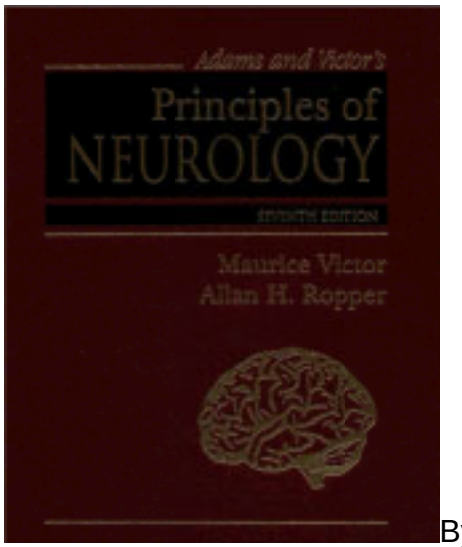
Adams & Victor's Principles Of Neurology 7th edition (December 19, 2000): by Maurice Victor



By OkDoKeY

Adams & Victor's Principles o

CONTENTS

PART 5 DISEASES OF SPINAL CORD, PERIPHERAL NERVE, AND MUSCLE

PART 6 PSYCHIATRIC DISORDERS

EDITORS

Profes

NOTICE

Medicine is an ever-changing science. As new research and clinical experience broaden our kno
authors and the publisher of this work have checked with sources believed to be reliable in their e
accord with the standards accepted at the time of publication. However, in view of the possibility
authors nor the publisher nor any other party who has been involved in the preparation or publica
in every respect accurate or complete, and they disclaim all responsibility for any errors or omissi
contained in this work. Readers are encouraged to confirm the information contained herein with
advised to check the product information sheet included in the package of each drug they plan to
work is accurate and that changes have not been made in the recommended dose or in the contr
particular importance in connection with new or infrequently used drugs.

PREFACE

In the first edition of the *Principles of Neurology*, we remarked that the preface to a textbook i
adding to the book's weight or distracting critics from its contents. The value of the book shou
foreword to *Cromwell*, Victor Hugo expressed this sentiment more figuratively: one seldom ins
examines the roots of a tree after eating its fruit.

Yet there has to be a place where authors can state the purpose of their work, the manner in
another book on a medical public already overburdened with an immense literature. To sustai
inspecting the cellar of a house, one is not sorry to have done so, especially if one is to purch

In writing the *Principles of Neurology*, we adopted a method that had for long been espoused
Hospital. Instead of the customary recitation of many diseases of the nervous system, we cho
phenomenology, or cardinal manifestations, of neurologic disease. Thus the first part of this b
signs of disordered nervous function, their analysis in terms of anatomy and physiology, and t
the natural clusterings of these phenomena, or syndromes, which are the lore of clinical neuro
categories and types of disease that express themselves by each syndrome. We believe this
practice the patient presents with symptoms of disordered nervous function, from which the cl
syndrome to disease recapitulates the rational process by which this is achieved. In teaching
be eminently successful.

The compass of our book differs in several other ways from most contemporary textbooks of
subjects that form the core of pediatric neurology, which is heavily weighted with developmen
presented in the context of normal development and maturation of the nervous system. And t
neurology) have been accorded a separate chapter. No distinction is drawn between neurosu
with reference to mode of therapy. A significant portion of the book has been allotted to psych
has been done in the belief that these diseases are neurologic in the strict sense. Further, it is
be knowledgeable about the diagnosis of depressive states, neuroses, and eccentricities of p
medical illnesses. The neuropsychiatric effects of alcoholism and drug abuse are also include
description of muscle diseases, which increasingly are coming under the purview of neurologi
only to the practice of neurology and neurosurgery, but also to the practice of internal medicin
various specialties in finding relevant material, we suggest they turn their attention to the follo
pediatricians: 16, 28, 37, 38; emergency and intensive care physicians: 16, 17, 18, 34, 35; ort
57, 58.

