles of mastication.

Paralysis of the first division (ophthalmic; V1) is usually seen in the superior orbital fissure syndrome and results in sensory loss over the forehead along with paralysis of CN III and CN IV. Paralysis of the second division (maxillary; V2) results in loss of sensation over the cheek and is due to lesions of the cavernous sinus; it also results in additional paralysis of V1, CN III and CN IV. Isolated V2 lesions result from fractures of the maxilla. Complete paralysis of CN V results in sensory loss over the ipsilateral face and weakness of the muscles of mastication. Attempted opening of the mouth results in deviation of the jaw to the paralyzed side.

Abducens nerve - CN VI

The nucleus of the nerve is located in the paramedian pontine region in the floor of the fourth ventricle. It innervates the lateral rectus, which abducts the eye. Isolated paralysis results in esotropia and inability to abduct the eye to the side of the lesion. Patients complain of double vision on horizontal gaze only. This finding is referred to as horizontal homonymous diplopia, which is the sine qua non of isolated CN VI paralysis. Paralysis of CN VI may result from increased intra cranial pressure without any lesion in the neuraxis, and it may result in false localization if one is not aware of it.

Facial nerve - CN VII

The nucleus of the nerve lies ventral, lateral, and caudal to the CN VI nucleus; its fibers elevate the floor of the fourth ventricle (facial colliculus) as they wind around the CN VI nucleus. The nerve leaves the cranial cavity through the stylomastoid foramen and innervates the muscles of facial expression and the stapedius.

Although it is considered a pure motor nerve, it also innervates a small strip of skin of the posteromedial aspect of the pinna and around the external auditory canal. The nervus intermedius of Wrisberg conducts taste sensation from the anterior two thirds of the tongue and supplies autonomic fibers to the submaxillary and sphenopalatine ganglia, which innervate the salivary and lacrimal glands.

A lower-motor-neuron lesion of the nerve, also known as peripheral facial paralysis, results in complete ipsilateral facial paralysis; the face draws to the opposite side as the patient smiles. Eye closure is impaired, and the ipsilateral palpebral fissure is wider. In an upper motor neuron lesion, also known as central facial paralysis, only the lower half of the face is paralyzed. Eye closure is usually preserved. In peripheral facial paralysis, different types of clinical presentations are seen with nerve lesions at 4 levels, as described below.

- Lesions of the meatal or canalicular segment: Facial paralysis with hearing loss (without hyperacusis) and loss of taste in the anterior two thirds of the tongue imply lesions in the internal auditory canal from fracture of the temporal bone or at the cerebellopontine angle from compression by a tumor.
- Lesions of the labyrinthine or fallopian segment
 - Lesions that spare hearing (with hyperacusis) indicate lesions further down the course of the nerve.

- Loss of taste in the anterior two thirds of the tongue and loss of tearing imply lesions that involve the chorda tympani and the secretomotor fibers to the sphenopalatine ganglion in the labyrinthine segment, proximal to the greater superficial petrosal nerve.
- With lesions distal to the greater superficial petrosal nerve, lacrimation is normal but hyperacusis is still present. Genuculate lesions in this segment cause pain in the face.
- Lesions of the horizontal or tympanic segment: The lesion is proximal to the departure of the nerve to the stapedius and results in hyperacusis, loss of taste in the anterior two thirds of the tongue, and facial motor weakness.
- Lesions of the mastoid or vertical segment: Hyperacusis is present if the lesion is proximal to the nerve to the stapedius. It is absent if the lesion is distal to the nerve to the stapedius, and only loss of taste and facial paralysis occur. If the lesion is beyond the chorda tympani in the vertical segment (as in lesions of the stylomastoid foramen), taste is spared and only facial motor paralysis is seen.

Vestibulocochlear nerve - CN VIII

The vestibulocochlear or statoacoustic nerve enters the brainstem at the pontomedullary junction and contains the incoming fibers from the cochlea and the vestibular apparatus, forming the eighth CN. It serves hearing and vestibular functions, each of which is described separately. Hearing loss may be conductive or sensorineural. Three tests help in evaluating the auditory component of the nerve.

The Weber test involves holding a vibrating tuning fork against the forehead in the midline. The vibrations are normally perceived equally in both ears because bone conduction is equal. In conductive hearing loss, the sound is louder in the abnormal ear than in the normal ear. In sensorineural hearing loss, lateralization occurs to the normal ear. The sensitivity of the test can be increased (up to 5 dB) by having the patient block his or her external ear canals by simultaneously pressing the index fingers at the introit.

To perform the Rinne test, the vibrating tuning fork is placed over the mastoid region until the sound is no longer heard. It is then held at the opening of the ear canal on the same side. A patient with normal hearing should continue to hear the sound. In conductive hearing loss, the patient does not continue to hear the sound, since bone conduction in that case is better than air conduction. In sensorineural hearing loss, both air conduction and bone conduction are decreased to a similar extent.

In the Schwabach test, the patient's hearing by bone conduction is compared with the examiner's hearing by placing the vibrating tuning fork against the patient's mastoid process and then to the examiner's. If the examiner can hear the sound after the patient has stopped hearing it, then hearing loss is suspected.

The vestibular portion of the nerve enters the brainstem along with the cochlear portion. It transmits information about linear and angular accelerations of the head from the utricle, saccule, and semicircular canals of the membranous labyrinth to the vestibular nucleus. Linear acceleration is monitored by the macules in the utricles and saccules; angular acceleration is monitored by the cristae contained in the ampullae in the semicircular canals. These signals reach the superior (Bechterew), lateral (Deiters), medial (Schwalbe), and inferior (Roller) nuclei and project to the pontine gaze center through the medial longitudinal fasciculus; to the cervical and upper thoracic levels of the spinal cord through the medial vestibulospinal tract; to the cervical, thoracic, and lumbosacral regions of the ipsilateral spinal cord through the lateral vestibulospinal tract; and to the ipsilateral flocculonodular lobe, uvula, and fastigial nucleus of the cerebellum through the vestibulocerebellar tract.

The Romberg test is performed to evaluate vestibular control of balance and movement. When standing with feet placed together and eyes closed, the patient tends to fall toward the side of vestibular hypofunction. When asked to take steps forward and backward, the patient progressively deviates to the side of the lesion. Results of the Romberg test may also be positive in patients with polyneuropathies, and diseases of the dorsal columns, but these individuals do not fall consistently to 1 side as do patients with vestibular dysfunction.

Another test is to ask the patient to touch the examiner's finger with the patient's hand above the head. Consistent past pointing occurs to the side of the lesion. Provocative tests include the Nysten-Bárány test and caloric testing (see Ancillary signs).

