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Manual of Neurologic Therapeutics

Sixth Edition

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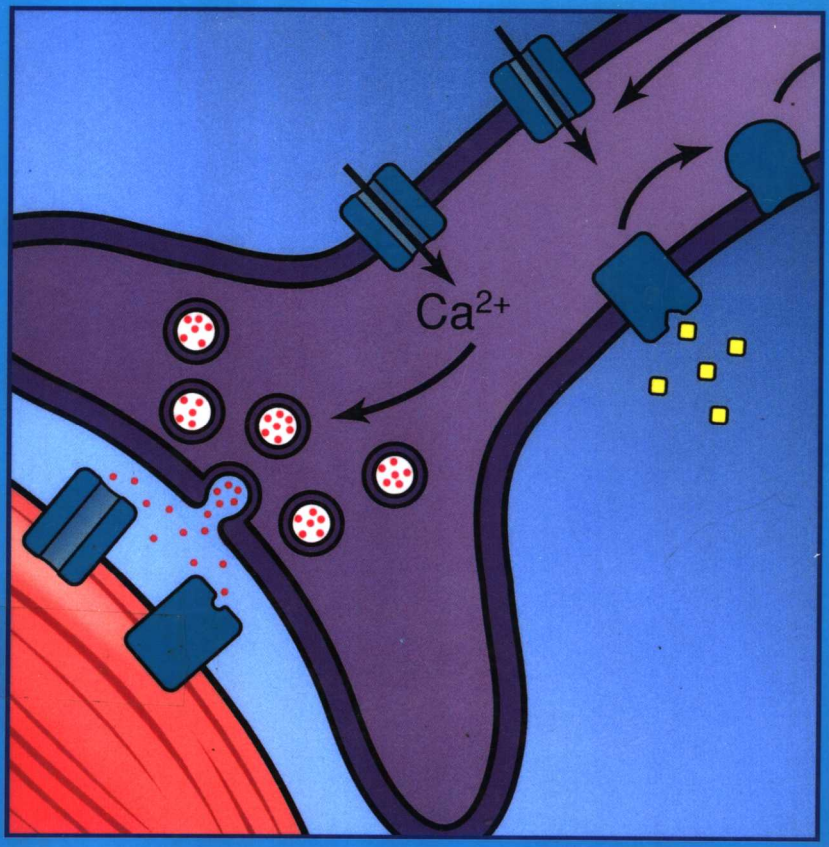
神经病治疗学手册

Edited by
Martin A. Samuels



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LIPPINCOTT
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SPIRAL®
MANUAL

(英文原版)

Manual of Nephrology

Fifth Edition

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肾病学手册

Edited by
Robert W. Schrier

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Manual of Neurologic Therapeutics

Sixth Edition

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PREFACE TO THE SIXTH EDITION

It has been twenty years since publication of the first edition of the *Manual of Neurologic Therapeutics.* At that time, the term neurologic therapeutics was considered an oxymoron, akin to airline food or military intelligence. I hatched the idea in 1974 while chief resident of internal medicine at the Boston City Hospital. Together with my Co-chief Resident Ralph Freidin and under the guidance of the chief of medicine, Alan B. Cohen, we had compiled a contributor volume called *Emergency Medicine: Diagnostic and Treatment Protocols* from Boston City Hospital. The book was published by Little, Brown, which had its medical division offices on Beacon Hill. On reentering my neurology training program at Massachusetts General Hospital (I had taken a year away to serve as medical chief resident), I called Jon Davidson, a medical editor at Little, Brown whom I had met through the *Emergency* book project. We met at the Bull & Finch Pub under the Hampshire House on Beacon Street (better known in subsequent years as Cheers) and I proposed a manual of neurologic therapeutics written entirely by me and my co-residents in neurology. It was to be completed during the leisurely second year of our training program, during which we spent most of our time in neuropathology. He agreed and gave me an opportunity that I had no right to expect, but for which I am eternally grateful.

I simply assigned two topics to each of my fellow residents. After months of agonizing editing and rewriting, mostly under the tutelage of our editor Diana Odell Potter, the book appeared in 1978, one generation ago. In the score of years that have intervened, the field of neurologic therapeutics has burgeoned. Annual popular courses in therapy are now offered at the major neurological societies. Several large books have been published and more are in production. The *Manual of Neurologic Therapeutics* has now sold over 100,000 copies in several languages around the world.

