CHAPTER 2

Neurodiagnostic Studies

After assessment of patient’s symptomatology and neurologic examination findings, neuro-imaging studies are performed to confirm the clinical impression. The studies delineate presence and location of pathological lesion causing neurologic dysfunction and sometimes may delineate pathologic lesion characteristics. Neuro-imaging studies may help to delineate pathological conditions and to visualize normal brain structures. Physicians should understand the appropriate utilization of these neuroimaging studies and order these studies only when they are of clinical value and not utilize these studies when there are no clinical indications to support their utilization. Avoid blind fishing expeditions when the clinical symptoms are vague and exam findings are normal, or the physician may detect incidental and asymptomatic comorbid pathology which does not explain the patients symptomatology and neuro-imaging studies are not cost-effective or beneficial to the patient. When you go “fishing,” you may “catch” an unexpected and asymptomatic lesion. To use these neuro-imaging studies most effectively, physician must be aware of methodology, parameters being measured, sensitivity, specificity, indications, contraindications, and risks of the procedures (Table 2-1).

SKULL AND SPINE ROENTGENOGRAPHY

Skull and spine roentgenograms (shadow radiography) provide visual representation of intracranial and vertebral distribution of crystallized calcium, displayed on photographic plates. These studies have good spatial resolution but limited contrast resolution. They are ideal for visualizing bone detail. Normally calcified structures (e.g., pineal body, sella turcica vertebral bodies) are frequently demonstrated, as are pathological calcification (e.g., meningioma, craniopharyngioma, oligodendroglioma, and angioma). Because of limited contrast resolution, normal intracranial and spinal structures (e.g., brain parenchyma, spinal cord, cerebrospinal fluid [CSF] spaces) are not visualized with shadow radiography.

Indications for skull roentgenograms include suspicion of skull fracture in head trauma, suspicion of juxtasellar lesion (endocrine and visual disturbances), systemic neoplasm with skull involvement (osteolytic, osteoblastic). In addition, isotope bone scanning is more sensitive than roentgenography to detect certain types of neoplastic or inflammatory bone involvement (e.g., metastases, multiple myeloma, osteomyelitis). Roentgenograms of spine are used to visualize bony encasement of spinal cord in patients with spinal root (radiculopathy) or spinal cord (myelopathy) compression. These films are indicated to assess congenital, degenerative, neoplastic, traumatic, infectious conditions. The major limitation of spine roentgenography is insufficient resolution to visualize neural structures, noncalcified soft tissue structures (intervertebral disk, ligaments, spinal cord, spinal nerve roots), or subarachnoid spaces. To define spinal cord and nerve root abnormalities (e.g., herniated intervertebral disk, spinal cord tumors) myelography with CT or magnetic resonance imaging (MRI) are necessary.
ELECTROENCEPHALOGRAPHY (EEG)

EEG is a reflection of surface brain electrical activity and is valuable in evaluating patients with paroxysmal episodes (e.g., seizures, unexplained loss of consciousness) especially when there is no evidence of structural brain dysfunction visualized with CT or MRI. Multiple electrodes are attached to the patient’s scalp. These electrodes sample surface cerebral electrical activity (frequency and voltage), amplify and record summated potentials originating from the cerebral cortex. There is normal pattern for awake and sleep states. Brain lesions alter the frequency, amplitude, and pattern of brain waves; however, location of maximal electrical abnormality may not correlate with precise location of lesion; for example, temporal neoplasm can cause maximal slow wave discharge in frontal or parietal region. Electrical activity is sampled in a standard pattern of connections (montages), representing both hemispheres. Electrical activity generated by the brain varies in frequency and amplitude in different regions. Normally there is symmetrical frequency of 8 to 10 cps. This is maximal in occipital region and is referred to as alpha rhythm. In frontal region, there is a symmetric, more rapid (14 to 18 cps) and lower amplitude beta rhythm. If patient is aroused by external stimulation (e.g., calling patient’s name), there is change from alpha rhythm to lower voltage rapid frequency; this is arousal response. Awake state shows specific EEG findings, and there are characteristic patterns in sleep state (see Chapter 23). Specific response can also be induced by hyperventilation and photic stimulation; these are important in patients with suspected seizure disorders.