Glossopharyngeal nerve - CN IX

The nucleus of the nerve lies in the medulla and is anatomically indistinguishable from the CN X and CN XI nuclei (nucleus

ambiguous). Its main function is sensory innervation of the posterior third of the tongue and the pharynx. It also innervates the pharyngeal musculature, particularly the stylopharyngeus, in concert with the vagus nerve.

Vascular stretch afferents from the aortic arch and carotid sinus, as well as chemoreceptor signals from the latter, travel in the nerve of Hering to join the glossopharyngeal nerve; they reach the nucleus solitarius, which in turn is connected to the dorsal motor nucleus of the vagus and plays a part in the neural control of blood pressure.

Lesions affecting the glossopharyngeal nerve result in loss of taste in the posterior third of the tongue and loss of pain and touch sensations in the same area, soft palate, and pharyngeal walls. CN IX and CN X travel together, and their clinical testing is not entirely separable. Therefore, examination of CN IX is discussed with that of the vagus nerve.

Vagus nerve - CN X

Starting in the nucleus ambiguus, the vagus nerve has a long and tortuous course providing motor supply to the pharyngeal muscles (except the stylopharyngeus and the tensor veli palati), palatoglossus, and larynx. Somatic sensation is carried from the back of the ear, the external auditory canal, and parts of the tympanic membrane, pharynx, larynx, and the dura of the posterior fossa. It innervates the smooth muscles of the tracheobronchial tree, esophagus, and GI tract up to the junction between the middle and distal third of the transverse colon.

The vagus provides secretomotor fibers to the glands in the same region and inhibits the sphincters of the upper GI tract. Along with visceral sensation from the same region, the nerve participates in vasomotor regulation of blood pressure by carrying the fibers of the stretch receptors and chemoreceptors (ie, aortic bodies) of the aorta and providing parasympathetic innervation to the heart.

The pharyngeal gag reflex (ie, tongue retraction and elevation and constriction of the pharyngeal musculature in response to touching the posterior wall of the pharynx, tonsillar area, or base of the tongue) and the palatal reflex (ie, elevation of the soft palate and ipsilateral deviation of the uvula on stimulation of the soft palate) are decreased in paralysis of CN IX and CN X. In unilateral CN IX and CN X paralysis, touching these areas results in deviation of the uvula to the normal side.

Unilateral paralysis of the recurrent laryngeal branch of CN X results in hoarseness of voice. Bilateral paralysis results in stridor and requires immediate attention to prevent aspiration and its attendant complications.

Spinal accessory nerve - CN XI

From the nucleus ambiguus, the spinal accessory nerve joins the vagus nerve in forming the recurrent laryngeal nerve to innervate the intrinsic muscles of the larynx. The spinal portion of the nerve arises from the motor nuclei in the upper 5 or 6 cervical segments, enters the cranial cavity through the foramen magnum, and exits through the jugular foramen, and provides motor innervation to the sternocleidomastoid (SCM) and the mid and upper thirds of the trapezius.

In testing, functional symmetry of the SCM and the trapezius muscles should be evaluated. Have the patient push the face against resistance to the right and to the left. When the right SCM is weak, pushing to the opposite (ie, left) side is impaired, and vice versa. Shrugging of the shoulder is impaired ipsilaterally when the trapezius is weak.

Hypoglossal nerve - CN XII

The nucleus of this nerve lies in the lower medulla, and the nerve itself leaves the cranial cavity through the hypoglossal canal (anterior condylar foramen). It provides motor innervation for all the extrinsic and intrinsic muscles of the tongue except the palatoglossus. To test the hypoglossal nerve, have the patient protrude the tongue; when paralyzed on 1 side, the tongue deviates to the side of paralysis on protrusion.

Examination of the Sensory and Motor Systems

Sensory System

Noncortical sensory system

This is constituted by the peripheral nerves with their central pathways to the thalamus. Light touch, pain, heat, cold, and vibration sensations can be included in this group.

Light touch is tested by touching the skin with a wisp of cotton or tissue. Pain is tested by using a sharp object such as an open safety pin. Temperature can be tested by touching the patient's skin with 2 test tubes, 1 with warm water and the other with cold water. Compare the 2 sides and also to a benchmark, such as the patient's own forehead (assuming sensation there is normal). Vibration is tested with a tuning fork, preferably with a frequency of 128 Hz. Compare findings on the 2 sides, and also compare findings with those in the same body part of the examiner.

Cortical sensory system

The cortical sensory system includes the somatosensory cortex and its central connections. This system enables the detection of the position and movement of the extremities in space (ie, kinesthetic sensation), size and shape of objects (ie, stereognosis), tactile sensations of written patterns on the skin (ie, graphesthesia), and tactile localization and tactile discrimination on the same side or both sides of the body.

Position sensation is tested with the patient's eyes closed. The examiner moves various joints, being sure to hold the body part in such a way that the patient may not recognize movement simply from the direction in which the patient may feel the pressure from the examiner's hand.

Stereognosis is tested by placing some familiar object (eg, ball, cube, coin) in the patient's hand while his or her eyes are closed and asking the patient to identify the object. Inability to recognize the size or shape is referred to as astereognosis. Agraphesthesia is the inability to recognize letters or numbers written on the patient's skin. These abilities are impaired in lesions of the right parietal region.

Motor System

Trophic state

Assess the 3 S s: size, shape, and symmetry of a muscle. Atrophy, hypertrophy, or abnormal bulging or depression in a muscle is an important diagnostic finding in the presence of different muscle diseases or abnormalities. Hypertrophy occurs with commensurate strength from use and exercise; on the other hand, hypertrophy with weakness is seen commonly in Duchenne muscular dystrophy. The shape may also be altered when the muscle or tendon is ruptured.

Muscle tone

Muscle tone is the permanent state of partial contraction of a muscle and is assessed by passive movement. The muscle may be hypotonic or hypertonic. Hypotonia is defined as decreased tone and may be seen in lower motor neuron lesions, spinal shock, and some cerebellar lesions. Hypertonia may manifest as spasticity or rigidity.

Pyramidal lesions result in spasticity that may manifest as a clasp-knife phenomenon (ie, resistance to passive movement with sudden giving way, usually toward the completion of joint flexion or extension). Bilateral frontal lobe lesions may result in paratonia or *gegenhalten* (German for against-stop), in which resistance increases throughout flexion and extension. Rigidity refers to increased tone associated with extrapyramidal lesions; it may result in a cogwheel (stepwise) or lead-pipe (uniform) resistance to passive movement.