Since the last edition of the *Manual,* which was published in 1994, three new triptan drugs (naratriptan, rizatriptan, and zolmitriptan) have appeared, and an oral and nasal form of sumatriptan have been released for the abortive treatment of migraines. A new less toxic anticholinesterase medication (donepezil) is now available to slow the progression of Alzheimer's disease. Several new antiepileptic drugs (topiramate, lamotrigine, gabapentin, and tiagabine) have been added to the armamentarium for the treatment of intermittent seizures and fosphenytoin has appeared for the management of status epilepticus. New potent drugs are available for the treatment of HIV infection, dramatically altering the neurologic manifestations of the disease, and the first scientifically approved treatment for acute stroke (intravenous tissue plasminogen activator) is available for general use.

Brain tumor treatment is far more effective using new stereotactic radiosurgical approaches. Patients with Parkinson disease benefit from better, less toxic dopamine

agonists and several creative surgical approaches to the disease, including pallidotomy and various deep brain stimulation techniques. Advances in neuroscience and molecular genetics have led to better, earlier, and more reliable diagnostic tests along with more accurate genetic counseling for an ever-enlarging array of genetically determined neurological diseases, such as the hereditary sensory-motor neuropathies and the spino-cerebellar degenerations.

The goal of the *Manual of Neurologic Therapeutics* continues to be a clear, logical presentation of proven, evidence-based therapeutic approaches to neurological illnesses. Enough diagnostic information is included to aid the experienced physician in properly categorizing a clinical problem followed by a stepwise approach to treatment.

On the twentieth anniversary of the *Manual of Neurologic Therapeutics*, I thank my colleagues who came together in 1978 to begin the project and have stayed together through six editions. Their dedication to the success of this book has, in part, elevated neurologic therapeutics to the level of prominence that it now enjoys

Martin A. Samuels, M.D.
Boston, 1998

PREFACE TO THE FIRST EDITION

Until very recently the neurologist's primary task was to categorize and organize the structure and pathologic alterations of the nervous system. In fact, neurology has long been known as a discipline with elegantly precise and specific diagnostic capabilities but little or no therapeutic potentiality. Further, many surgeons, pediatricians, and internists have traditionally thought of the neurologist as an impractical intellectual who spends countless hours painstakingly localizing lesions while ignoring pragmatic considerations of treatment. Perhaps this conception is largely attributable to the peculiar complexity of the nervous system and the consequent relative naivete of physicians in their understanding of its functions.

Many of the classic descriptions of disease states in other medical disciplines were completed in the last century; in neurology, these have only been described in the past generation, and only in the last ten years has neurology begun to be characterized by subcellular mechanistic concepts of disease. This maturity has meant that the neurologist is now as much involved in the therapeutic aspects of his specialty of medicine as any of his colleagues. Certain neurologic diseases, such as epilepsy, have been treatable for relatively long periods of time, but understanding of the subcellular mechanisms of other diseases has led to newer, more effective forms of therapy.

An example of this is the enlarged understanding we now have of the biochemical alterations in Parkinson's disease, and the resultant therapeutic implications. Now, much as the endocrinologist treats diabetes with insulin and the cardiologist treats congestive heart failure with digitalis, the neurologist treats Parkinson's disease with L-dopa. In all these situations, the underlying condition is not cured; rather, an attempt is made to alter the pathophysiologic processes by utilizing a scientific understanding of the function of the diseased system.

This manual embodies a practical, logical approach to the treatment of neurologic problems, based on accurate diagnosis, that should prove useful to both clinician and student. No attempt is made to reiterate the details of the neurologic examination; it is assumed that the reader is competent to examine the patient—although particularly important or difficult differential diagnostic points are mentioned when appropriate. In this regard, it should be emphasized that this manual is only a guide to diagnosis and therapy, and each patient must be treated individually. The manual is organized to best meet the needs of the clinician facing therapeutic problems. Thus, the first seven chapters are concerned with symptoms, such as dizziness and headache, while the last ten consider common diseases, such as stroke and neoplasms.