Abnormal electrical potentials are recorded as slow waves or spike discharges; these can occur in either generalized or focal distribution. Superficial cortical lesions are more likely to be detected than deeply located lesions (thalamus or basal ganglia); infratentorial (brain stem or cerebellum) lesions do not usually cause EEG abnormalities. If EEG demonstrates focal abnormality, it may be possible to determine the precise cerebral hemispheric location of a lesion; however, maximal electrical dysfunction does not always correlate with lesion location.

There are two types of abnormal EEG patterns: spikes and slow waves. Spikes are typical of epileptogenic activity. Slow waves can occur in a diffuse distribution as in metabolic conditions (e.g., hepatic encephalopathy) or in focal disturbances as seen with intracranial mass lesions. The slowing can be mild in range of three to seven cps (theta rhythm) or severe with less than three cps (delta rhythm). In patients with metabolic disorders, EEG shows either diffuse slowing or paroxysms of high-voltage bifrontal delta activity. Because of limited EEG response patterns, it is not possible to make specific pathological diagnoses from EEG findings alone. For example, focal slow wave patterns caused by infract, hemorrhage, neoplasm, or abscess can appear identical.

In patients with suspected seizure disorder, EEG is performed to help establish this diagnosis and allow precise seizure classification. For example, certain clinical seizures appear generalized, but EEG may show focal abnormality. All patients with epilepsy have abnormal EEGs during seizures. Because 10% to 15% of normal persons have nonspecific EEG abnormalities, clinical diagnosis of a seizure disorder cannot be made from the EEG findings alone (See Chapter 11). In patients who have unexplained episodes or “spells” and abnormal nonspecific EEG patterns, combined simultaneous video-EEG monitoring can differentiate true epilepsy from other paroxysmal conditions such as syncope, breath-holding spells, and pseudoseizures (seizures not accompanied by EEG abnormalities). Between clinical episodes presence of epileptiform discharges usually suggest the diagnosis of epilepsy; however, absence
of these abnormal spikes in the inter-ictal state does not exclude the diagnosis. Epileptiform discharges must be present during and immediately after the seizure to support epilepsy diagnosis.

**EVOKE POTENTIALS (EP)**

EEG records spontaneous superficial electrical cerebral activity; whereas EP reflects CNS activity in response to specific sensory stimulus. The basic EP signal is buried in background noise or interference. It is possible to amplify the wave by summatting and averaging the brain wave recorded after evoked stimulation. The wave can represent an EP to sensory stimulation such as a flashing light, auditory sound clicks, and stimulation of peripheral nerve. In usual EP, an electronic device – average response computer – can summate and average the occipital cortical response to a series of light flashes presented over short intervals. Other sensory-evoked potentials assess auditory system by recording over primary auditory cortex in temporal region or somatosensory system by recording over postcentral parietal region. Each evoked wave has its characteristic latency of response, amplitude, and morphology (form). Sensory EP differs from routine EEG in two ways: (1) the response is evoked, and (2) the response is computer averaged to accentuate the response. EP traces transmission of sensory nerve impulses through nerves, spinal cord, brain stem, thalamus and to sensory portions of cerebral cortex.

The clinical applications of this technique are rapidly expanding. For example, visual response assesses retinal and optic nerve function. In patients with visual loss, different abnormalities have been reported in patients with demyelinating disease, tumor, or ischemic lesions. In addition, by performing EP in 50% of the visual field (hemifield), abnormalities or asymmetries of the optic chiasm, optic radiation, or occipital cortex can be assessed. Auditory-evoked potentials EP are being used to evaluate patients with hearing impairment and vertigo to determine the level of involvement within the auditory system (cochlea, auditory nerve, brain stem, thalamus, auditory cortex). EP can be monitored during surgery to assess the spinal and cerebral pathways, for example. EP can delineate dysfunction in specific sensory systems and possibly locate the level of the neurological dysfunction in the neuro-axis.

**CRANIAL ULTRASOUND**

A surface probe with an electrically activated piezoelectric crystal transducer is used with amplifier to record returning echoes. These points of deflection (echoes) represent interfaces between structures of different acoustic densities such as brain ventricles and brain parenchyma. Ultrasonography is of limited value in adults because skull bones cause dispersion of sonar waves before they pass through the intracranial region; however, in squamous portion of temporal bone located directly above and posterior to the ear, bone is unilaminar without diploic spaces, and this is adequate for ultrasound propagation.