Muscle strength

Use this muscle-strength scale when assessing and documenting muscle strength (Table 2).

Table 2. Muscle-Strength Scale

Score	Description
0	Absent voluntary contraction
1	Feeble contractions that are unable to move a joint
2	Movement with gravity eliminated
3	Movement against gravity

- | | |
|---|-------------------------------------|
| 4 | Movement against partial resistance |
| 5 | Full strength |

Involuntary movements

Involuntary movements include fibrillations, fasciculations, asterixis, tics, myoclonus, dystonias, chorea, athetosis, hemiballismus, and seizures.

Fibrillations are not visible to the naked eye except possibly those in the tongue.

Fasciculations may be seen under the skin as quivering of the muscle. Although fasciculations are typically benign (particularly when they occur in the calf), if widespread, they can be associated with neuromuscular disease, including amyotrophic lateral sclerosis (ALS).

Asterixis can be elicited by having the patient extend both arms with the wrists dorsiflexed and palms facing forward and eyes closed. Brief jerky downward movements of the wrist are considered a positive sign. Asterixis is commonly seen with metabolic encephalopathies.

Tics are involuntary contractions of single muscles or groups of muscles that result in stereotyped movements. Gilles de la Tourette syndrome can manifest with multiple tics and elaborate, complex movements and vocalizations.

Myoclonus, as the word implies, is a muscle jerk; it is a brief (<0.25 seconds), generalized body-jerk, which is sometimes asymmetric. These occur alone or in association with various primarily generalized epilepsies.

Dystonias are muscle contractions that are more prolonged than myoclonus and result in spasms. Examples include blepharospasm, spasmodic torticollis, oromandibular dystonia, spasmodic dysphonia, and writer's cramp.

In athetosis, the spasms have a slow writhing character and occur along the long axis of the limbs or the body itself; the patient may assume different and often peculiar postures.

The term chorea means dance. Quasi-purposeful movements affect multiple joints with a distal preponderance.

Hemiballismus is a violent flinging movement of half of the body. It is associated with lesions of the subthalamic nucleus (ie, body of Louis).

Seizures may result in orofacial or appendicular automatisms, repeated eye blinks, or tonic or clonic motor activity.

Examination of Reflexes, Cerebellum, and Meninges

Reflexes

The different reflex responses may be grouped into 3 categories on the basis of their clinical significance.

Primitive reflexes

These include the glabellar tap, rooting, snout, sucking, and palmomental reflexes. As a rule, these signs are generally absent in adults. When present in the adult, these signs signify diffuse cerebral damage, particularly of the frontal lobes (hence the term frontal-lobe release signs).

Superficial reflexes

These are segmental reflex responses that indicate the integrity of cutaneous innervation and the corresponding motor outflow. These include the corneal, conjunctival, abdominal, cremasteric, anal wink, and plantar (Babinski) reflexes.

The corneal and conjunctival reflexes may be elicited by gently touching the appropriate structure with a sterile wisp of cotton. The normal response is bilateral winking. Absence of such a response implies CN V paralysis. Blinking of only 1 eye suggests weakness

of CN VII on the side that does not wink.

The abdominal reflex can be elicited by drawing a line away from the umbilicus along the diagonals of the 4 abdominal quadrants. A normal reflex draws the umbilicus toward the direction of the line that is drawn.

The cremasteric reflex is elicited by drawing a line along the medial thigh and watching the movement of the scrotum in the male. A normal reflex results in elevation of the ipsilateral testis.

The anal wink reflex is elicited by gently stroking the perianal skin with a safety pin. It results in puckering of the rectal orifice owing to contraction of the corrugator-cutis-ani muscle.

The best known of this group of reflexes is the plantar reflex. This reflex may be elicited in several ways, each with a different eponym. The most commonly performed maneuver is stroking the lateral aspect of the sole with a sharp object. The normal response is plantar flexion of the great toe, which is considered an absent (negative) Babinski sign. Dorsiflexion of the great toe (Babinski sign present) suggests an upper motor neuron lesion and also is referred to as a positive Babinski sign. Dorsiflexion of the big toe also may be associated with fanning out of the other toes, as detailed in Babinski's original description, but most neurologists consider this an unnecessary accompaniment of an abnormal response.

Flexion of the knee and hip may occur in the paretic leg with urinary and fecal incontinence. This is referred to as the en-mass reflex. Lack of either response may indicate absence of cutaneous innervation in the S1 segment or loss of motor innervation in the L5 segment ipsilaterally.

Deep tendon reflexes

These are monosynaptic spinal segmental reflexes. When they are intact, integrity of the following is confirmed: cutaneous innervation, motor supply, and cortical input to the corresponding spinal segment.

These reflexes include the biceps, brachioradialis, triceps, patellar, and ankle jerks. The musculocutaneous nerve supplies the biceps muscle. The radial nerve supplies the brachioradialis and triceps. The femoral nerve supplies the quadriceps femoris, which enables the knee jerk, and the tibial nerve supplies the gastrocnemius and the soleus.

Spinal roots that subserve these reflexes are listed below.

Table 3. Muscles and Spinal Roots

Muscle	Spinal Roots
Biceps	C5, 6
Brachioradialis	C6
Triceps	C7
Patellar	L2-4
Achilles	S1

On occasion, these root numbers are offset by +/- 1 when the cervical and/or lumbosacral plexuses are prefixed or postfixed as the case may be.