Throughout this text the emphasis is on the clinical aspects of disease. Of course, pertinent n
presented with the view of how they bear upon and explain neurologic phenomena and disea
tradition, is to present the clinical phenomena that we ourselves have observed. We persist in
enables the authors to select what they believe to be essential knowledge about common dis
assures an evenness and uniformity of style that is more likely to please the reader.

The warm reception accorded the first six editions of *Principles of Neurology* has led us to be
emboldened us to carry this work forward. During the editing of the seventh edition, each of u
and developments in clinical neurology and has endeavored to incorporate them. Every chap
of molecular genetics have been added where relevant. The clarification of physiologic functio
has been expanded. Considerably greater use has been made of tables and MRI illustrations
has been made to provide the most detailed and current information about the treatment of ne

It is hardly possible to enumerate and adequately thank our many colleagues who in one way
growth of this textbook. Foremost is our indebtedness to colleagues and teachers who had an
Denny-Brown, C. M. Fisher, Paul Yakovlev, E. P. Richardson, and Mandel Cohen. A special c
chapters on muscle disease and for her help on all matters, large and small, pertaining to this
colleagues with whom we have repeatedly discussed the substantive material of previous edi

Chapter 1

THE CLINICAL METHOD OF NEUROLOGY

Neurology is regarded by many as the most difficult and exacting medical specialty. Students and r
easily discouraged by what they see. Having had brief contact with neuroanatomy, neurophysiology
the complexity of the nervous system. The ritual they then witness of putting the patient through a s
the names of which are difficult to pronounce, is hardly reassuring; in fact, the procedure often appe
diagnosis is attained. Moreover, the students have had little or no experience with the many specia
electroencephalographic, angiographic, and imaging procedures—nor do they know how to interpre
fears as they read the detailed accounts of the many rare diseases of the nervous system.

The authors believe that many of the difficulties in comprehending neurology can be overcome by a
foremost, it is necessary to learn and acquire facility in the use of the *clinical method*. Without a ful
with a new clinical problem as a botanist or chemist who would undertake a research problem witho

The importance of the clinical method stands out more clearly in the study of neurologic disease tha
method will prove to consist of an orderly series of steps, as follows:

1. The symptoms and signs are secured by history and physical examination, respectively.
2. The symptoms and physical signs considered relevant to the problem at hand are interpreted
   disorder(s) of function and the anatomic structure(s) implicated by such a disorder. Often one
   constituting a *syndrome*. The formulation of symptoms and signs in syndromic terms is partic
   This step may be called *syndromic diagnosis*.
3. These correlations permit the physician to localize the disease process, i.e., to name the part
   anatomic, or *topographic, diagnosis*.
4. From the anatomic diagnosis and other medical data—particularly the mode of onset, evolutio
   organ systems, the relevant past and family histories, and the laboratory findings—one deduc
   causation of the disease can be determined, the *etiologic diagnosis*.
5. Finally, the physician should assess the degree of disability and determine whether it is tempo
   managing the patient's illness and judging the potential for restoration of function, i.e., progno

The foregoing approach to the diagnosis of neurologic disease is summarized in Fig. 1-1, a proced
sequential finite steps. This systematic approach, allowing the confident localization and often prec

Different disease processes may cause identical symptoms, which is understandable in view of the
any one of several diseases. For example, a spastic paraplegia may result from spinal cord tumor,
may present with different groups of symptoms and signs. However, despite the many possible cor
combinations occur with greater frequency than others and can be recognized as highly characteris
attempting to categorize every case in terms of a characteristic symptom complex, or *syndrome*. O
entities but rather abstractions set up by clinicians in order to facilitate the diagnosis of disease. Fo
to write, calculate, and identify individual fingers constitutes the so-called Gerstmann syndrome, re
(region of the left angular gyrus) and at the same time narrows the range of possible etiologic facto

In the initial analysis of a neurologic disorder, anatomic diagnosis takes precedence over etiologic d
without first ascertaining the parts or structures that are affected would be analogous in internal me
whether the disease involved the lungs, stomach, or kidneys. Discerning the cause of a clinical syn
different order. Here one must be conversant with the clinical details, including the mode of onset, c
of these facts are well known and not difficult to master; they form the substance of later chapters.