Ultrasonography is useful for infants because of infant bone structure. The open fontanelles allow better ultrasound propagation with good spatial and contrast resolution such that lateral and fourth ventricles can be visualized. Ultrasound depends more on skill of technician and provides less definition of intracranial anatomy than CT or MRI; however, ultrasound can be performed at the bedside in nursery and does not require transporting a sick infant to radiology department.
LUMBAR PUNCTURE (LP)

With insertion of special spinal needle into lumbar subarachnoid space, it is possible to measure intracranial pressure and analyze CSF content. The patient is placed in knee-chest position. Under aseptic conditions and with dermal anesthesia, 19- or 21-gauge needle is inserted below the L-2 interspace to perforate dura. After subarachnoid space is entered, needle stylet is removed, and needle is connected to pressure manometer. For accurate pressure recordings, patient should be breathing normally and patient’s legs should be straightened. If patient is agitated, performs Valsalva maneuver (by straining), or shows increase in intraabdominal pressure (by not having legs straightened), CSF pressure can by falsely elevated (greater than 250 mm H₂O). If needle is positioned correctly, fluctuations in pressure occur with respiration and pulse. Hyperventilation causes hypocapnia (low carbon dioxide content), resulting in intracranial vasoconstriction, and this can falsely lower CSF pressure. The pressure that is recorded is usually accurate reflection of intracranial pressure and can be falsely low because intracranial and lumbar spaces are no longer in free communication. CSF pressure recorded by lumbar puncture is less reliable than continuous intracranial pressure monitoring with subdural transducer; however, this requires neurosurgical intervention. Accurate continuous pressure measurements may be necessary in certain disorders (e.g., Reye’s syndrome, head trauma, idiopathic intracranial hypertension).

LP with CSF analysis is necessary to establish diagnosis of meningitis (see Ch ____), subarachnoid hemorrhage (SAH), neurosyphilis, and multiple sclerosis. CT is the initial procedure if SAH is suspected as LP may increase transmural pressure of aneurysm to increase bleeding; however, if CT is negative, LP must be performed, as it is most sensitive test for SAH. For diagnosis of intracerebral hemorrhage, CT is most sensitive study and LP is unnecessary and may be negative in 20% of cases. Contraindications for LP are relative and not absolute, but usually include suspicion of mass lesion (i.e., altered mentation, focal deficit, papilledema, coagulation disturbance, and local infection at site of spinal needle puncture). Complications of LP include backache, headache, leg motor or sensory disturbances, bleeding (epidural, subdural, subarachnoid), infection, transient abducens nerve paresis, intraspinal epidermoid tumor. Treatments for headaches include fluid hydration, prone positioning of the patient, caffeine, or, if headache persists, epidural blood patch. If intracranial lesion is suspected CT or MRI should be performed initially. If there is lumbosacral infection involving skin, subcutaneous tissue, or bone, cervical cisternal tap should be performed. Remember more patients die from the failure to perform LP than die from the performance of LP (e.g., herniation).

Quantity of CSF that is removed does not correlate with occurrence of post LP headache; therefore, enough CSF should be removed for necessary studies. In most cases, 5 ml of CSF is sufficient for cell count, protein content, sugar content, serology, bacterial culture, and Gram stain. It is important to describe the color of CSF (clear, cloudy, bloody, xanthochromic) and compare this with a tube of water as both water and CSF are viewed against white background. If CSF appears bloody, the following procedure should be followed: first observe for clot formation to occur because blood-CSF mixture does not clot; next determine if fluid changes to clear appearance after several drops form because this is consistent with traumatic spinal tap;
then place bloody CSF in hematocrit tube and centrifuge because in spontaneous subarachnoid hemorrhage, supernatant appears xanthochromic. Xanthochromic fluid can also occur if CSF protein content exceeds 1 g/dl. If tap has been traumatic, white blood cells will also be present in CSF. The correction factor is one white blood cell for every 500 to 1000 red blood cells. In addition, 1000 red cells increase protein content by 1 mg/dl.

ULTRASOUND CEREBROVASCULAR IMAGING STUDIES
Noninvasive ultrasound vascular imaging studies assess presence of stenotic, occlusive, or ulcerative lesions involving cerebral circulation, and are used most commonly to image carotid arteries.

DOPPLER DUPLEX ULTRASOUND
With carotid duplex ultrasound it is possible to delineate flow velocity analysis and image structure of carotid artery bifurcation. This site is common location for atherosclerotic plaque formation. With increasing severity of carotid stenosis, flow velocity progressively increases. With high-resolution ultrasound scanners, it is possible to demonstrate morphologic characteristics of arterial wall. If there is more than 50% carotid stenosis, carotid duplex studies will detect this stenosis 95% of the time. Doppler ultrasound cannot differentiate severe stenosis from complete occlusion. This is important limitation because the patient with occlusion should not undergo endarterectomy, whereas patients with high-grade stenosis should have endarterectomy.

