HANDBOOK OF

Symptom Oriented Neurology

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Preface

The prediction in the preface to the first edition of this book published 7 years ago, that CT scans would probably never be available in small communities, has turned out to be very wrong. In fact, advances in computerized medical technology have been revolutionary. In most communities now, CT scans are not only readily available, but also considered routine in the evaluation of neurologic disorders. At the present time, magnetic resonance imaging (MRI) with its superb resolution of detail is the leading technological innovation for evaluating nervous system structural integrity, but is only available at the larger medical centers. However, considering the fate of our prediction regarding CT scans, it is possible that in half a dozen more years MRI scans will also be readily available throughout the country.

Also since the last edition, two diseases — Alzheimer's disease and the acquired immunodeficiency syndrome (AIDS) — have moved to the forefront of the health concerns of the general public. With our increasingly aged population, more and more cases of the progressive dementing disorder Alzheimer's disease are being recognized. With the greater public awareness of this devastating and untreatable condition, it is imperative that physicians not only recognize the clinical symptoms and provide patient counseling, but also identify treatable conditions with similar symptomatology. On the other hand, AIDS provides a much more difficult problem, since the causative viral agent is highly neurotropic and can produce a wide variety of symptoms related to this infection. In many respects this infection deserves the appellation once applied to syphilis infections — "the great imitator."

In this edition, we bid farewell to Dr Lee A Christoferson who was a valued collaborator in the first edition. Fortunately, his place has been amply filled by Dr Vasudeva Iyer as a new coauthor. We would also like to acknowledge the helpful comments of Mr Larry Burd and Drs Philip Becker, Jack Kerbeshian, and Ed Harder concerning several of the chapters.

Preface to the First Edition

This book was written to assist the primary care physician in diagnosing and managing the common, the treatable, and the emergency neurologic problems. No attempt was made in this text to write an exhaustive treatise of neurologic disease, to present neurologic pathophysiology, or to detail the diagnosis of rare, untreatable neurologic disorders. We believe that this book will enable primary care physicians to take a neurologic symptom and arrive at a neurologic diagnosis; this in turn will improve patient service and reduce referrals to tertiary care centers.

The content and philosophy of this manual was based on a paper by Dr T. J. Murray (Concepts in undergraduate teaching. *Clin Neurol Neurosurg* 1976; 79[4]:237-284) which addressed the issue of common neurologic complaints presenting to family physicians in Canada. Although 10% of all patients seen in family practice had neurologic complaints, only 2% received a "neurologic diagnosis." For example, the diagnosis of a patient whose major presenting problem is diabetic neuropathy instead of fluctuating blood sugar levels is still diabetes rather than peripheral neuropathy.

Dr Murray's list of primary care complaints is based on frequency of the problem, potential seriousness of the disease, and the effect of intervention on the outcome. Headache is included because it is a frequent complaint, although intervention is rarely a life-or-death matter. Infections of the nervous system are also included because, although they are relatively rare, they require immediate diagnosis and treatment. Our chapter headings closely follow Dr Murray's list.

Primary care physicians are forced by circumstances to see a large number of outpatients per day. There is often insufficient time to evaluate each patient's problem in depth. While it is an easy approach for the specialist in neurodiagnosis to advise the primary care physician to do a "complete history and neurologic examination," such advice is unrealistic. Therefore, we have prepared a symptom-oriented, problem-oriented manual, which falls short of complete neurologic diagnosis and treatment, but which should improve present patient care. The "minimum histories" and "minimum examinations" suggested in the text certainly do not define every patient's problem but have been developed to encourage a uniform, reasoned approach, which is more efficient and increases the probability of a correct diagnosis in the majority of cases. Specific diagnosis will assist in making appropriate referrals for special tests and treatment. Once a diagnosis is established, the primary care practitioner may refer to any one of a number of fine neurologic textbooks for

a more detailed description of the disorder. In addition, selected references are listed at the end of each chapter.

A major problem in writing a text of this sort is a lack of uniformity of diagnostic resources available to the primary care physician. For example, the authors believe that most stroke victims need four-vessel cerebral angiography and computed tomographic (CT) scans for optimal care, but (1) angiography has its risks and should be undertaken only by a radiologist with considerable experience; and (2) it is unlikely that CT scans will be available in every community in this country. Therefore the use of these diagnostic procedures in establishing the diagnosis may be unrealistic in a particular community. A similar statement may be applied to other neurodiagnostic tests such as electromyography, electroencephalography, myelography, etc. Although we have no solutions, we have considerable empathy with the primary care physician faced with limited resources. In such situations we believe that this text will help the primary care physician to make an accurate diagnosis and to confidently make a clinical judgment regarding the need for further tests and appropriate treatment.