TAKING THE HISTORY

In neurology more than any other specialty, the physician is dependent upon the cooperation of the
symptoms that are unaccompanied by observable signs of disease. And if the symptoms are in the
feels. The first step in the clinical encounter is to enlist the patient's trust and cooperation and make
following points about taking the neurologic history deserve further comment:

1. Special care must be taken to avoid suggesting to the patient the symptoms that one seeks. T
of the examiner has a great influence on the patient. Repetition of this truism may seem tedio
leading questions that either suggested symptoms to the patient or led to a distortion of the pa
as often the fault of the physician as of the patient. As a corollary, the patient should be disco
may have heard, but should be urged to give as accurate a description of the symptom as pos
describes the quality of his pain.
2. The practice of making notes at the bedside or in the office is particularly recommended. The
can be kept on the subject of his illness by discrete questions that draw out essential points. I
course, no matter how reliable the history appears to be, verification of the patient's account b
3. The setting in which the illness occurred, its mode of onset and evolution, and its course are c
each symptom began and progressed. Often the nature of the disease process can be decide
the patient or his family, it may be necessary to judge the course of the illness by what the pa
when he could no longer negotiate stairs or carry on his usual work) or by changes in the clini
physician had recorded the findings accurately and quantitated them in some way.
4. Since neurologic diseases often impair mental function, it is necessary, in every patient who r
initial assessment of the mental status and the circumstances under which symptoms occurre
illness. If the patient's power of attention, memory, and coherence of thinking are inadequate,
Also, illnesses that are characterized by seizures or other forms of episodic confusion abolish
episodes. In general, students (and some physicians as well) tend to be careless in estimatin
made to take histories from patients who are feebleminded or so confused that they have no
who for other reasons could not possibly have been aware of the details of their illnesses.

THE NEUROLOGIC EXAMINATION

The neurologic examination begins with observation of the patient while the history is being obtaine
betray confusion or incoherence in thinking, impairment of memory or judgment, or difficulty in com
integral part of the examination and provides information as to the adequacy of cerebral function. T
embarrassment to the patient. A common error is to pass lightly over inconsistencies in history and
these flaws in memory were the essential features of the illness. Asking the patient to give his own
expose unnatural concern, anxiety, suspiciousness, or even delusional thinking.

If there is any suggestion of a speech or language disorder, the nature of the patient's spontaneous
spell; execute spoken commands; repeat words and phrases spoken by the examiner; name objec
should be assessed. Bisecting a line, drawing a clock or the floor plan of one's home or a map of o
perception in cases of suspected cerebral disease. The testing of language, cognition, and other as
Chap. 22, and Chap. 23.

Testing of Cranial Nerves

The function of the cranial nerves must be investigated more fully in patients who have neurologic s
anterior fossa, the sense of smell should be tested in each nostril, and then it should be determined
outlined by confrontation testing, in some cases by testing each eye separately; if any abnormality
sought on the tangent screen or, more accurately, by computed perimetry. Pupil size and reactivity
rate of ocular movements, and the presence or absence of nystagmus should next be observed. D
in Chap. 12, Chap. 13, and Chap. 14.