TRANSCRANIAL DOPPLER STUDY (TCD)
Using Doppler principal (signals emanating from ultrasound probe reflect off red blood cells in the artery such that frequency of reflected signal is shifted in proportion to velocity of flowing blood), it is possible to perform extracranial (carotid) and transcranial vascular imaging studies. With a pulsed-wave Doppler probe with high tissue penetration characteristics applied at areas of thin skull bone (e.g., temporal bone superior to zygoma between the ears, directly over the orbits, suboccipital region of foramen magnum, intracranial vessels (carotid artery siphon, initial portions of anterior, middle, and posterior cerebral arteries, distal intracranial segments of vertebral arteries and basilar artery) can be insonated. An increased blood flow, as measured by transcranial Doppler technique, indicates arterial lumen narrowing (stenosis, vasospasm). This is useful study in detecting intracranial arterial stenosis in patients with ischemic stroke (e.g., has thrombolytic drug re-established blood flow) and for following patients with SAH for the development of vasospasm. The major limitation of TCD is that the study can be technically difficult or even impossible to perform because of an inadequate transcranial temporal ultrasound window.

MAGNETIC RESONANCE ANGIOGRAPHY – SEE MRI

CONVENTIONAL CATHETER ANGIOGRAPHY (CCA)
Prior to CT and MRI, CCA was utilized to delineate vascular anatomy and to demonstrate the location of intracranial lesions as the lesion distorted and displaced the normal position of the intracranial vessels. By knowing the position of displacement of and arteries and veins, the vector of vascular displacement localizes the location of the lesion. This is not needed as CT and...
MRI shows lesion location more precisely than angiography and shows the distortion of normal structures by the lesion. Angiography now is limited to showing cranial vascular disease which is not visualized by CT, MRI, carotid ultrasound or magnetic resonance angiography (MRA). In addition, CCA can be used for interventional (intra-arterial) techniques for the treatment of cerebrovascular disease (coiling of aneurysm, arterial thrombolytic therapy, angioplasty).

Because intravascular blood pool is slightly more radiodense than CSF and less dense than brain, water-soluble, iodinated material is necessary to create sufficient artificial contrast to visualize blood vessels but does not visualize arterioles. In femoral percutaneous angiography, flexible, plastic catheter is introduced into this vessel and directed upward through the aortic arch. By using fluoroscopy, it is possible to inject contrast media selectively into carotid or vertebral arteries. Iodinated contrast material is injected into each vessel, and series of films is rapidly taken to outline arterial, capillary, and venous circulation to visualize anatomical detail of extracranial and intracranial vessels. The vascular lumen is opacified and this film is photographed by conventional radiographic or digital subtraction techniques. CAA may visualize detail of large and small extra- and intracranial blood vessels, but does not visualize arterioles. Tumors are detected because they cause mass effect with distortion and displacement of certain intracranial vessels. On basis of direction of major vectors of vascular distortion, location (e.g., intraaxial versus extraaxial) of tumor is established; however, this vascular pattern does not define whether the nature of the lesion’s, neoplastic or non-neoplastic (e.g., abscess, intracerebral hematoma). The location of the mass is now better detected with CT or MRI, but tumor vascularity pattern is better demonstrated by CCA. Neoplasms can be associated with new vessel formation (neovascularity). Certain neoplasms (i.e., meningiomas, gliomas) have a characteristic stain pattern. Angiography accurately delineates morphologic detail of abnormal vessels (e.g., aneurysm, vascular malformation). It also shows degree of stenosis or occlusion of the extracranial vessels and defines the presence of an ulcerated plaque.

Complications of angiography can result from technique itself or toxicity of contrast material. Movement of catheter can dislodge emboli from intima of aorta or carotids; in addition, clot formation can occur on catheter and embolize to the brain. The organically bound iodide contrast agents can cause systemic (e.g., anaphylactic, cardiovascular) or neurotoxic (e.g., seizures, stroke) reactions. Because contrast material is excreted by and potentially toxic to kidney, renal dysfunction can preclude angiography.