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I

THE NEUROLOGIC EXAMINATION

Contrary to popular opinion, there is no "standard" neurologic examination. When we are requested to teach the neurologic examination, our response is "The neurologic examination of what? The ambulatory adult? The infant? The comatose patient?" A neurologic examination should be *problem-oriented*, and in reality there are different examinations for different clinical situations. Therefore we have included many of our suggestions for the neurologic examination under specific chapter headings. In most circumstances we suggest that common sense should prevail. For example, testing smell is of little help in the diagnosis of a primary muscle disease, and testing the anal wink is of little value in diagnosing the average patient with a headache. In essence, the neurologic examination is a process of gathering objective data for the hypotheses formed during the process of history taking.

THIS CHAPTER IS INTENDED TO PROVIDE HINTS ON THE MORE COMMONLY USED (AND ABUSED) PORTIONS OF THE NEUROLOGIC EXAMINATION. IT IS NOT A COMPLETE GUIDE TO THE ENTIRE PROCEDURE.

Screening of Neurologic Abnormalities — Station and Gait

Table 1.1 outlines the procedure for station and gait testing, and Figure 1.1 illustrates the procedure. It usually can be performed in less than a minute. Table 1.2 emphasizes that virtually every aspect of the central and peripheral nervous system is tested. A patient with a normal station and gait is unlikely to have any serious structural neurologic abnormality. Twenty feet of straight walking space is desirable, and the patient should be barefooted and clothed only in underwear or a gown.

TABLE 1.1
Procedure for Station and Gait Testing

INSTRUCTIONS	THINGS TO NOTE
1. Walk the distance normally	Asymmetric arm swing, abnormal arm and hand postures, and instability of the trunk
2. Rapidly turn around and walk on tiptoes	Extra steps while turning around and inability to rise completely on the tips of the toes
3. Rapidly turn and walk on heels	Foot drop
4. Turn and walk with heels touching toes (tandem walk)	Instability characteristic of midline cerebellar lesions
5. Turn and "walk on outsides of feet like a bowlegged cowboy does" (walking on lateral aspects of feet)	This maneuver specifically brings out hemiplegic posturing of an arm from subtle or old upper motor neuron damage
6. Do a deep knee bend (preferably with hands on hips; if there is an obvious balance problem, patient may hold onto an object, such as a chair)	Loss of balance indicates cerebellar difficulties; inability to rise indicates proximal weakness
7. Stand with feet together, eyes closed, arms outstretched with palms facing ceiling and fingers spread apart	Increased swaying with eyes closed indicates either posterior column disease or a peripheral neuropathy; with subtle hemiparesis affected arm will pronate, while in more obvious hemiparesis the arm will pronate and then drift downward and outward.

By observing the station and gait, a skillful examiner can obtain in one minute a glimpse of mental status (how well the patient comprehends and follows instructions), upper motor neuron function (posturing of arms and gait), lower motor neuron function (muscle atrophy and weakness), muscle disease (prox-

imal weakness), basal ganglia function (abnormal posture and movement), cerebellar function (balance and tandem walk), and the sensory system (poor