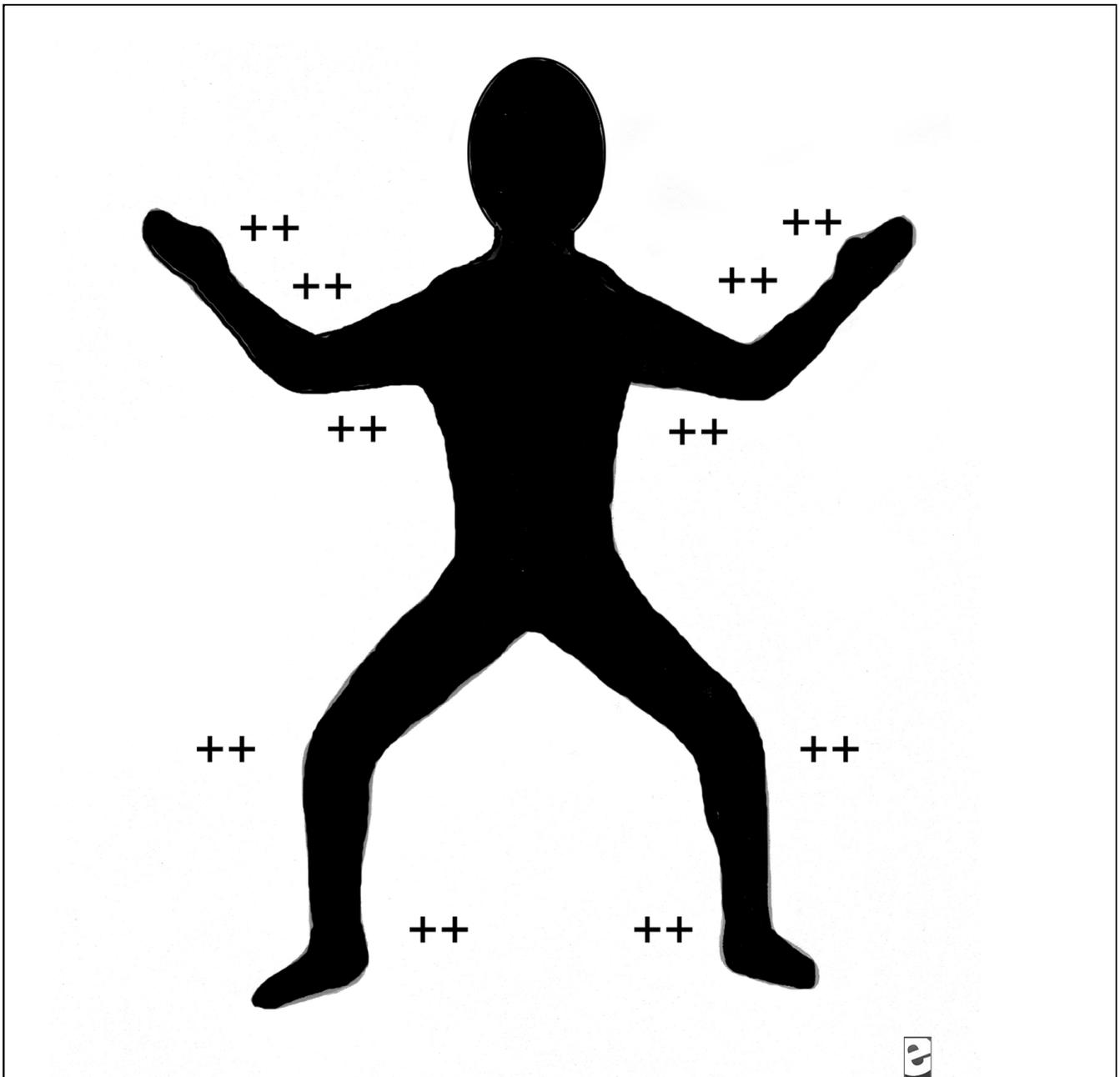
Several systems for reflex grading exist. An example is provided below.

Table 4. Reflex-Grading System

Score	Reflexes
0	Absent

- 1 Hypoactive or present only with reinforcement
- 2 Readily elicited with a normal response
- 3 Brisk with or without evidence of spread to the neighboring roots
- 4 Associated with a few beats of unsustained clonus
- 5 Sustained clonus

All textbooks now use a 0-4 scale to grade deep tendon reflexes, without a number assigned for sustained clonus. The addition of the number 5 allows for easy representation by using a stick figure. For a quick method of recording the reflex pattern see the image below.



Technique for documenting deep tendon reflexes. An arrow may be used to indicate the direction of toe movement with regard to the Babinski sign.

Cerebellar Signs

The cerebellum provides an important feedback loop for coordination of muscle activity by integrating the functions of the cortex, basal ganglia, vestibular apparatus, and spinal cord. Midline cerebellar dysfunction results in ataxia of gait, difficulty in maintenance of upright posture, and truncal ataxia. Acute neocerebellar hemispheric lesions result in additional signs.

The following are various cerebellar signs:

- Ataxia, atonia, and asthenia (the classic triad)
- Intention tremor (tremor that increases on activity)
- Dyssynergia (incoordination)
- Dysmetria (overshooting or undershooting)
- Dysrhythmia (inability to repeat a rhythmic tap)
- Dysdiadochokinesis (difficulty with rapid alternating movements)
- Dysarthria (staccato or scanning speech)

Gait is tested by having the patient walk normally and in tandem. In the latter, the patient is asked to walk with 1 foot immediately in front of the other (ie, heel to toe). A tendency to sway or fall to 1 side indicates ataxia, suggesting ipsilateral cerebellar dysfunction. Atonia and asthenia can occur in other lesions of the nervous system and are not specific to the cerebellum; their testing is described elsewhere.

Intention tremor refers to an oscillating tremor that accelerates in pace on approaching the target. Dyssynergia or incoordination results in loss of smoothness of execution of a motor activity. Dysmetria results in overshooting or undershooting of a target while attempting to reach an object. All 3 of these can be elicited by having the patient attempt to touch alternately his or her nose and the examiner's finger.

Dysrhythmia refers to the inability to tap and keep a rhythm. It can be tested by tapping the table with a hand (or the floor with a foot) and asking the patient to repeat the maneuver. Dysdiadochokinesis is the inability to perform rapid alternating movements; it can be tested by asking the patient to tap 1 hand on the other (or on the thigh) repeatedly while simultaneously pronating and supinating the hand. Various combinations of the above signs appear, depending on the extent and location of the lesion in the cerebellum.

Dysarthria is usually a sign of diffuse involvement of the cerebellum. It is characterized by poor modulation of the volume and pitch of the speech, causing oscillations of these 2 qualities.