I thank the many colleagues and friends whose criticism and comments were useful in the preparation of this book, in particular Drs. G. Robert DeLong, C. Miller Fisher, George Kleinman, James B. Lehrich, Steven W. Parker, Henry C. Powell,

E. P. Richardson, Jr., Maria Salam, Bagwan T. Shahani, Peter Weller, James G. Wepsic, and Robert R. Young. In addition I am indebted to Sara Nugent and Helen Hyland for their assistance in the preparation of the many manuscripts, and to Diana Odell Potter, formerly of Little, Brown and Company, for her editorial skills. Jane Sandiford, formerly of Little, Brown, and Kathleen O'Brien and Carmen Thomas of Little, Brown provided invaluable assistance in the final preparation of this material. Deep appreciation goes to Lin Richter, Editor in Chief of the Medical Division, Little, Brown and Company, for her support throughout this effort. I further thank Jon Paul Davidson, also formerly of Little, Brown, for his valuable encouragement and help early in the course of this project. Much support and encouragement was derived from my new colleagues in the Peter Bent Brigham Hospital Neurology Section, The Longwood Avenue Neurology Program, and the West Roxbury Veterans Administration Hospital. A great deal of inspiration came from the birth of my daughter Marilyn, and my deepest thanks go to my wife, Linda, who provided constant encouragement, editorial skill, and infinite patience.

M.A.S.

CONTENTS 目 录

I. NEUROLOGIC SYMPTOMS

1. COMA AND OTHER ALTERATIONS IN CONSCIOUSNESS

Telmo M. Aquino and Martin A. Samuels

I. General principles. When confronted with a patient with impaired consciousness, the clinician proceeds in an orderly, systematic manner. He or she gathers information while performing specific therapeutic maneuvers aimed at maintaining vital functions and avoiding further neurologic damage.

Impairment of consciousness may derive from a variety of causes. The first priority is to define and treat, as expeditiously as possible, those causes that are potentially reversible (see sec. **V**).

II. Pathophysiology

Consciousness consists of two components: **awareness** and **arousal** (or wakefulness). Awareness refers to the higher level integration of multiple sensory inputs that permit meaningful understanding of self and environment. The mechanisms of awareness reside diffusely in the cerebral cortex. **Arousal** refers to a more primitive set of responses, the structures for which are located entirely within the brainstem and diencephalon and that are synchronized by a diffuse network of nuclei and interconnecting tracts. The ascending reticular activating system (ARAS) mediates such responses as eye opening to painful stimuli, which is one clinical expression of intact arousal mechanisms. Other testable aspects of ARAS functioning include corneal reflexes, pupillary reactions, and ocular motility, either spontaneous or reflex (e.g., oculocephalic and vestibulo-ocular reflexes). Via thalamic relay nuclei, the ARAS projects diffusely to the cerebral cortex, acting thus as a "switch" for the cortical awareness system. In normal circumstances, it is the cycling of this system that accounts for the sleep-wake cycles and the corresponding EEG findings.

With these simple anatomic and physiologic points in mind, one can conceive of 3 mechanisms by which consciousness may be impaired:

A. Bilateral diffuse cerebral cortex failure, leading to a state of impaired awareness with intact arousal mechanisms (the so-called vegetative state). This circumstance most commonly results from a diffuse anoxic or ischemic insult such as cardiac arrest or the end stage of degenerative diseases.

B. Brainstem or thalamic failure, leading to a state of impaired arousal. In such cases, awareness is untestable, since the ARAS "switch" is shut off and would produce, in effect, a state of pathologic sleep. In clinical practice, a state of brainstem failure could be due to either:

1. **Primary brainstem pathology,** such as midbrain and/or diencephalic hemorrhage or infarction.
2. **Secondary brainstem injury** due to compression from masses that are normally situated in other compartments; examples of this are transtentorial (uncal) or cerebellar herniations due to a mass in the temporal lobe or the cerebellum, respectively. Such compressing masses can cause permanent brain lesions (e.g., Duret hemorrhages) by distorting the brainstem's vascular supply through stretch or torque.

C. Combined bilateral cortical and brainstem failure is seen most commonly in cases of metabolic encephalopathy and intoxications in which the relative participation of brainstem, as opposed to cortical, dysfunction varies, depending on the toxin involved and the type and severity of the metabolic derangement.

III. Diagnosis

A. History. Frequently, no information is available. Whenever possible, family, friends, ambulance personnel, and physicians who have previously treated the patient should be contacted. **Important features** of the history are **trauma, previous illnesses, medications, use of drugs or alcohol, and psychiatric disorders.**

B. General physical examination

1. **Vital signs:** airway patency, circulatory and ventilatory status, and fever.
2. **Skin:** signs of trauma, stigmata of liver disease, needle marks, and infective or embolic phenomena.
3. **Head:** Battle sign (i.e., hematoma over the mastoid process), localized tenderness, and crepitus and/or hemorrhage from ears or nostrils indicate basilar skull fracture.
4. **Neck stiffness** might be indicative of infection, trauma, or subarachnoid bleeding. (Do not manipulate the neck if there is suspicion of cervical spine fracture.)
5. **Chest, abdomen, heart, and extremities** must be examined routinely. Rectal and pelvic examinations plus a stool test for blood should also be performed.
6. **Breath** may suggest liver failure (fetor hepaticus, "liver breath"), ketoacidosis, alcohol ingestion, or uremia.

