POCKET BOOK OF CLINICAL NEUROLOGY

SUCHENWIRTH

SECOND EDITION

Pocket Book of

Clinical Neurology

By

Richard Suchenwirth

With 86 figures, including 69 diagrams by
Dieter Freiherr Von Andrian

Translated and Adapted by

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To My Neurology Students

Preface

Planned to assist the clinician in his handling of neurologic cases, this concise work covers those principles and facts which, in the author's experience, are most frequently required in practice. Wherever possible, reduplication and unimportant or unproved details are omitted. The numerous illustrations are an integral part of the text. A selection of tables for convenient reference will be found at the back of the book.

Separation of the textual matter into topographic neurology and neurologic nosology is retained as far as possible, and no attempt is made to deal with examination technique. The diagnosis becomes obvious at the conclusion of each exercise, but no account is taken of its implications, which would demand a complete review of the patient's somatic, psychologic and sociomedical background. In the orchestra of modern medicine, the neurologist plays only one instrument, albeit an important one.

Investigative techniques receive only brief mention. In many instances, they are unavoidable but they can be overused: the case history, the health of the patient's family and the clinical examination at the bedside are the three pillars of diagnosis and treatment of neurologic diseases. They also form the basis for evaluating progress.

Drugs also receive sparse attention. The reason is that, apart from those that are proved and established, many remain of limited value in modern neurology.

The author thanks his teachers, colleagues and medical students, whose questions always provoke cleared explanations of problems. Credit is also due the publisher for the attractive format of the book.

Here and there, the experienced may criticize passages as being too elementary or too brief, and the author welcomes critical suggestions.

Richard Suchenwirth

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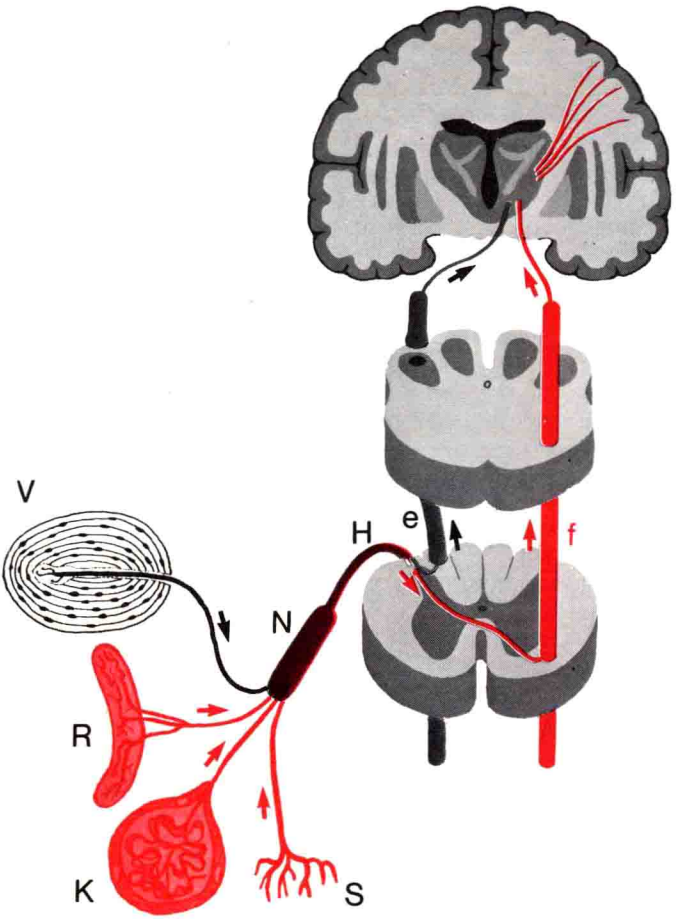


FIG. 1.—Sensory impulses pass toward the brain along the peripheral nerve *(N)* and the posterior nerve root *(H)* into the spinal cord. The impulses arise in the sensory receptors for tactile sensation (Pacinian corpuscles, *V*), heat (Ruffini's corpuscles, *R*), cold (End Bulbs of Krause corpuscles, *K*) and from pain receptors *(S)*, which are distributed widely in the skin. Tactile sensations pass in the posterior *(e)* and lateral *(f)* columns, pain and temperature sensations only in the lateral columns, to the brain.