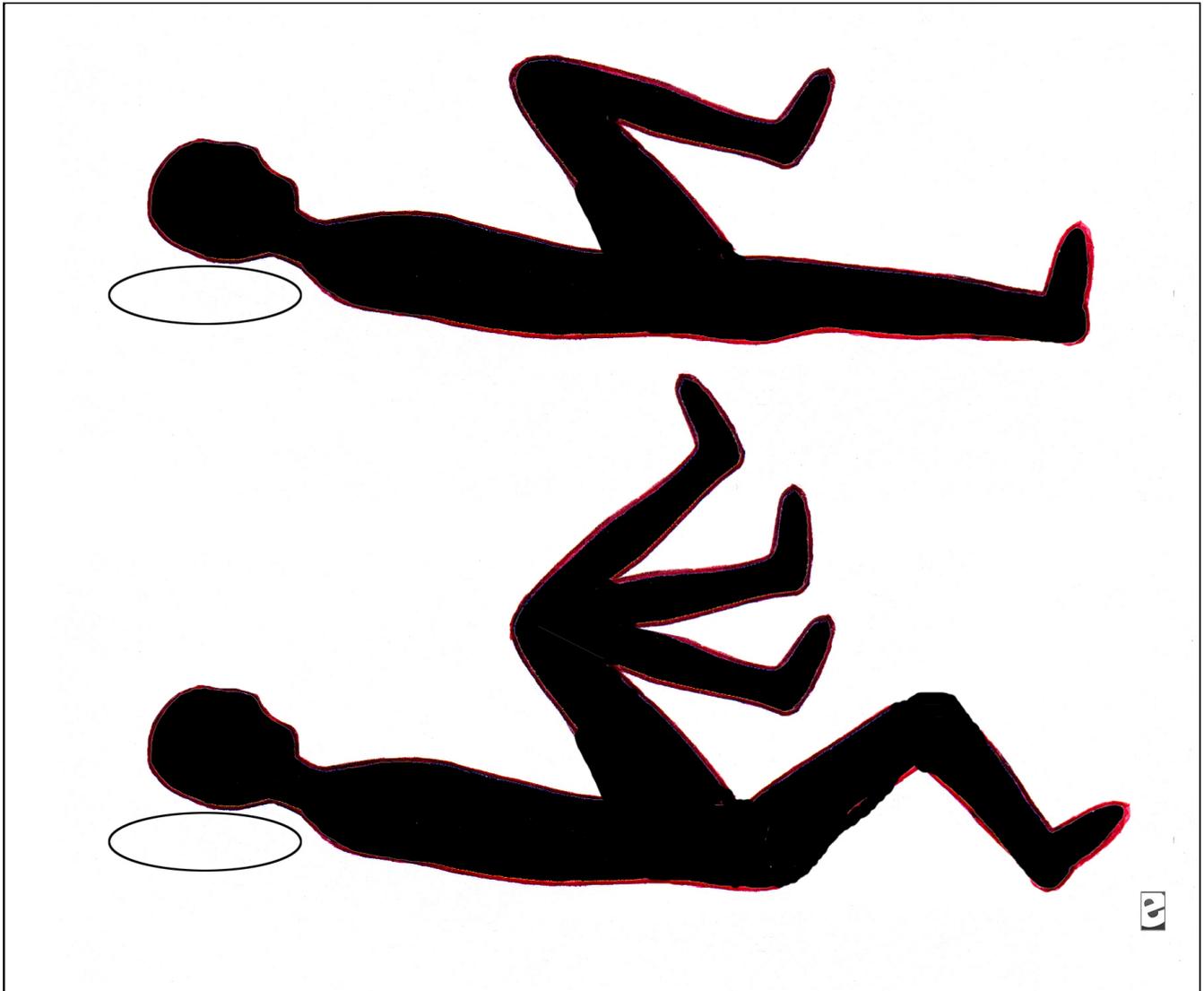
Meningeal Signs

Signs of meningeal irritation indicate inflammation of the dura; these signs are described below.

Nuchal rigidity or neck stiffness is tested by placing the examiner's hand under the patient's head and gently trying to flex the neck. Undue resistance implies diffuse irritation of the cervical nerve roots from meningeal inflammation.

The Brudzinski sign is flexion of both knees during the maneuver to test nuchal rigidity. This indicates diffuse meningeal irritation in the spinal nerve roots.

The Kernig sign is elicited by flexing the hip and knee on 1 side while the patient is supine, then extending the knee with the hip still flexed. Hamstring spasm results in pain in the posterior thigh muscle and difficulty with knee extension. With severe meningeal inflammation, the opposite knee may flex during the test.



Maneuver to demonstrate nuchal rigidity, the Kernig sign, and the Brudzinski sign.

The Lasègue or straight-leg raising (SLR) sign is elicited by passively flexing the hip with the knee straight while the patient is in the supine position. Limitation of flexion due to hamstring spasm and/or pain indicates local irritation of the lower lumbar nerve roots. Reverse SLR is elicited by passively hyperextending the hip with the knee straight while the patient is in the prone position. Limitation of extension due to spasm and/or pain in the anterior thigh muscles indicates local irritation of the upper lumbar-nerve roots.

System Survey and Ancillary Signs

System Survey

Autonomic nervous system

Autonomic dysfunction results in abnormalities in the following: sweating, skin temperature, cyanosis or pallor, trophic changes of skin or nails, and postural changes in blood pressure. Observation (and any necessary additional testing) easily demonstrates the presence or absence of these signs. Understanding these signs helps the examiner assess the patient's neurologic condition.

Neurovascular system

The following may be tested by palpation of the pulses and use of appropriate instruments:

- Brachial plexus and bilateral blood pressures
- Cranial and peripheral pulses
- Arterial bruits

Neurocutaneous system

Several neurologic conditions have telltale cutaneous stigmata. Evaluation for the following can provide valuable diagnostic clues: loss of skin pigmentation as in vitiligo, white hair-lock in Vogt-Harada-Koyanagi disease, cutaneous tumors or ash-leaf spots in tuberous sclerosis, and cutaneous eruptions over a dermatome which may signify herpes zoster.



Ash-leaf spots. Light, oblong patches of depigmented areas are seen on the skin of patients with tuberose (tuberous) sclerosis.



Herpes zoster (ie, shingles) results in painful skin eruptions in the distribution of specific dorsal root ganglia.

Coffee-brown pigmented (ie, café au lait) spots of varying sizes, usually greater than 1.5 cm in diameter, and axillary freckling are seen in neurofibromatosis. These are observed in addition to or in the absence of the characteristic blubbery subcutaneous tumors that give the condition its name.



Axillary freckling as seen in neurofibromatosis.

Tufts of hair (satyr's tail), dimples, and large moles along the spine may indicate spina bifida occulta or diastematomyelia of the spinal column.