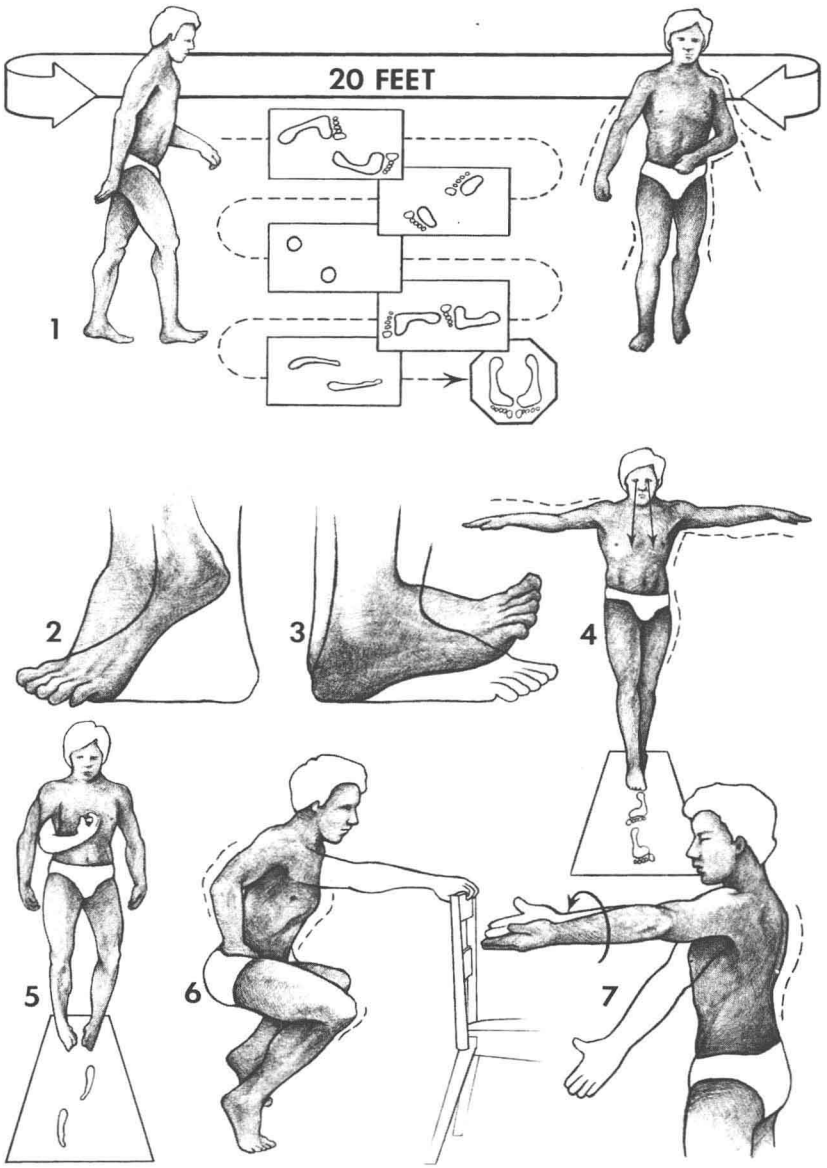

Figure 1.1 Testing of station and gait. Pay particular attention to arm swing, arm posture, body posture, instability while turning around, tendency to look at floor. See text for additional information.

TABLE 1.2
Possible Abnormalities of Station and Gait

Abnormal mentation: Patient follows directions poorly, slowly; needs examiner to demonstrate instructions; tendency to continue doing same task (perseveration)
Hemiplegia: Decreased arm swing on affected side, circumduction of leg, pronation of arms when held outstretched with palms up, flexion of arm when walking on sides of feet
Cerebellum: Unsteadiness when turning around, in tandem walking, and in deep knee bending
Sensation: Increased swaying when eyes are closed (positive Romberg test)
Muscle disease: Difficulty with deep knee bend, waddling gait
Basal ganglia: Abnormal postures and movement (eg, Parkinson's syndrome, Huntington's disease)
Lumbar disc: Inability to walk on heels or toes on one side; spinal list
Peripheral neuropathy: Bilateral foot drop; cannot walk on heels

balance with eyes closed — the Romberg test). A patient who can perform all the maneuvers normally will rarely have a significant neurologic abnormality. Abnormalities noted can be more specifically tested in the remainder of the neurologic examination. For example, if station and gait testing suggests a cerebellar abnormality, more specific cerebellar tests should be performed.

Deep Tendon Reflexes

The most difficult part of the neurologic examination to perform correctly (and one that medical students think is easiest) is the evaluation of deep tendon reflexes (Figures 1.2 — 1.5). If at all possible, have the patient undressed and sitting with legs dangling freely over the edge of the table. The reflex elicited will depend on:

1. Whether or not the tendon is struck
2. How hard the tendon is struck
3. How quickly the tendon is struck

To avoid striking an improper area, the tendon should first be palpated. The lightest tap that will still elicit the response should be given. A hammer with a relatively soft rubber end and a flexible handle will best allow the rapid, light tap. The examiner will most often find asymmetry of reflexes rather than gross hyperactivity or absence of reflexes. Reflexes may be normal, hyperactive or hypoactive, clonic or absent, or symmetric or asymmetric and should be recorded as such. Recording pluses, minuses, or whatever, unless carefully defined, do little to convey accurate information in the chart.

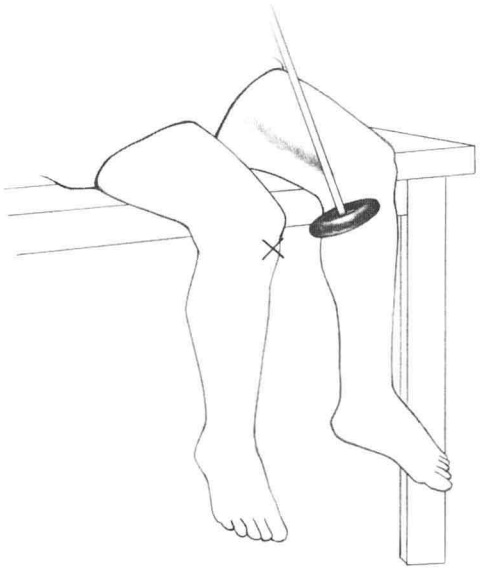

Figure 1.2 The patellar reflex: Note that legs do not touch the floor. The type of hammer illustrated was developed in England and is especially effective. Look not only for reflex contraction of the quadriceps but also contralateral contraction of the adductor muscle and the number of swings the leg makes. *Remember*: Dysfunction of either the afferent or efferent nerves may diminish the reflex.

