NEUROLOGIC EXAMINATION

Performance of the main elements of a general screening neurologic examination is imperative (Table 104-5), but the examination should be tailored to confirm or disprove the clinical hypotheses generated from the patient's history. Unexpected signs must be explained, often with a return to the history for further clarification.

The examination is approached as if only one of two possible injuries has occurred—the final common pathway to a structure is disrupted, or the input to that pathway is disrupted (Fig. 104-1). In the case of the motor system, the *final common pathway* is the motor unit and includes the anterior horn cells giving rise to axons in a nerve, the nerve itself, the neuromuscular junction, and the muscle. Injury to any of these structures results in dysfunction of the muscle. Conversely, if these structures are intact, observing the muscle function may be possible under the right circumstances. If all modes of engaging the final common pathway fail to elicit a response, the clinician can conclude that the lesion is located somewhere within the final common pathway.

For example, a man with paralysis of facial movement on one side that is caused by a lesion of cranial nerve VII cannot smile voluntarily, close his eye, or wrinkle his forehead on the affected side. Spontaneous laughter or smiling as an automatic response to a joke also fails to move the paretic side. If the problem is central, however, facial movement with involuntary (spontaneous) smiling may be preserved or increased. This observation is common in patients with facial weakness caused by a stroke.

Central input to a final common pathway in the nervous system is usually tonically inhibitory. Damage to this input typically results in overactivity of the involved muscle group. Signs of damage to central inhibitory systems include spasticity and hyperreflexia (i.e., motor cortex, subcortical white matter,

TABLE 104-5 ELEMENTS OF A GENERAL SCREENING NEUROLOGIC EXAMINATION

SYSTEMIC PHYSICAL EXAMINATION
Head (trauma, dysmorphism, and bruits)
Neck (tone, bruits, and thyromegaly)
Cardiovascular (heart rate, rhythm, and murmurs; peripheral pulses and jugular venous distention)
Pulmonary (breathing pattern, cough and cyanosis)
Abdomen (hepatosplenomegaly)
Back and extremities (skeletal abnormalities, peripheral edema, and straight-leg raising)
Skin (neurocutaneous stigmata and hepatic stigmata)
MENTAL STATUS
Level of consciousness (awake, drowsy, and comatose)
Attention (coherent stream of thought, serial 7s)
Orientation (temporal and spatial)
Memory (short and long term)
Language (naming, repetition, comprehension, fluency, reading, and writing)
Visuospatial skills (clock drawing and figure copying)
Judgment, insight, thought content (psychotic)
Mood (depressed, manic, and anxious)
CRANIAL NERVES
Olfactory (smell in each nostril)
Optic (afferent pupillary function, funduscopic examination, visual acuity, visual fields, and structural eye findings)
Oculomotor, trochlear, and abducens (smooth pursuit and saccadic eye movements, nystagmus, efferent pupillary function, and eyelid opening)
Trigeminal (jaw jerk, facial sensation, afferent corneal reflex, and muscles of mastication)
Facial (efferent corneal reflex, facial expression, eyelid closure, nasolabial folds, and power and bulk)
Vestibulocochlear (nystagmus, speech discrimination, Weber test, and Rinne test)
Glossopharyngeal and vagus (afferent and efferent gag reflex and uvula position)
Spinal accessory (power and bulk of sternocleidomastoid and trapezii muscles)
Hypoglossal (position, bulk, and fasciculations of tongue)
MOTOR EXAMINATION
Pronator drift (subtle corticospinal lesion)
Tone and bulk of muscles (basal ganglia lesion yields rigidity, cerebellar lesion yields hypotonia, corticospinal lesion yields spasticity, nonspecific bihemispheric disease yields paratonia, hypertrophy indicates dystonia, pseudohypertrophy indicates muscle disease, and atrophy indicates lower motor neuron disease)
Adventitious movements (tremor, tic, dystonia, and chorea indicate disease of the basal ganglia; asterixis and myoclonus may indicate toxic metabolic process)
Power of major muscle groups (scale 0-5)
Upper extremities: deltoids, biceps, triceps, wrist extension and flexion, finger extension and flexion, and interossei
Lower extremities: hip flexion, extension, abduction, and adduction; knee extension and flexion; ankle dorsiflexion, plantar flexion, inversion, and eversion; toe extension and flexion
SENSORY EXAMINATION
Light touch (posterior columns)
Pinprick (spinothalamic tract)
Temperature (spinothalamic tract)
Joint position sense (posterior columns)
Vibration (posterior columns)
Graphesthesia (cortical sensory)
Double simultaneous stimulation (cortical sensory)
Two-point discrimination (posterior columns and cortical sensory)
REFLEX EXAMINATION
Standard reflexes (grades 0-4)
Biceps
Triceps
Brachioradialis
Knee jerk
Ankle jerk
Pathologic reflexes
Babinski's sign (if present)
Myerson's sign (if present)
Snout (if present)
Jaw jerk (if brisk)
Palmomental (if present)
Hoffmann sign (if brisk)
COORDINATION AND GAIT
Finger-nose-finger (action tremor suggesting cerebellar disease)
Rapid alternating movements (dysdiadochokinesia suggesting cerebellar disease)
Fine motor movements (slowness and small amplitude suggesting basal ganglia or corticospinal tract abnormalities)
Heel-to-shin (ataxia suggesting cerebellar disease)
Arising from chair with arms folded across chest (inability in advanced basal ganglia, cerebellar, corticospinal, or muscle disease)
Walking naturally (look for decreased arm swing, spasticity, broad base, festination, waddle, footdrop, start hesitation, and dystonia)
Tandem gait (look for ataxia)
Walking with feet everted or inverted (look for latent dystonia)
Hopping on each foot separately (look for latent dystonia)
Stand with feet together and eyes open, eyes closed (sensory ataxia and cerebellar disease)
Response to retropulsive stress (loss of postural righting mechanisms)