Manifestations of Peripheral Nerve, Spinal Cord Disorders, and CSF Syndromes

Sensory Disturbances

The patient describes the symptoms of *spontaneous sensory phenomena* (pain and paresthesias: itching, prickling, burning, sensation of pins and needles, and *sensory deficits* (reduced or absent sensitivity to touch, pain, hot and cold). Spontaneous phenomena of sensation point to a mild dysfunction, a deficit to a more severe disturbance of the sensory system. *Local changes* in the epidermis or subcutaneous tissues may stimulate or damage some of the 500,000 pressure receptors, 3 million pain receptors or 100,000 hot and 60,000 cold receptors, just like a skin disease. The topographic extent of the sensory disturbance will indicate whether the deficit involves only one *peripheral nerve* (most frequently the ulnar nerve, then the peroneal, radial and median) or several nerves (polyneuropathy). All modalities of sensation will be affected within the territory of one or more nerves (cutaneous fields of peripheral nerves, see p. 184). In *diseases of a posterior root* (or the posterior horn cells in certain spinal cord diseases), a segmental loss of all sensory modalities occurs in the corresponding dermatome (commonly S1, L5 and C8). If several segments are involved, a plexus lesion may be present (brachial or lumbosacral). In a transverse lesion causing *damage to the spinal cord* (transverse myelopathy), all sensation below the level of the lesion is lost, including the awareness of bladder and rectal distention.

If the damage is confined to the *anterior spinal cord* (usually syringomyelia), intraspinal tumors and the anterior spinal artery syndrome), a *dissociated sensory loss* may be present: the modalities of pain and temperature are abolished or significantly reduced below the level of the lesion.

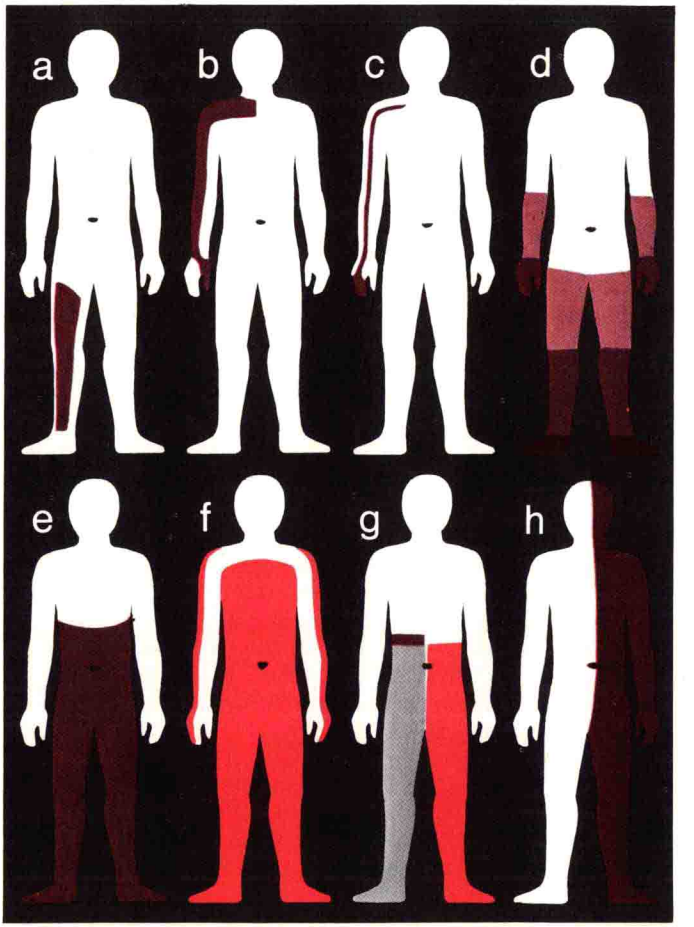


FIG. 2.—Depending on the site of the lesion, all sensory modalities are disturbed (brown), only temperature and pain (red) or only touch (gray). The following types may be distinguished: mononueropathy (e.g., femoral nerve: **a**), plexopathy (e.g., upper brachial plexus **b**), radiculopathy (C7:**c**), distal polyneuropathy **d**), transverse myelopathy **e**), anterior spinal cord syndrome **f**), or unilateral spinal cord syndrome **g**), as well as cerebral hemihypesthesia **h**).