Interventional catheter angiography can be used therapeutically. Using arterial catheterization equipment and fluoroscopic control, it is possible to reduce regional arterial blood flow (embolization). This is valuable in patients with arterial-venous malformations or in patients with meningioma that has marked neovascularity supplying the tumor. Embolization (with reduction in the blood flow) can be achieved by ablating abnormal blood vessels with solid materials such as carbon or ferromagnetic microspheres, detachable balloons, or gelatin sponge. In other patients who have blood vessel occlusion, it is possible to increase blood flow by injecting a thrombolytic agent such as tPA, urokinase or streptokinase or to insert and inflate a balloon catheter within stenotic or vasospastic arterial segments. Utilization of coils may replace surgical clipping of intracranial aneurysms.
MYELOGRAPHY AND SPINE MRI

In myelography, patient is positioned prone on x-ray table and 5 to 15 ml of oil-soluble (e.g., iophendylate [Pantopaque]) or water-soluble (e.g., metrizamide [Amipaque]) substance is injected into lumbar subarachnoid spaces. Dye is maneuvered into lumbar, thoracic, and cervical regions by tilting x-ray table. The dye fills subarachnoid space and outlines spinal cord and nerve roots. If herniated disk impinges on nerve root, myelography shows asymmetry or indentation of nerve root sheath. If spinal cord compression is present, myelographic findings define distortion and displacement of cord; the position (extradural, intradural, extramedullary, or intramedullary) or mass is defined by findings in lateral or anteroposterior projection. With MR, indications for myelogram have diminished; however, myelogram with post-myelogram CT is utilized if bony abnormality is suspected and MRI findings are equivocal or negative.

COMPUTED TOMOGRAPHY (CT)

CT is brain-imaging technique providing direct visual images of intracranial contents (CSF spaces, blood vessels, gray and white matter) based on radiographic attenuation values (representing tissue density measurements). CT provides better contrast resolution than other diagnostic procedures such that brain image findings are equivalent to findings at gross autopsy.

        X-ray source is thinly collimated beam that penetrates patient’s head at multiple angles. Transmitter x-ray attenuation readings are recorded by series of multiple crystal or gas detectors. These summed x-ray profiles are analyzed by high-speed computer program. The result reflects radiodensities within specific axial (transverse) tissue sections. These radiodensities are expressed in numerical values; in this scale, water is zero, with bone being +2000, and air being -2000. The value of normal brain constituents represent approximately 120 units (2.5%) of total scale. These radiodensities are visually displayed on cathode-ray oscilloscope using gray scale. Each brain tissue section is usually 8mm in thickness. Within each section there are multiple rectangular regions (pixels) for which radiodensity values are determined. It is possible to increase the spatial resolution by decreasing size of each pixel, therefore increasing number of attenuation values obtained.

        The characteristic appearance of intracranial content is displayed at multiple levels extending from the base to vertex. CT scans are usually displayed in axial plane, although coronal and sagittal sections can be obtained. Pathologic processes cause abnormal radiodensity patterns and can show position of normal intracranial structures. Following rapid intravenous infusion of iohinated contrast material, it is possible to visualize major intracranial vessels and to detect enhancement patterns caused by impaired blood-brain barrier in neoplastic or infectious-inflammatory disorders. On basis of plain and postcontrast appearances, it is sometimes possible to predict underlying pathologic process; however, CT findings are not always characteristic enough to make specific diagnoses without surgical biopsy.

MAGNETIC RESONANCE IMAGING (MRI)

MRI uses radiowaves and a externally applied magnetic field to align protons (usually hydrogen ions contained in water molecules) parallel to a magnetic field. When radiofrequency stimulus is applied, the protons gyrate. As stimulus is turned off, radio-receiver can detect signals as protons relax and reorient themselves within magnetic field. MRI has the potential to provide quantitative brain biochemistry analysis (magnetic resonance spectroscopy). This allows
assessments of markers of gray and white matter (N-acetyl aspartate, choline) and anaerobic metabolism (lactic acid, high-energy phosphates [ATP]). For example, conventional MRI may show normal appearing white matter in multiple sclerosis patients; however, MRS may show that the biochemical properties of this white matter may be abnormal. Using MRI contrast agents (gadolinium), the integrity of the blood-brain barrier can be assessed. Utilizing functional MRI it is possible to achieve noninvasive visualization of brain function (physiological imaging). With this technique it is possible to map functional areas of the cerebral cortex in response to activation techniques (e.g., motor, speech, visual, auditory), or in response to hemodynamic stress e.g., ischemic cerebrovascular disease). Functional MRI is achieved by taking advantage of the magnetic property differences between oxygenated and deoxygenated blood. With functional activity the involved cortex shows focal increase in cerebral blood flow with shift from deoxyhemoglobin to oxyhemoglobin in cerebral activated regions.