C. A neurologic examination is performed in all patients and recorded. Aiming to define presence, location, and nature of the causal process, special emphasis is placed on the following:

1. **Observation of the patient**
   a. If the patient **lies in a natural, comfortable position,** as though in natural sleep, coma is probably not very deep. Yawning and sneezing have the same significance, although other automatisms such as coughing, swallowing, or hiccuping do not necessarily reflect light coma.
   b. **Jaw and lid tone** also indicates the severity of unconsciousness. Open lids and hanging jaw bespeak deep coma.
2. **Level of unconsciousness.** Abnormalities of consciousness comprise a continuum, ranging from mild confusion to total unresponsiveness. It is useful in clinical practice to categorize patients with abnormal consciousness according to stages of progressive unresponsiveness. Given the imprecision surrounding the meanings of terms describing levels of consciousness, it is good practice to describe in detail on the record the responses of the patient to various stimuli. Use of terms such as **lethargy, somnolence,** or **obtundation** should be substantiated by a brief descriptive paragraph in the record.
   a. **Confusion** (or encephalopathy) is defined as the inability to maintain a coherent stream of thought or action. The neurologic substrate for confusion is inattention. **Attention** is difficult to define, but it refers to the ability of the individual to sort out and stratify the many sensory inputs and potential motor outputs so that a particular thought or action may be completed in an organized and logical fashion. It is evident from the concept that the mechanisms for attention must involve both arousal and awareness. Thus, confusion may be seen in states of cortical and/or ARAS dysfunction. The most common cause of confusion is metabolic or toxic derangement, although it may be seen with certain focal cortical lesions, particularly those of the right parietal lobe. Confusion is evident clinically when the apparently awake patient fails tasks requiring sustained attention, such as the serial 7's test, a go/no-go protocol, digit span, or spelling the word *world* backwards. Such patients may also show very disturbed writing with characteristic perseveration on letters with curved portions such as Os, Cs, and Ds.

      Delirium is a confusional state plus excess sympathetic activity. The term *delirium* applies when the confused patient also has tachycardia, diaphoresis, tremor, mydriasis, and hypertension. In general, pure confusion is seen in most metabolic encephalopathies including mild intoxication with sedative drugs, whereas delirium is caused by disorders that lead to increased levels of circulating catecholamines, such as intoxication with stimulant drugs (e.g.,

phencyclidine, amphetamines), high fever, and withdrawal from alcohol or sedative drugs (e.g., benzodiazepines, barbiturates).

b. **Drowsiness** is characterized by ready arousal, ability to respond verbally, and fending-off movements induced by verbal stimuli.

c. **Stupor** is characterized by incomplete arousal to noxious stimuli. There is no or little response to verbal commands. No verbal response or moaning is elicited. The motor responses are still of the purposeful, fending-off type.

d. **Light coma** is characterized by primitive and disorganized motor responses to noxious stimuli. There is no response to attempts at arousal.

e. **Deep coma** is characterized by absence of response to noxious stimuli.

f. When there is a question of **psychogenic unresponsiveness,** try to obtain a forced conscious response, e.g., by letting the patient's hand fall toward the face. Do not apply noxious stimuli to eyes, testicles, breasts, or other sensitive areas. Tickling the nostrils with cotton is usually sufficient for this purpose.

3. **Respiration.** The respiratory pattern is helpful in localizing and, in certain instances, determining the nature of the process.

a. **Cheyne-Stokes respiration** is characterized by periods of hyperventilation that gradually diminish to apnea of variable duration; breathing then resumes and gradually builds up again to hyperventilation. Cheyne-Stokes breathing indicates bilateral deep hemispheric and basal ganglionic dysfunction. The upper brainstem also may be involved.

Note: Cheyne-Stokes respiration is most commonly observed in nonneurologic conditions, such as congestive heart failure.

b. **Central neurogenic hyperventilation** refers to continuous rapid, regular, and deep respirations at a rate of about 25 per minute. It has no segmental localizing significance. Regularity is an unfavorable prognostic sign since increasing regularity correlates with increasing depth of coma.