Touch sensation is also mediated by the posterior columns; therefore, in such lesions, it escapes extinction.

Unilateral damage to the spinal cord (Brown-Séquard type) is characterized by loss of homolateral tactile sensation (also motor strength) and of contralateral pain and temperature sensation below the level of the lesions. The damaged half of the cord contains the uncrossed tracts for tactile sensation and the crossed pathway for pain and temperature. Ataxia on the side of the lesion is an additional feature, caused by damage to the spinocerebellar tracts. At the level of cord injury, all modalities of segmental sensation are disturbed, since the fibers mediating pain and temperature are affected before they cross. Signs of a segmental deficit of anterior horn cell function are also present; the tendon reflexes are lost at this level.

Thalamic lesions affect all modalities of sensation on the opposite side of the body, causing either irritation or loss; one speaks of a hemihypesthesia or hemianesthesia. At the level of the thalamus, all the tracts subserving sensation have crossed. The sensory phenomena characteristically do not extend to the midline, possess a burning and radiating quality and often are accompanied by excessive affective responses.

Central disturbances that localize to the cerebral cortex (postcentral region) often accompany curtailment of higher perceptive functions (agnosia). Pain perception is affected less severely. Specific features of integrated *epicritic function* are affected early and recover late. They include graphesthesia—recognition of numbers drawn on the skin; pallesthesia—vibration sense (tuning fork); tactile localization; two-point discrimination.

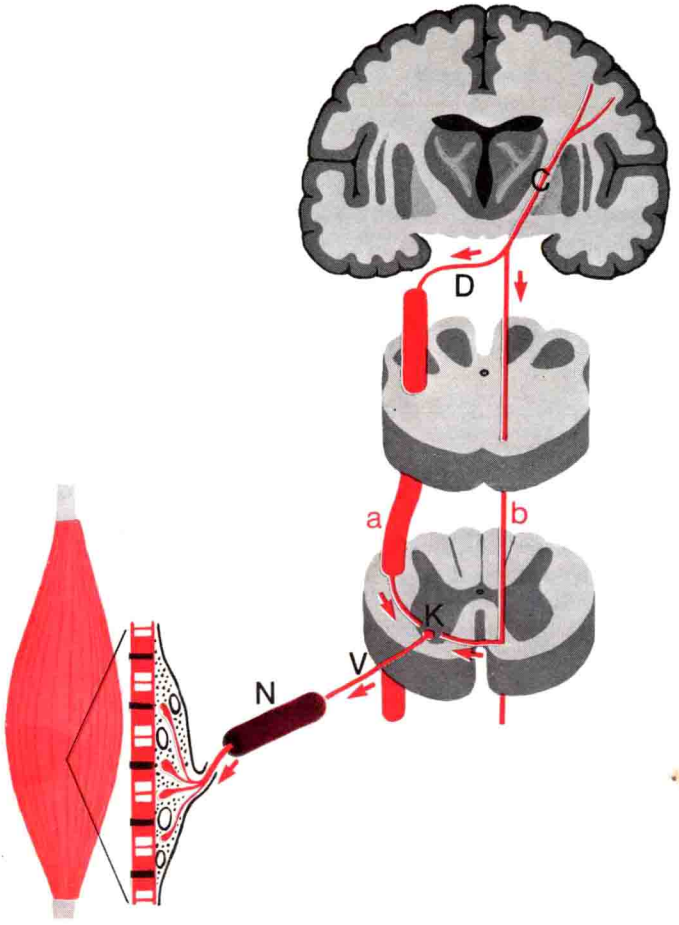


FIG. 3.—Fibers of the pyramidal tract arise in the Betz cells of Area 4 of the cerebral cortex. Most fibers cross and descend in the lateral corticospinal tract *(a)* (to supply the extremity muscles); some uncrossed fibers descend in the ipsilateral anterior corticospinal tract *(b)* (to supply the spinal muscles). Motor unit: the anterior horn cell *(K)* innervates the muscle fibers via the anterior nerve root *(V)*, peripheral nerve *(N)* and motor end-plate.

