Chapter 1 – The Neurologic Examination

Section 1

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As family practitioners you will see patients with complaints that cover the full spectrum of medical practice. Many of these patients present with symptoms of pain, dizziness, forgetfulness, numbness, weakness and difficulty speaking or comprehending as their primary complaint, or as a portion of their history. In addition to a full medical evaluation, accurate assessment of these neurologic complaints will be of increasing importance to our current health care environment. Since up to 10–15 percent of a family practitioner’s workload consists of neurologic problems, it is the goal of this program to provide an effective and efficient means of gaining this knowledge.

As a first step in evaluating the patient with a neurologic problem, the practitioner must obtain an accurate history. A good history alone will often suggest the correct diagnosis, and the examination can be tailored to specifically search for corroborating physical signs. Patients with neurologic disease may have impairments that make it difficult to elicit accurate information, and the diligent examiner may need to spend extra time questioning the patient or obtaining information from family or friends.

An important consideration in history-taking is to not only record the patient’s complaint, e.g., dizziness, but to question exactly what the patient means by that complaint. The symptom “dizziness” often has different meanings to the lay public and the term could be used to connote lightheadedness, vertigo, tiredness, or malaise. If the examiner assumes it means vertigo then needless time and resources may be wasted in pursuing a non-existent complaint. Another example is “weakness” which to many patients may mean fatigability or lack of energy rather than loss of strength in specific muscle groups.

The temporal profile, i.e., whether it is acute, subacute, or chronic, is important in determining the diagnosis. For example, numbness and weakness of an extremity that is abrupt in onset suggests transient cerebral ischemia or stroke, while the same symptoms, if they develop over minutes, may be associated with
the aura of migraine. Progression of the same symptoms over days may be due
to a brain abscess and over weeks to months, a brain tumor.

One can develop a pattern of questioning for this portion of the history, which
is facilitated by a predetermined outline. A partial, but useful list should include
questions about the following symptoms:

- Visual defect
- Diplopia
- Hearing disturbance
- Vertigo, lightheadedness
- Swallowing difficulty
- Speech disturbance
- Behavior/mood change
- Aphasia
- Headache
- Bladder/bowel control
- Involuntary movement
- Weakness
- Gait disturbance
- Incoordination
- Muscle atrophy
- Tremor
- Pain
- Numbness
- Paresthesia/anesthesia
- Memory
- Seizure/syncpe

**The Neurologic Examination**

The neurologic examination should always be included as part of the general
physical examination.

An adequate neurologic examination requires a few simple tools: a reflex
hammer, disposable pin, cotton, tuning fork, tape measure, visual acuity card,
aromatic substance to test smell, and printed copies of the Mini-Mental State
Examination to include on the patients chart. These are small enough to carry
in laboratory coat pockets or in a small travel case.

The following overview emphasizes points that a family practitioner should
keep in mind; Examples will be used to point out the significance of each portion
of the examination and how the findings relate to the patient’s medical picture as
a whole.

Basic neuroanatomy will be reviewed but it will be done in a way that is clinically
useful and easy to remember. Note that the descriptions may differ slightly from
precise anatomical pathways and neurophysiologic relationships, but will serve
adequately for clinical localization and treatment application. In this fashion we
hope to provide an educational tool that has utility and is user friendly.

A video is included that will demonstrate how to correctly perform and interpret
the neurologic exam. It is to be used in conjunction with the text. Individual
performance of the examination can be refined by repeated performance and
evaluation by your clinical neurology faculty person.

**Inspection and Observation**

From the time the physician greets the patient he gets to observe him speaking,
sitting, walking, making facial expressions and socially interacting. Examples
are the hemiparetic or parkinsonian gait, facial asymmetry due to facial muscle
weakness, presence of tremor, dysphasic or dysarthric speech and a whole host of other clinical signs. Patients may have pronounced muscle atrophy, the distribution of which can help provide important diagnostic clues. These signs are often missed solely because they are not looked for and, once pointed out, are obvious. Obtaining and utilizing information such as this is what makes a good clinician.

The psychological state of the patient should also be noted. Patients may be depressed, hostile, apprehensive, preoccupied and even uncooperative. Recognizing such moods will help the examiner choose the approach best suited to maximize the information he obtains from the encounter. Reassurance and patience on the part of the examiner go a long way in gaining a patient’s trust and cooperation.

The presence of pain may affect a patient’s countenance, gait, and even ability to cooperate during the examination. It takes experience, gained by doing many of these examinations, to be able to recognize when patients are truly impaired or when symptoms and signs are exaggerated for secondary or other gain.

System Integration

When neurology is learned in the classic sense, the student studies individual functional neurological systems such as the motor, sensory, and cerebellar systems. This enables a greater depth of understanding, but in a one-month clinical rotation, time does not permit this luxury. If one is to gain clinically useful information in a limited time frame, it is best to provide understanding of broader functional concepts. These concepts must easily lend themselves to clinical application in patient care settings. In the spirit of this approach the following description will illustrate how the body moves by discussing the interactions of the extrapyramidal, pyramidal, sensory and cerebellar functional systems.