Sensation over the face is tested with a pin and wisp of cotton; also, the presence or absence of th
be observed as the patient speaks and smiles, for a slight weakness may be more evident in these
meati and tympanic membranes should be inspected with an otoscope. A 256 double-vibration tuni
loss and distinguishes middle ear (conductive) from neural deafness. The vocal cords need to be in
or vagus nerve disease, especially when there is hoarseness. Pharyngeal reflexes are meaningful
reflexes is seldom significant. Inspection of the tongue, when protruded and at rest, is helpful; atrop
deviation of the protruded tongue as a solitary finding can usually be disregarded. Any abnormality
buccal, and sucking reflexes should be sought, particularly if there is a question of dysphagia, dysa

Tests of Motor Function

In the assessment of motor function, it should be kept in mind that observations of the speed and s
are usually more informative than the tendon reflexes. It is essential to have the limbs fully exposed
watch the patient maintain the arms outstretched in the prone and supine positions; perform simple
finger; make rapid alternating movements, particularly such movements that necessitate sudden ac
the thumb to each fingertip and supinate and pronate the forearm; and accomplish simple tasks su
tools. Estimates of the strength of leg muscles with the patient in bed are often unreliable; there ma
step up on a footstool without help or arise from a kneeling position. Running the heel down the fro
only tests of coordination that need be carried out in bed. The maintenance of the outstretched and
tiring first, soon begins to sag, or, in the case of a corticospinal lesion, to resume the more natural p
either with the patient supine and the legs flexed at hips and knees, or with the patient prone and th
tremors may be exposed (see Chap. 4 and Chap. 6).

Tests of Reflex Function

Testing of the biceps, triceps, supinator (radial-periosteal), patellar, Achilles, and cutaneous abdom
activity of the spinal cord. Elicitation of tendon reflexes requires that the involved muscles be relaxe
voluntary contraction of other muscles (Jendrassik maneuver). The interpretation of the plantar resp
responses can be evoked by stimulating the sole of the foot along its outer border from heel to toes
response; (2) the slower, spinal flexor noci-fensor reflex (flexion of knee and hip and dorsiflexion of
the well-known Babinski sign; and (3) the plantar grasp reflex. Avoidance and withdrawal response
sometimes be overcome by utilizing the several alternative stimuli that are known to elicit the Babin
fourth toe, downward scraping of the shin, lifting the straight leg, etc.). An absence of the superficia
muscles is a useful ancillary test for detecting corticospinal lesions.

Testing of Sensory Function

This is undoubtedly the most difficult part of the neurologic examination. Usually sensory testing is

Accurate recording of negative data may be useful in relation to some future illness that requires ex

THE COMATOSE PATIENT

Although subject to obvious limitations, careful examination of the stuporous or comatose patient yi
nervous system. The demonstration of signs of focal cerebral or brainstem disease or of meningea
diseases that cause stupor and coma. The adaptation of the neurologic examination to the comatos

THE PSYCHIATRIC PATIENT

One is compelled in the examination of psychiatric patients to rely less on the cooperation of the pa
The depressed patient, for example, may claim to have impaired memory or weakness when actua
sociopath or hysteric may feign paralysis. The opposite is sometimes true—a psychotic patient may
them ignored because of his mental state.

If the patient will speak and cooperate even to a slight degree, much may be learned about the func
can, in nearly every instance, be recognized by the manner in which the patient expresses ideas, re
it is possible to determine whether there are hallucinations or delusions, defective memory, or othe
listening to the patient. Ocular movements and visual fields can be tested with fair accuracy by obs
four quadrants of the fields. Cranial nerve, motor, and reflex functions are tested in the usual mann
never complete unless the patient will speak and cooperate in testing. On occasion, mute and resis
widespread cerebral disease such as hypoxic or hypoglycemic encephalopathy, a brain tumor, a va

INFANTS AND SMALL CHILDREN

The reader is referred to the methods of examination described by Gesell and Amatruda, Dubowitz
listed in the references and described in Chap. 28.