Irritative phenomena may be observed as visible muscular twitches (fasciculations), or with the aid of electromyography, as fibrillations. EMG signs always—and visible fasciculations frequently—are evidence of a lesion of the anterior horn cell or anterior nerve root. Continuous undulations (myokymia) are a less characteristic feature. Sudden contractions of the entire muscle (myoclonic movements) may emanate from a brain stem lesion; complex involuntary movements—often provoked by simple stimuli—are encountered in those diseases of the spinal cord in which pyramidal tract damage has resulted in spinal automatism, which substitutes for central control. In myotonic disorders (muscle membrane irritability due to decreased chloride conductance), percussion of a single muscle belly will result in a persistent contraction lasting for ten seconds or more.

Signs of motor deficit consist of muscle weakness (paresis) or total paralysis. Useful in follow-up evaluation is the grading of individual muscles according to strength: the range extends from 0 = paralysis to 5 = full strength even after prolonged physical activity. Classification of the paralysis depends on the location of the lesion: *myopathies* have a proximal distribution; lesions of the motor endplate (myasthenia) tend to affect the head, neck and bulbar muscles. Here, rapid exhaustion occurs in the presence of initial full strength. In diseases of the *peripheral nerves*, the pattern of involvement follows the anatomic relations; in *nerve root lesions*, the deficit involves a segment (myotome). Transverse spinal cord lesions can cause paraparesis/plegia if only the lower limbs are involved or tetraparesis/plegia if all four limbs are affected. The clinical picture will depend on the level of the lesion in the spinal cord, the former being encountered in thoracic, the latter in cervical cord lesions. Unilateral spinal cord damage causing weakness of one arm or one leg is referred to as monoparesis/plegia. In lesions of the *pyramidal tracts* (containing about 30,000 axis cylinders, with about one million extrapyramidal fibers) the resulting lesion is a hemiparesis/plegia. Different muscle groups may not be affected equally: the forearm, hand and finger extensors, hip flexors, dorsi and plantar flexors of the foot are often totally paralyzed; one speaks of a predilection paralysis (Wernicke-Mann). The more proximal muscles usually are spared.