In patients with cerebral ischemia, diffusion weighted imaging (DWI) and perfusion weighted imaging (PWI) provide important information of early tissue change. In DWI, ischemic areas appear as bright areas (light-bulb effect) within one to three hours of vascular occlusion as intracellular water is more restricted than extracellular water (Brownian motion effect). The DWI effect lasts for 7 to 14 days and then disappears. A positive DWI study is most sensitive measure of acute ischemia and can differentiate acute from chronic ischemia. DWI may be combined with perfusion-weighted imaging (use of paramagnetic gadopentetate dimeglumine to demonstrate blood volume, blood flow and vascular transit time) to serve as indicator of reduced cerebral perfusion. The PWI/DWI ratio can show diminished perfusion areas and areas of abnormal parenchyma, which may define the “penumbra” which is ischemic, but not infarcted potentially salvageable tissue (see ch.____ ).

The advantages of MRI over CT include avoidance of ionizing and radiation, ease with which multiplanar scanning can be achieved (e.g., axial, coronal, sagittal), and improved contrast resolution such that discrimination of gray and white matter is reliably achieved. Disadvantages are that MRI requires a longer time for data acquisition than CT and that MRI images are susceptible to motion artifacts. MRI cannot be performed on patients who have had prior aneurysm surgery or shunts using cranial metallic clips or who have a cardiac pacemaker. Because of the ease with which multiplanar images can be achieved and because bone does not generate artifacts, MRI provides excellent detail of the posterior fossa and spinal canal.

**MAGNETIC RESONANCE ANGIOGRAPHY (MRA)**

This technique is noninvasive and requires no contrast injection. This avoids the risk of neurologic and cardiovascular complications of catheter angiography. Utilizing a specific pulse sequence, flowing blood appears as hyper-intense signal and brain parenchyma appear hypo-intense. These MRA images show vascular anatomy in multiple planes. Because of turbulent flow and vascular position, MRA may overestimate degree of vascular stenosis; therefore, CCA remains the “gold standard” for delineation of vascular anatomy and pathology. If venous sinus thrombosis is suspected, magnetic resonance venography is very useful procedure to delineate venous occlusion.

**POSITRON EMISSION TOMOGRAPHY (PET)**

PET uses positrons (positively charged electrons that are generated using a cyclotron), present in normal brain substances. These include carbon ($^{11}$C), oxygen ($^{15}$O), and nitrogen ($^{13}$N); fluorine
The PET technique is similar to that of CT, except that the data collected represents the
distribution of photons emitted from isotopes within the brain. With PET, brain physiology and
function can be studied in both normal and pathologic states. For example, it is possible to
determine brain blood flow and metabolism in aging patients and to compare this with the
findings in demented patients. It is possible to determine blood flow and metabolism in the
resting state and compare it with changes occurring in specific brain regions after visual or
auditory stimulation. Pathologic conditions in which blood flow and tissue metabolism have
been studied include ischemic cerebravascular disease, epilepsy, dementia, and malignant brain
neoplasms. There is also great potential for the use of PET in investigating patients with
“functional” psychiatric disorders (e.g., schizophrenia, affective disorders) and in patients with
dyskinesias and Parkinson disease to image neurotransmitter receptors to compare these findings
in untreated versus treated patients.

**SINGLE-PHOTON EMISSION COMPUTED TOMOGRAPHY (SPECT)**

For PET, a cyclotron is needed to generate positron-emitting radionuclides. These have a short
half-life, which means PET can only be performed in a medical center facility that has a
cyclotron. This limits PET; however, with SPECT the procedure is performed with
radionuclides, gamma-emitting isotopes. With SPECT, there is no need for cyclotron, and
radionuclides are commercially available; however, spatial resolution of SPECT is lower than
that of PET. SPECT uses routine nuclear medicine camera and is available in most general
hospitals. SPECT can demonstrate areas of cerebral ischemia as shown by reduced areas of
perfusion in patients with Alzheimer’s, Parkinson’s, and Huntington’s diseases can be valuable
in showing neurotransmitter (dopamine) abnormalities. SPECT images cerebral perfusion but
not brain metabolism.