Figure 1.3 Achilles reflex: While striking the tendon, have the patient apply *light* pressure with the sole of his foot to the palm of the examiner.

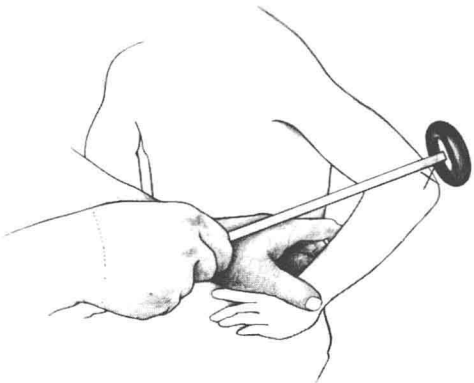

Figure 1.4 Triceps reflex: This reflex is most easily elicited when the patient rests his arms on his hips.

The Babinski Reflex

The Babinski reflex (Figure 1.6) is the eponym given to the plantar response, which may be present depending on:

1. Type of stimulation used
2. Rapidity with which the stimulus is delivered
3. The position of the patient

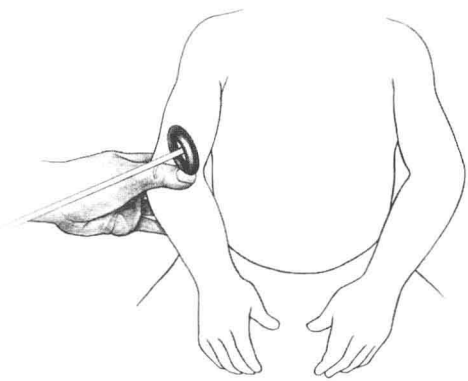

Figure 1.5 Biceps reflex: It is very important to have the arms symmetrically flexed and relaxed as illustrated.

A sharp object (safety pins or the sharpened ends of some hammers) will produce little more than a withdrawal response, while too light a touch will produce no response. We find a key to be the most readily available, appropriate stimulus. The key is used to stimulate the *lateral* aspect of the plantar surface of the foot, beginning at the heel and moving up to the ball of the foot but staying lateral to the great toe. Examples of some responses to plantar stimulation are shown in Table 1.3.

Because the abnormal response is such an important sign of nervous system disease, the best approach to recording the results, if in doubt, is to record exactly the observed movements. It is totally inadequate simply to say "Babinski absent." Of course he is — he died a half century ago.

Examination of the Optic Fundus

Examination of the optic fundus with the ophthalmoscope is the only opportunity the physician has to look directly at the brain, and it is imperative to do so on *every* patient with neurologic symptoms. This should be done even in difficult cases, such as a crying, hyperactive 4-year-old child. Mentally make a list of those parts of the fundus which must be seen to confirm the hypotheses formed during the history; for example, in the patient with suspected multiple sclerosis, look particularly for temporal pallor of the optic disc. Adjust the size of the beam to match the size of the pupil (too large a beam causes light to reflect from the iris). Using too bright a beam may cause excessive pupillary constriction. In general, use the brightest light possible that still allows visualization of the retina. Darkening the room may be helpful in certain difficult patients. Pupillary dilating agents are usually not necessary.

The Pharyngeal Reflex

The pharyngeal reflex (see Figure 1.7) should be tested on each side by stimulating the pharyngeal pillars with a cotton swab on an applicator. After observing the motor response (elevation of the palate), ask the patient if the sensation was the same on both sides of the pharynx. (Simply jamming a tongue depressor down the patient's throat not only gives very little neurologic information but is downright ungentlemanly.) Response is significant only if it is asymmetric; the normal gamut of responses runs from hyperactive to hypoactive.