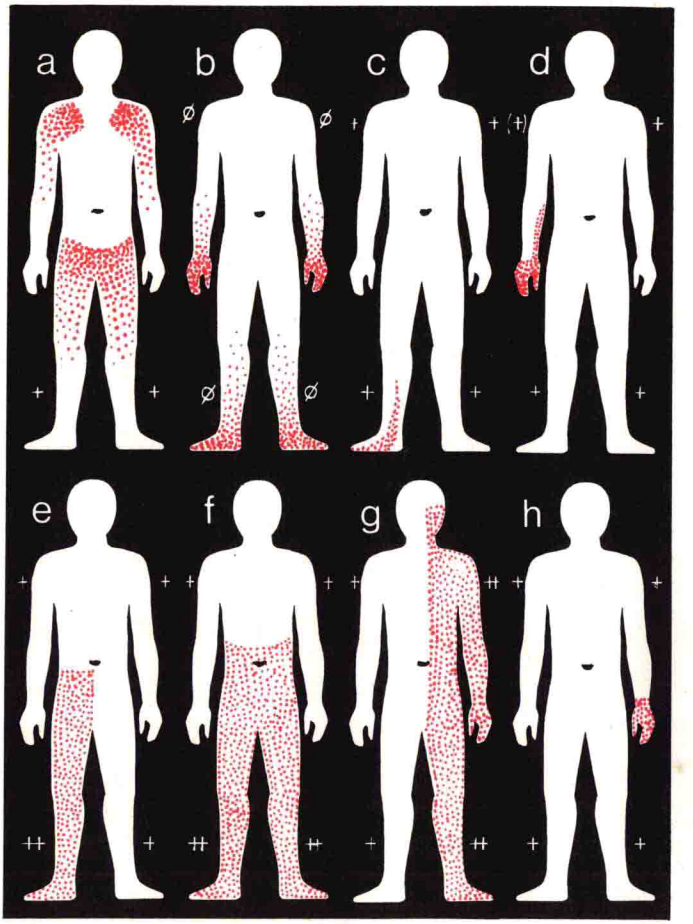


FIG. 4.—Depending on the site of the lesion, the following motor deficits may be differentiated: proximal myopathy (**a**); distal polyneuropathy (**b**); radiculopathy, e.g., L5 (**c**); plexopathy, e.g., lower brachial plexus (**d**); hemisection of the spinal cord (**e**); transverse myelopathy (**f**); contralateral cerebral lesion (**g**); and focal disturbance of the motor cortex (**h**). The degree of the motor deficit may vary from weakness (paresis) to complete paralysis (plegia). Tendon reflexes: absent = ∅, normal = +, increased = ++.

Classification into upper motor and lower motor neuron lesions is important. If only the *upper motor neuron* is involved, the anterior horn and reflex arc remain largely intact but a spastic increase in tone and pyramidal signs develop. If the *lower motor neuron* is damaged, trophic changes will occur in the motor unit of the appropriate segment. The extent and progress of *muscle atrophy* can be gauged by measuring the circumference of muscle bulk in the affected extremity and comparing it with that of the opposite limb. Electrical reactivity of the denervated muscles alters within 3–14 days *(reaction of degeneration)*. Faradic excitability disappears completely. *Rheobase*, as determined with a galvanic current pulse of one second, initially falls and then rises steeply (varies normally from individual to individual and muscle to muscle by 3–8 mA). *Chronaxy*, which is the duration of a pulse twice the rheobasic strength, may rise to levels of 100 msec or more (normal variation always under one msec). The higher the chronaxy the more complete the denervation of muscle fibers. Muscle reactivity also alters qualitatively: in place of the normal situation, i.e., cathodal closure contraction occurring ahead of anodal closure contraction, a reversal or other adjustment may occur. Muscle contractions are prolonged and finally become worm-like. In the most severe neurogenic or anterior horn damage, electrical reactivity ceases completely and the impulse spreads to the adjacent muscles. The prognosis for recovery then is poor. Electromyography reveals fibrillations or positive denervation potentials and a decreased interference pattern (see p. 66).

The motor nerve conduction velocity, which is determined by the latency difference of evoked action potentials between proximal and distal stimulation points on a nerve, is markedly decreased in some peripheral nerve lesions.

The signs of involvement of the lower motor neuron are discussed with the *anterior horn syndrome* (see p. 14).

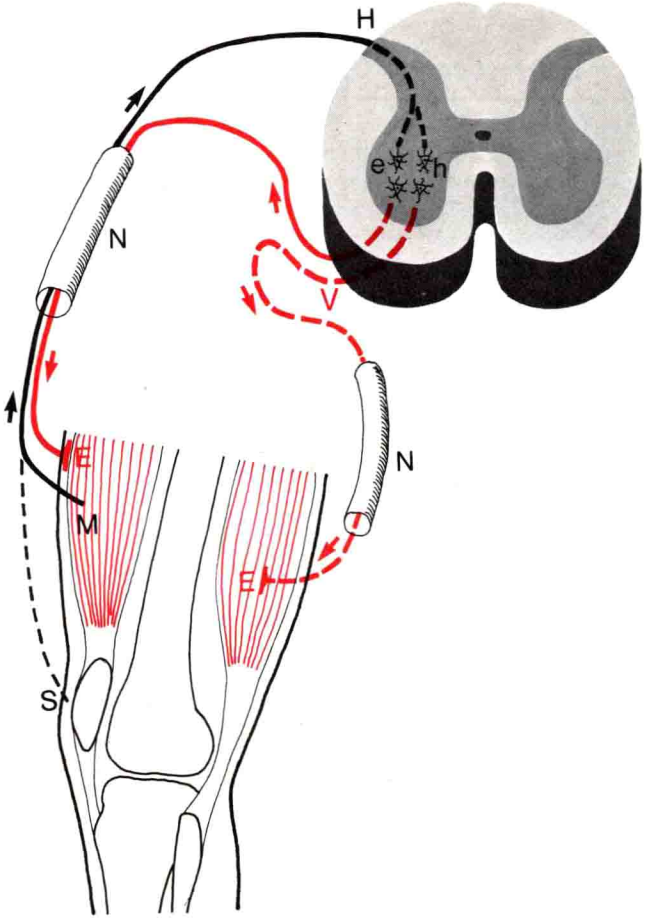
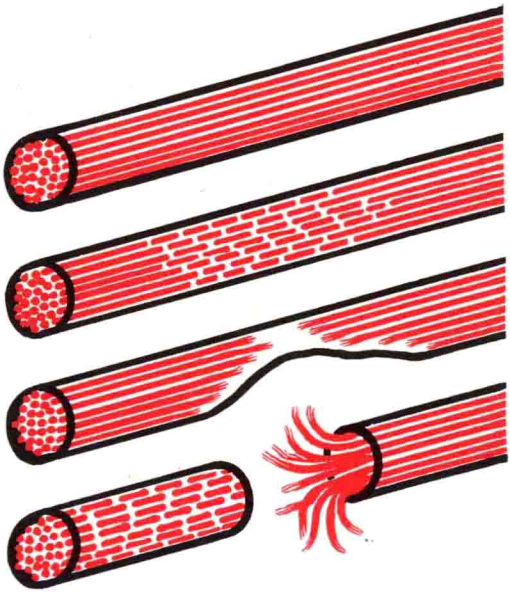