A useful concept is to imagine the human body as a marionette, all of whose controlling strings have no tension, and the puppet lies crumpled on the floor (Figure 1-1). For the puppeteer to create life-like movements, he must first stand the puppet up. To do this he puts tension on the strings that cause the trunk to become erect and the legs to extend (Figure 1-2). This provides the basic framework to initiate motion of the extremities. By analogy, a similar but lengthier process occurs in human infants as their nervous systems mature. The infant first lies supine on its trunk, with arms and legs flexed. Gradually, extensor tone straightens the legs and trunk, and eventually the baby is able to sit up and then stand even though it wobbly and must grasp for support.

The portion of the nervous system responsible for this function is the extrapyramidal system. It consists of a number of reverberating circuits in the basal ganglia and brain stem that ultimately send impulses through spinal cord pathways that tonically innervate spinal interneurons controlling the tone of muscles which
support the spine and keep the body erect. This is all done on an unconscious level. When something goes wrong with this system, as in Parkinson disease, the normal erect posture of the body becomes flexed, and more rigid. As the extrapyramidal system matures, fluid control of body posture provides the framework for initiation of individual extremity movements.

Voluntary movements, (Figure 1-3), are largely initiated by the pyramidal system. Impulses go from premotor integrating areas in the frontal lobe to the upper motor neuron, which sends a crossed axon to the anterior horn cell in the spinal cord. Pyramidal tract initiated movements are crude and lack finesse. They are smoothed out and made more agile by the influence of other systems, such as the cerebellar, and via practice effect. There is also an inherent ability of the system to mature to certain degrees explaining the phenomenon of the “natural born athlete.” Lesions of the pyramidal tract produce weakness, increased clumsiness, and alteration of motor tone. This will be discussed in greater detail later.

The development of erector tone, which has provided the supporting framework, and the initiation of voluntary movements are, however, still rudimentary and uncoordinated. What is needed is a system that monitors the motor activity and then smoothes out any irregularities in the desired action.

The cerebellum occupies a large portion of the posterior fossa, and is in a unique position to monitor impulses entering and leaving the brain. It’s foreboding anatomical structure with its many lobes and folia often discourages students from getting a better understanding of how this elegant structure works. A most useful concept is to think of the cerebellum as a servomechanism. A good example of such a device is the automatic piloting system on an airplane. The pilot will set the autopilot to control speed at “x” knots, the course at a certain latitude-longitude, and the altitude at so many thousand feet. The servomechanism is basically a computer which compares the actual airplane speed, read from the speedometer; the altitude, read from the altimeter; and course (direction), read from a compass; to the settings that the pilot has entered as desired. Any discrepancy between the desired and actual readings will be corrected by output from the computer. Altitude and position can be adjusted by moving the wing flaps, direction changed in similar manner, and speed by increasing or decreasing engine revs.

The cerebellum works in a manner similar to a servomechanism. It receives input from the sensory system and information about output from the pyramidal and extrapyramidal systems. When a person swings a tennis racquet, impulses travel down the pyramidal pathway to specific anterior horn cells in the spinal cord, which initiate movement. These same impulses are sent to the cerebellum, which receives them before the anterior horn cells, so that the cerebellum knows what movement is intended. As the arm begins to move, sensory proprioceptors send information back to the thalamus and sensory cortex so that the person is aware of his achieved arm movement. The cerebellum “knows” what was intended and what is actually being achieved. If there is any discrepancy, the cerebellum corrects this via inhibitory outflow pathways, which alter muscle tone and action.
One can thus visualize a dynamic and fluid interaction between these three systems, which enable the body to move in the most efficient manner. It is derangement of one or more of these systems that produce the pathologic states seen in symptomatic neurologic disease. Understanding how these systems work will enable the clinician to recognize and localize nervous system disorders.

An example from the preceding concept is illustrated by the clinical finding of ataxia. Ataxia is defined as motor incoordination but may be produced by lesions involving motor, sensory or cerebellar pathways. If a patient is noted to have arm clumsiness on finger to nose testing, this could be secondary to weakness of arm and hand muscles, to loss of proprioception in the upper extremity or due to a cerebellar lesion. If weakness is present, the clumsiness is defined as motor ataxia.

If strength is normal, and there is a marked proprioceptive deficit, such that the arm’s position can only be determined by the patient looking at it, then we have sensory ataxia as the cause of arm clumsiness. Sensory ataxia of lower extremity and truncal muscles produces Romberg’s sign whereby the patient can only maintain balance while standing if his eyes are open. This is because he has absent proprioceptive cues and must rely on vision to keep his balance.

Finally, if motor strength and sensation are normal and incoordination is still present, it is most likely of cerebellar origin. Localizing lesions to specific portions of the cerebellum will be covered later in this chapter.