Sensory Examination

The sensory examination under most clinical conditions does not produce objective, "hard" data because it involves subjective judgments by both the patient and the examiner. Beginning medical students are often fascinated by

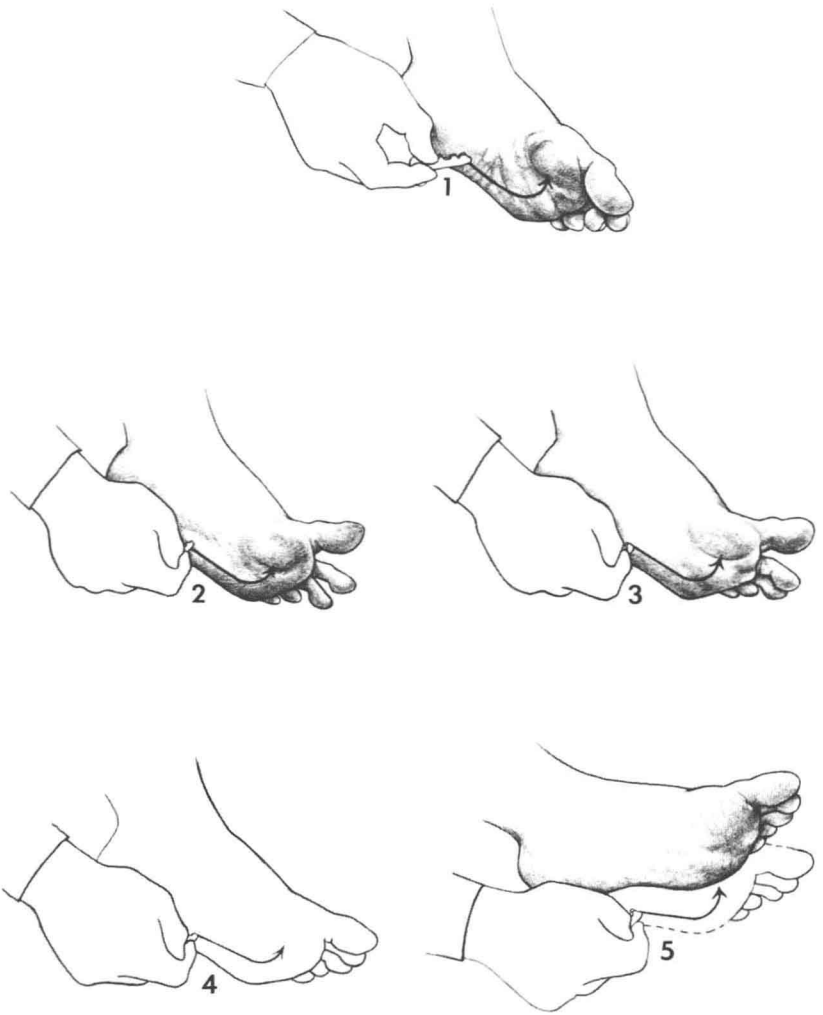

Figure 1.6 Plantar stimulation: Note that only two of the five possibilities constitute a "positive Babinski." See text for additional information.

the sensory examination and spend an inordinate amount of time performing it. If pinprick, vibration, light touch, and position sensation are present in the feet and if the patient can recognize numbers written on the palms of the hands with eyes closed, a major sensory deficit in unlikely. On the other hand an intelligent, cooperative patient with a circumscribed sensory deficit, given a pin and a marking pencil, can often outline the deficit accurately. Likewise, when looking for a sensory level, have the patient run his or her own finger

TABLE 1.3
Responses to Plantar Stimulation

NAME	OBSERVATION	INTERPRETATION
1. Normal response (flexor plantar response)	First movement of great toe is flexion	Normal
2. Classic Babinski reflex (classic extensor plantar response)	Extension of great toe with extension and fanning of other toes	Most often seen in upper motor neuron lesions (above the L-5 spinal segment)
3. Babinski reflex (extensor plantar response)	First movement of great toe is extension (there may be subsequent flexion of great toes); other toes either show no movement or flexion	Seen in all types of upper motor neuron lesions (above the L-5 spinal segment)
4. Mute plantar response	Nothing happens	Severe sensory loss or paralysis of foot
5. Withdrawal	Patient pulls foot back	Often seen in metabolic neuropathies or if examiner uses excessively sharp object
6. Asymmetric response	Mute plantar response on one side and flexor plantar response on other side	Indication of need to look for other signs of neurologic disease

up the body until sensation changes. When a peripheral neuropathy is suspected, ask the patient to compare a single pinprick proximally (such as on the chest) with a single pinprick distally (such as on the foot). Use a hat pin and allow the shaft to slide through the finger in order to deliver a relatively quantitative response (see Figure 1.8). Simply comparing sharp and dull on the foot is inadequate. The examiner should attempt to quantitate any difference between the proximal and distal stimulation sites. For example, say, "If this [chest] pinprick is worth $100, how much is this [foot] pinprick worth?" and consider a response less than $75 as significant. A useful, objective sign