Systemic acidosis (e.g., diabetic ketoacidosis, lactic acidosis) and hypoxemia should be excluded (normal pH, bicarbonate, and 2 partial pressure of oxygen [PO_2] determinations over 70 mm Hg in 24 hours is considered adequate for this purpose) before it is concluded that hyperventilation is of neurogenic origin.

c. **Apneustic breathing** consists of a prolonged inspiratory phase followed by apnea (the inspiratory cramp). It may be followed by cluster breathing, which consists of closely grouped respirations followed by apnea. Either pattern implies pontine damage.

d. **Ataxic breathing** and **gasping breathing** (Biot respirations) imply damage to the medullary respiratory centers. In ataxic breathing, respirations are chaotic. Gasping breathing is characterized by gasps followed by apnea of variable duration. Both are agonal events and usually precede respiratory arrest.

e. Depressed breathing consists of shallow, slow, and ineffective breathing caused by medullary depression, usually produced by drugs.

f. **Coma with hyperventilation** is seen frequently in metabolic disorders.

(1) **Metabolic acidosis** (e.g., diabetic ketoacidosis, uremia, ingestion of organic acids, lactic acidosis).

(2) ***Respiratory alkalosis*** *(e.g., hepatic encephalopathy).*

4. **Position of the head and eyes.** The normal cerebral hemisphere tends to move both head and eyes conjugately toward the opposite side. In hemispheric lesions, the healthy hemisphere becomes unopposed, deviating the head and eyes toward the lesion and away from the hemiparesis. The reverse occurs in pontine lesions, in which the eyes deviate toward the hemiparesis and away from the lesion.

5. **Visual fields and funduscopy**
   a. In patients who are not completely unresponsive, visual fields should be tested with threatening movements, which normally evoke a blink. Asymmetry of the blink response suggests hemianopia (in the absence of blindness or optic nerve damage). Air movement in the eyes can produce a false-positive response.
   b. Funduscopy may reveal papilledema suggestive of increased intracranial pressure. A **subhyaloid hemorrhage**—a rounded, well-defined clot on the retinal surface—is commonly associated with subarachnoid hemorrhage.
6. **Pupils.** Note size, roundness, and equality to light reaction, both directly and consensually.
   a. **Midposition (3–5 mm)** nonreactive pupils are evidence of midbrain damage.
   b. **Reactive pupils** indicate midbrain intactness. In the presence of unresponsiveness and absent extraocular movements and corneal reflexes, reactive pupils suggest metabolic abnormality (e.g., hypoglycemia) or drug ingestion (e.g., barbiturate).
   c. **A unilaterally dilated and unreactive pupil** in a comatose patient (Hutchinson pupil) may be a sign of third nerve compression due to temporal lobe herniation. Other components of third nerve dysfunction (e.g., drooping of the eyelid and abduction of the eye as the result of unopposed action of the lateral rectus muscle) may occur concomitantly or follow pupillary dilatation. Less frequently, direct or compressive midbrain damage is expressed by a dilated, nonreactive pupil.

      Note: A fixed and/or dilated pupil in an alert patient is not a sign of brain herniation. This finding may be due to essential anisocoria, old injury to the iris, migraine, a posterior communicating artery aneurysm, Adie syndrome, or a mydriatic agent that was inadvertently or purposefully instilled in the eye. One or 2 drops of 1% pilocarpine (a parasympathetic agonist) instilled into the eye will result in miosis in patients in whom the oculomotor nerve is compressed (e.g., aneurysm) but will fail to do so if parasympathetic receptors in the iris are occupied by a mydriatic drug (e.g., scopolamine).
   d. **Small but reactive pupils** signify pontine damage, as in infarction or hemorrhage. Opiates and pilocarpine also produce pinpoint reactive pupils. A magnifying glass may be necessary to appreciate the pupillary reaction.
   e. **Dilatation** of the pupils in response to a painful stimulus in the neck (the normal ciliospinal reflex) indicates lower brainstem integrity.
7. **Extraocular movements.** If the patient is responsive enough to follow commands, saccadic and pursuit eye movements should be tested. A large number of ocular and gaze palsies may be present. In unresponsive patients, a great deal of information may be obtained by testing the vestibulo-ocular reflex (VOR), which is mediated by pathways that traverse the brainstem from the vestibular nuclei in the medulla to the oculomotor nuclei in the midbrain. **The most useful tests of the VOR** are as follows:
   a. **Doll's-head maneuver, passive head turning, or oculocephalic reflex (do not perform this maneuver when there is a question of cervical spine injury)** is performed by turning the patient's head with quick lateral and vertical displacements. In unconscious patients, **the reflex is normal** or **preserved** if the eyes move in the orbits in the direction opposite to the rotating head, maintaining their position in relation to the environment.