FIG. 5.—The stretch impulse from the muscle spindle *(M)* and tendon receptor *(S)* passes via the peripheral nerve *(N)*, plexus and posterior nerve root *(H)* to the spinal cord. There, it provokes a contraction of the appropriate muscles by stimulating anterior horn cells *(e)* and inhibiting the innervation of antagonist muscles. Invariably the muscle reflex arc is activated in more than one segment.

The reflex arc commences in the receptors (muscle spindles, tendons), passes via the peripheral nerve, plexus and posterior nerve root to the posterior horn and then synapses at the anterior horn cells. Here, the second neuron begins: it extends via the anterior nerve root, plexus, peripheral nerve and motor end-plates to the muscle fibers. In healthy subjects, prompt stretching of a muscle (e.g., tendon tapping) provokes a contraction in the muscle that varies in degree; it may be sluggish or lively. This tendon reflex always can be elicited equally on each side. Each muscle possesses a tendon reflex. The following tendon reflexes are easy to elicit and are tested routinely: triceps brachii, brachioradialis, quadriceps femoris (knee jerk) and triceps surae (ankle jerk). Less often, the semimembranosus, semitendinosus, the thigh adductor and tibialis posterior reflexes are tested.

In pyramidal tract injury, loss of inhibitory tone leads to increased reflexes *(hyperreflexia)*. If the reflex arc is damaged in any way, e.g., diseases of the first sensory or lower motor neuron, or of the spinal cord itself at the level of the reflex arc, the reflex is weakened or abolished *(hypo- or areflexia)*.

The same situation applies to muscle tone: in lesions of the upper motor neuron, tone is increased, usually with *spasticity*. Rapid movements prompt severe tonic contractions. If a lesion of the extrapyramidal system is present, *rigidity* may occur also. The increased tone appears as a continuous resistance ("lead-pipe rigidity") or is interrupted by a series of consecutive "gives" in the "cog-wheel rigidity"). If either the afferent or the efferent part of the reflex arc itself is interrupted at the appropriate spinal cord level, tone is diminished or abolished *(hypo- or atonia)*.



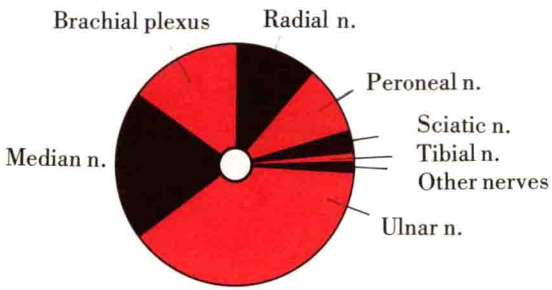


FIG. 6.—Diagrammatic representation of a normal nerve, neurapraxia, axonotmesis and neurotmesis. Circular diagram left: the incidence of peripheral nerve lesions (after Krenkel).