Skeletal system - Cranium, spine, bones, joints

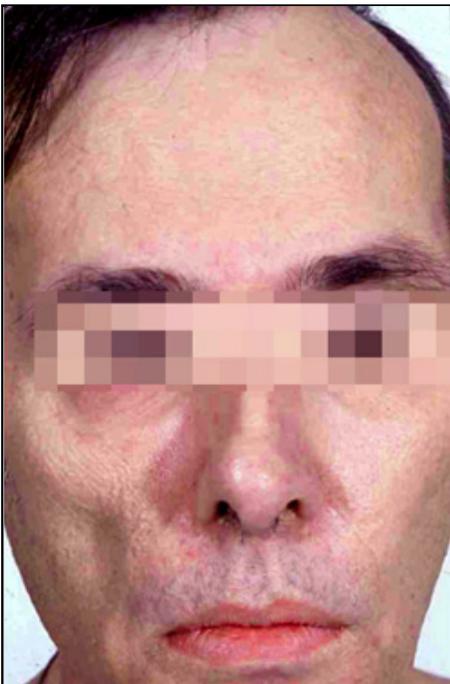
Palpation of the skull can reveal congenital anomalies that may indicate underlying abnormalities of the brain. In cephaloplegia, one half of the skull may be smaller than the other, possibly signifying asymmetric brain development. Microcephaly or macrocephaly may be detected by measuring the circumference of the head. Observation of the spine may reveal the presence of myelomeningocele, scoliosis, and/or kyphosis. In cases of prenatal brain injuries, the length of the long bones may be reduced on the side opposite the cephaloplegia.

Trophic changes in the joints can be associated with denervation in tabes dorsalis or Charcot-Marie-Tooth (CMT) disease. The distal muscular atrophy seen in CMT disease gives the legs the appearance of inverted champagne bottles.



Example of Charcot-Marie-Tooth disease (ie, peroneal muscular atrophy). This disease is associated with progressive weakness and wasting of the intrinsic muscles of the feet and calves.

Muscular atrophy seen in the region of the temporalis muscles and facial musculature associated with frontal balding is typical of myotonic dystrophy.



Typical appearance in myotonic dystrophy (ie, Steinert disease) includes frontal baldness, temporal atrophy, and narrow facies.

Pes cavus deformity can be associated with spina bifida and other spinal dysraphisms. A young person with mental retardation, genu valgum, pes cavus, and stroke may have homocystinuria, an inborn error of metabolism typically associated with mental retardation (usually severe) and intimal thickening and necrosis of the media of blood vessels, resulting in strokes and coronary artery disease.



Pes cavus deformity can be associated with many conditions including spina bifida, other spinal dysraphisms, and homocystinuria.

Ancillary Signs

Anisocoria

This refers to pupillary asymmetry, which may result from sympathetic or parasympathetic dysfunction. Sympathetic dysfunction results in Horner syndrome, in which the pupil is small but reacts to light. Hippus, a series of oscillating pupillary contractions seen in response to light, is a benign condition. Argyll-Robertson pupil, seen in neurosyphilis, is irregular and small; it does not react to light, but does accommodate.

In parasympathetic paralysis, the affected pupil is larger and reacts poorly or not at all to light. Injury to the ciliary ganglion or short ciliary nerves results in a tonic pupil, which is large and has slow or absent reaction to light. A benign form of tonic pupil is seen in Adie syndrome, Holmes-Adie syndrome (ie, tonic pupil with absent patellar and Achilles reflexes), and Ross syndrome (ie, tonic pupil with hyporeflexia and progressive segmental hypohidrosis).

Anosognosia

This refers to denial of illness and typically is seen in patients with right frontoparietal lesions, resulting in left hemiplegia that the patient denies. A form of visual anosognosia (Anton syndrome) is seen in patients with bilateral occipital lobe infarctions; these patients with double hemianopsia (bilateral cortical blindness) deny that they are blind.

Asterixis

This is seen in patients with metabolic encephalopathies. Momentary loss of tone and flapping of the hand are seen when the patient extends his arms in front with the wrists dorsiflexed.

Ataxia

Heel-to-toe tandem gait is tested by asking the patient to walk with 1 foot directly in front of the other. Ataxia can be demonstrated in this manner.

Beevor sign

This is seen with bilateral lower abdominal paralysis that results in upward deviation of the umbilicus when the patient tries to raise his head and sit up from the supine, recumbent position.

Benediction hand

This is seen with lesions of the median nerve in the axilla and upper arm. When present, the index finger remains straight and the middle finger partially flexes when the patient tries to make a fist (assuming the position of the hand of a clergyman while saying the benediction).

Bielschowsky sign

This refers to increasing separation of the images seen when a patient's head is tilted toward the side of a superior oblique (trochlear nerve) paralysis. This sign by itself is not diagnostic and should be used only as a supplement to other tests in suspected CN IV paralysis.

Chvostek sign

This is seen in hypocalcemia. Tapping the cheek at the angle of the jaw precipitates tetanic facial contractions.

Cogan sign

This is seen in myasthenia gravis. It refers to transient barring of the sclerae above the cornea as the patient resumes the primary eye position after looking down.

Dalrymple sign

This refers to the upper-lid retraction seen in thyroid ophthalmopathy.

Doll's-eye maneuver

This refers to turning the head passively with the patient awake and fixated or when the patient is in a coma. In the former, the eyes remain fixated at the original focus when all gaze pathways are normal; in the latter, the eyes deviate in the opposite direction when the brainstem is intact.

Gower sign

This sign, seen in severe myopathies, occurs when the patient attempts to stand up from the floor. Patients first sit up, then assume a quadrupedal position, and then climb up their own legs by using their arms to push themselves up.

Heterochromia iridis

This term refers to the difference in color of the 2 irides. It indicates early injury to the sympathetic system. Ipsilateral to the injury the iris is blue or green, while the contralateral iris is darker.

Jaw jerk

This is elicited by placing the examiner's index finger on the patient's lower jaw and then striking it with the reflex hammer. An exaggerated reflex indicates the presence of a pontine lesion. When the rest of the examination findings are normal, it may indicate physiologic hyperreflexia.

Kayser-Fleischer ring

This is a brownish ring around the limbus of the cornea. It is best demonstrated during an ophthalmologic slitlamp examination.

Lhermitte sign

This refers to the sensation of electricity associated with cervical spinal cord lesions during passive or active flexion and extension of the neck. Once considered pathognomonic of multiple sclerosis, it simply is the result of electricity generation by the hypersensitive, demyelinated, or injured spinal cord; this sign can be associated with any lesion in or around the cord.

Marcus-Gunn pupil

This sign requires a swinging-flashlight test to assess. As the flashlight swings from 1 eye to the other, the abnormal pupil dilates as the light swings back from the normal side. No anisocoria is seen. The phenomenon is also called a paradoxical pupillary reflex and indicates an afferent (optic nerve) pupillary defect.

Milkmaid's grip

This refers to the inability to maintain a sustained grip commonly seen in patients with chorea.

Moebius sign

This refers to weakness of ocular convergence (associated with proptosis) seen in dysthyroid ophthalmopathy.

Myerson sign

Patients with Parkinson disease, particularly those with bilateral frontal lobe dysfunction, continue to blink with repeated glabellar taps.