**ELECTRICAL DIAGNOSTIC STUDIES**

**Nerve Conduction Velocity (NCV)**

Conduction velocity of peripheral (not central) motor nerves is determined by stimulating the
nerve at two points (proximally and distally) and recording the action potential over the muscle
with surface electrodes. By determining conduction time from distal and proximal stimulation
and measuring distance between these two points, conduction velocity is calculated. The
velocities are determined for superficial (median, ulnar, peroneal, and posterior tibial) nerves.
More deeply situated nerves are not usually analyzed because their direct stimulation is not
technically feasible. Recent technical advances have made possible measurement of sensory
conduction velocities.

NCV depends on the integrity of surrounding myelin sheath, and is determined by the
diameter of the largest axons. In demyelinating neuropathy, the NCV is markedly slowed. If
pathologic condition has caused peripheral nerve demyelination, generalized slowing of
conduction velocity occurs. In other cases, nerve conduction velocity can be slowed in a focal
segment; this is characteristic of compression (entrapment) neuropathy. For example, slowing of
the median nerve distally (below the wrist) with normal velocities above the wrist occurs in
carpal tunnel syndrome.
Electromyography (EMG)

EMG is performed by inserting a coaxial needle electrode in the muscle and recording motor unit potentials on a cathode-ray oscilloscope. The muscle potentials are characterized in three phases: resting state, mild muscle contraction, and maximal exertion. At rest, there is normally brief insertional activity and later no spontaneous motor potentials. The presence of prolonged spontaneous discharges is consistent with that of denervating (anterior horn cell or axonal neuropathy) disorder. During mild muscle contraction, muscle potentials are evaluated (e.g., size, amplitude, duration, form). With maximal contraction, there should be sustained electrical activity; this is decreased in myopathic, but not in neuropathic, disorders. On the basis of characteristics of electrical activity, it is possible to define the cause of weakness and usually to differentiate disorders of myopathic or neuropathic origin. If a disorder of neuromuscular junction (e.g., myasthenia gravis) is suspected, repetitive stimulation of a motor nerve should also be performed. The myasthenic syndromes show deremental changes in action potential amplitude with repetitive stimulation.

Muscle and Nerve Biopsy

In patients with symmetric muscle diseases, EMG is used to define which muscle groups show abnormal electrical response, and biopsy is performed to define specific pathologic changes in the involved muscles. Biopsy is performed on a muscle that has not been sampled by EMG and is free of needle artifacts. The muscle to be biopsied is determined by clinical, and EMG findings. It is important to select a muscle that is clinically and electrically in an active phase of involvement. If muscle sampled is not chosen carefully, the biopsy may show no abnormality or only end-stage atrophic changes; therefore, it would not be possible to determine if primary pathologic process was myopathic or neuropathic. The biopsy is performed by making an incision in skin and excising a small piece of muscle. This specimen should not be placed in or irrigated with alcohol or saline. The tissue should be divided into three fragments: one fragment is frozen for cryostat sections and histochemistry, the second is fixed for electron microscope analysis, and the third is used for biochemical (metabolic) analysis.

The sural nerve is a cutaneous sensory nerve that is available for biopsy. Biopsy is indicated in patients in whom the cause of nerve disease cannot be established by other diagnostic modalities. The major limitation is that only sensory nerves can be studied; motor fibers cannot be evaluated. If the pathologic process is multifocal rather than diffuse, the abnormal area can not be included in biopsy specimen. Nerve biopsy is most helpful to delineate these pathologic processes: inflammatory demyelinating neuropathy, amyloid, sarcoid, leprosy, vasculitis, and biochemical disorders affecting peripheral nerve (e.g., metachromatic leukodystrophy).

Summary

Neuroimaging studies are valuable when used appropriately after a careful history and examination have suggested the possible location and type of neuropathologic process. With sophisticated neuroimaging studies it is not only possible to detect a brain or spinal cord lesion but also to image the effect of the lesion on the surrounding structures. In the future, these imaging techniques will provide biochemical and physiological information of brain dysfunction as well as structural imaging the brain or spine lesion.
SUGGESTED READINGS

Electroencephalography


Lumbar Puncture and Cerebrospinal Fluid Analysis

Attia J, Hatala R, Cook DJ, Does this adult patient have acute meningitis, JAMA 282:175, 1999.


Vascular Ultrasound


Moore WS. For severe carotid stenosis found on ultrasound further arterial evaluation is unnecessary, Stroke 34:1816, 2003.