Nylen-Bárány sign

This is elicited by having the patient quickly lie down from the sitting position with the head turned to 1 side and hanging down 30° below the horizontal over the edge of the examining table. The procedure is then repeated with the head turned to the other side.

The test is positive when the patient experiences vertiginous discomfort and exhibits nystagmus after a latency period of about 10 seconds. The nystagmus increases for about 10 seconds then fatigues in peripheral vestibular disease. In central lesions, nystagmus may occur with the head turned to either side, without discomfort to the patient, and without latency of onset or fatigue.

Ondine curse

This refers to the failure of autonomic control of breathing when the patient falls asleep.

Oommen sign

Have the patient close the eyes and place a pebble the size of an M&M candy on the palm of the examiner's left hand. Cross the patient's middle finger over the index finger on its dorsal aspect. With the examiner's right hand, hold the patient's crossed fingers and have the patient's 2 (crossed) fingertips touch the pebble at the same time. Ask the patient how many pebbles are in the examiner's hand. With normal stereognosis, the patient should answer that there are 2 pebbles. In cases of astereognosis, the patient reports feeling only 1 pebble.

Opsoclonus

This refers to large-amplitude saccadic oscillations of the eyes in all directions, often exacerbated by refixation. They persist during sleep and are associated with brainstem and cerebellar lesions as well as a remote effect of certain carcinomas.

Optokinetic nystagmus

This is elicited by using a rotating, striped drum or a moving, striped piece of cloth. As the patient's eyes fixate on a stripe, nystagmus seen in healthy individuals is due to the optokinetic reflex. Lesions in the anterior aspects of the visual pathways decrease the response, and lesions of the vestibular system result in a directional preponderance to the elicited nystagmus.

Phalen sign

This refers to the aggravation of paresthesia and pain when the wrist is held in flexion (in patients with carpal tunnel syndrome).

Roger sign

This is numbness of the chin in patients with lymphoreticular (and other types of) malignancies.

Stellwag sign

This refers to decreased blinking frequency seen in thyroid ophthalmopathy.

Summerskill sign

This refers to the bilateral upper- and lower-lid retraction associated with severe liver disease.

Tinel sign

This refers to the tingling sensation elicited by tapping along the path of a regenerating nerve following injury. It helps to delineate the extent of nerve regeneration. The Tinel sign also can be observed in tardy ulnar palsy (palpation at the elbow) and carpal tunnel syndrome (tapping at the wrist).

Trendelenburg sign

This refers to the pelvic tilt toward the side of the unaffected raised leg when walking in patients with lesions of the superior gluteal nerve.

Trombone tongue

This is seen in patients with chorea. It refers to the unsteadiness of the tongue when the patient tries to protrude it outside the mouth.

Tullio phenomenon

This refers to the induction of vertigo and nystagmus with acoustic stimuli in patients with labyrinthine disease.

von Graefe sign

This refers to the lid lag on down gaze in patients with thyroid ophthalmopathy.

Definition of Terms

Apoplexy - Stroke (see definition of Stroke)

Cataplexy - Sudden fall, usually due to loss of muscle tone; may be precipitated by sudden changes in affect or mood in narcolepsy (see definition of Narcolepsy)

Cerebritis - Inflammation of the cerebral hemispheres

Encephalitis - Inflammation of the brain and brainstem structures

Encephalopathy - Dysfunction of the brain

Epilepsy - Recurrent seizures (see definition of Seizure)

Mononeuropathy - Dysfunction of individual nerves

Mononeuritis multiplex - Dysfunction of multiple single nerves

Myelitis - Inflammation of the spinal cord

Myelopathy - Dysfunction of the spinal cord

Myopathy - Primary muscle disease

Myositis - Inflammation of the muscles

Narcolepsy - Sudden attacks manifesting as an uncontrollable urge to sleep

Neuronopathy - Dysfunction of the cortical, cranial, or spinal neurons

Neuropathy - Dysfunction of the cranial or spinal nerves

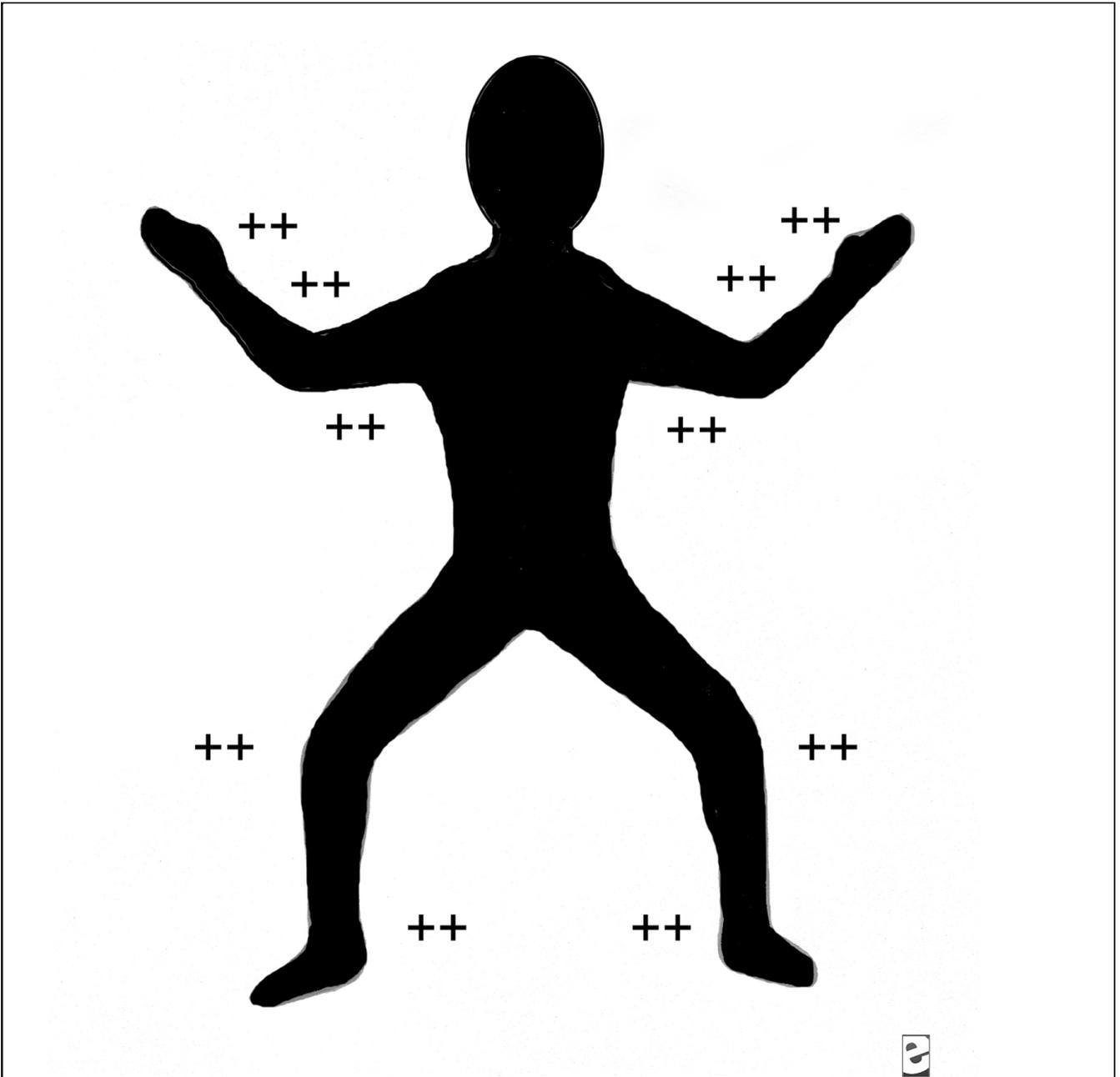
Polyneuropathy - Bilateral symmetric ascending (stocking and glove) or descending dysfunction of the peripheral nerves