Rothwell PM. For severe carotid stenosis found on ultrasound further arterial evaluation is unnecessary: the argument against, Stroke 34:1816, 2003.


**Conventional Angiography and Magnetic Resonance Imaging Angiography (MRA)**


**Computed Tomography and Magnetic Resonance Imaging**


Lufkin RB: Magnetic resonance imaging of the central nervous system, Semin Neurol 6:1, 1986.


**Positron Emission Tomography and Single Photon Emission Tomography**


**Electromyography and Nerve Conduction Velocity**

Evoked Potentials


TABLES AND FIGURES

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<tr>
<td>NCV</td>
<td>Measurement of integrity of myelin sheath</td>
<td>Suspected neuropathy</td>
<td>Minimal electrical shock</td>
</tr>
<tr>
<td>EMG</td>
<td>Determine electrical properties of muscle, nerve, anterior horn cell</td>
<td>Suspected neuro-muscle disease</td>
<td>Needed insertion in muscle causes mild discomfort</td>
</tr>
<tr>
<td>EP</td>
<td>Summated brain wave after visual, auditory, or sensory stimulation</td>
<td>Optic nerve, brain stem, spinal disease</td>
<td>None</td>
</tr>
</tbody>
</table>
Figure 2-1. A, Skull roentgenogram showing postoperative defect and surgical clips in a patient with an enlarged sella turcica and suprasellar calcification caused by a craniopharyngioma. B, Normal skull roentgenogram with normal size sella turcica

Figure 2.1
Figure 2-2

A. Well-modulated symmetric normal alpha rhythm.

B. Diffuse severe delta rhythm in a patient with a metabolic encephalopathy.

C. Right hemisphere (even number of the montages) slow wave focus in a patient with malignant brain neoplasm.
Figure 2.3

**A**. Normal carotid angiogram visualizing the caliber and position of the intracranial vessels.

**B**. Giant aneurysm arising from the basilar artery.

**C**. Aneurysm arising from the middle cerebral artery.
FIGURE 2.4. A, Base of brain shows the triangular-shaped fourth ventricle and directly anterior is the brain stem and surrounding basal cisterns. The dorsum sella is seen and obscures the suprasellar cistern. The anterior clinoids are lateral to the dorsum sella. Anterior to sphenoid bone is frontal lobes. Between sphenoid and petrous bone is temporal lobe. Cerebellar hemispheres are posterior to petrous bone.

FIGURE 2.4, cont’d. B, Midventricular level shows anterior frontal horns of lateral ventricles, midline third ventricle, calcified pineal gland and quadrigeminal cistern. C, High ventricular level shows anterior frontal horns, bodies and occipital horns of lateral ventricles.

FIGURE 2.4, cont’d. D, Supraventricular level shows cortical sulcal spaces. The gray matter appears slightly brighter than deep white matter.

Figure 2.4
Figure 2.5

**FIGURE 2.5.** Postcontrast CT. A, Carotid arteries are seen in lateral-anterior aspect of suprasellar cisterns branching into middle cerebral artery extending laterally and anterior cerebral arteries extending medially. The right posterior cerebral artery is seen extending posteriorly from the midline basilar artery. B, The round vein of Galen extends to straight sinus and posteriorly into sagittal sinus. C, The midline falx cerebri appears as linear enhancing band.
Figure 2.6

**Figure 2.6.** CT visualized pathological processes. A, Blood in the basal ganglia extending into anterior frontal horn, third ventricle, and occipital horn of lateral ventricle.

**Figure 2.6, cont’d.** B, Enhancing thalamic and posterior third ventricular neoplasm elevating the third ventricle. The large, comma-shaped structure is the dilated temporal horn.
Figure 2.7

**Figure 2.7**

**A**. T₁ weighted axial MRI shows base of skull, including triangular-shaped fourth ventricles. CSF spaces appear dark, similar to appearance on CT.

**B**. T₂ weighted axial MRI shows CSF spaces, including lateral ventricles and spaces appearing white.

**C**. T₁ weighted sagittal scan shows cerebral hemispheres, corpus callosum, brain stem, cerebellum, and cervical spinal cord.
FIGURE 2.8. A, High-signal intensity (white) periventricular lesions consistent with multiple sclerosis.

FIGURE 2.8, cont’d. B, Coronal craniospinal study shows large CSF filled (dark) space in central spinal cord consistent with syringomyelia.

Figure 2.8