Radiculopathy - Dysfunction of the nerve roots

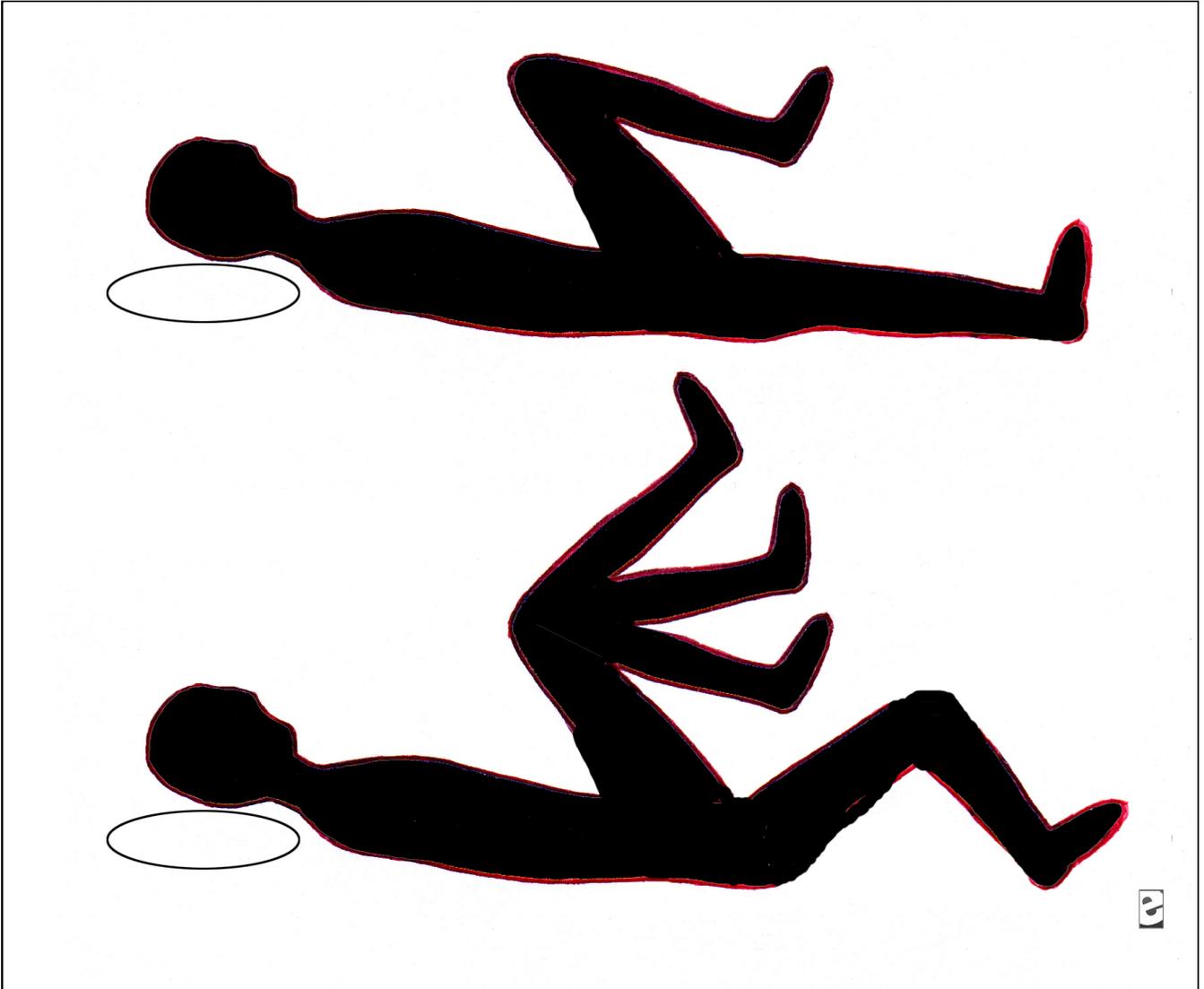
Seizure - Subjective or objective behavioral manifestation of an abnormal and excessive electrical discharge in the CNS

Stroke - Sudden onset of a neurological deficit, also known as a cerebrovascular accident

Multimedia



Media file 1: Technique for documenting deep tendon reflexes. An arrow may be used to indicate the direction of toe movement with regard to the Babinski sign.



Media file 2: Maneuver to demonstrate nuchal rigidity, the Kernig sign, and the Brudzinski sign.



Media file 3: Ash-leaf spots. Light, oblong patches of depigmented areas are seen on the skin of patients with

tuberose (tuberous) sclerosis.



Media file 4: Herpes zoster (ie, shingles) results in painful skin eruptions in the distribution of specific dorsal root ganglia.



Media file 5: Axillary freckling as seen in neurofibromatosis.



Media file 6: Example of Charcot-Marie-Tooth disease (ie, peroneal muscular atrophy). This disease is associated with progressive weakness and wasting of the intrinsic muscles of the feet and calves.



Media file 7: Typical appearance in myotonic dystrophy (ie, Steinert disease) includes frontal baldness, temporal atrophy, and narrow facies.



Media file 8: Pes cavus deformity can be associated with many conditions including spina bifida, other spinal dysraphisms, and homocystinuria.

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