THE GENERAL MEDICAL EXAMINATION

Never to be overlooked in the assessment of a neurologic problem are the findings on general mec
systemic disease that has secondarily affected the nervous system. In fact, many of the most serio
will suffice: the finding of adenopathy or a lung infiltrate implicates neoplasia or sarcoidosis as the c
fever, anemia, a heart murmur, and splenomegaly in a case of unexplained stroke points to a diagn
brain arteries. Certainly no examination of a patient with stroke is complete without a search for hyp

IMPORTANCE OF A WORKING KNOWLEDGE OF NEUROANATOMY, NEUROPH

Once the technique of obtaining reliable clinical data is mastered, students and residents may find t
lack of knowledge of neuroanatomy and neurophysiology. For this reason, each of the later chapter
consciousness, language, etc., is introduced by a review of the anatomic and physiologic facts that

At a minimum, physicians should know the anatomy of the corticospinal tract; the motor unit (anteri
cerebellar motor connections; the sensory pathways; the cranial nerves; the hypothalamus and pitu
system; the functional areas of the cerebral cortex and their major connections; the visual, auditory
working knowledge of neurophysiology should include an understanding of the nerve impulse, neur
spinal reflex activity; central neurotransmission; the processes of neuronal excitation, inhibition, and

From a practical diagnostic and therapeutic point of view, the neurologist is helped most by a know
disease processes such as infarction, hemorrhage, demyelination, physical trauma, compression, i
ones. Experience with the gross and microscopic appearances of these disease processes greatly

the dimension of histopathology to the clinical study.

As pointed out by Chimowitz, students tend to err in failing to recognize a disease they have not se
of a common disease. There is no doubt that some clinicians are more adept than others at solving
is presumed, but is attributable to having paid close attention to the details of their experience with
The unusual case is recorded in memory and can be resurrected when another one like it is encou
accept the obvious explanation.

THE PURPOSE OF THE CLINICAL METHOD OF NEUROLOGY

Accurate diagnosis serves four main purposes: (1) it enables the physician to determine the proper
predicting the outcome of the illness; (3) if the disease is hereditary, it allows for genetic counseling
clinical phenomena and disease. The medical profession is primarily concerned with the prevention
well-defined end. A major aim of the neurologist, therefore, is not to overlook a disease for which th
given syndrome must be carefully considered and excluded by clinical and laboratory methods. For
one must take special care to exclude the presence of a tumor, subacute combined degeneration (
hematoma, herniated disc, and cervical spondylosis, for these are all treatable. In this respect, failu

THERAPEUTICS IN NEUROLOGY

Among medical specialties, neurology has traditionally occupied a somewhat anomalous position, b
concerned with making diagnoses of untreatable diseases. This disdainful view of our profession is
medical and others surgical, for which specific therapy is now available, and—through advances in
pertaining to these therapies and to the dosages, timing, and manner of administration of particular
description of individual diseases.

There are, in addition, many diseases in which neurologic function can be restored to a varying deg
of therapeutic agents that have not been fully validated. Claims for the effectiveness of a particular
studies, must be treated circumspectly. Was the study well conceived, was there rigid adherence to
study, were the statistical methods standardized, were the controls truly comparable? And most imp
to the clinical problem at hand? Also, it has been our experience, based on participation in a numbe
accepted with caution. Since newly proposed therapeutic agents are often risky and expensive, it is
have been claimed for them or expose flaws in the design or fundamental assumptions of the origin

Even when no treatment is possible, neurologic diagnosis is far more than an intellectual pastime. T
identification in the living patient. Until this is achieved, it is impossible to apply adequately the “mas
neurology thus serves both the physician in the practical matters of diagnosis, prognosis, and treatm
cause of the disease.

---

*Throughout this text we follow the traditional English practice of using the pronoun *he, his,* or *him* in the generic sense

CHAPTER REFERENCES

BERG BO (ed): *Principles of Child Neurology*. New York, McGraw-Hill, 1996, pp 5–22.

BICKERSTAFF ER, SPILLANE JA: *Neurological Examination in Clinical Practice*, 5th ed. Oxford, Blackwell Scientific, 1

CHIMOWITZ MI, LOGIGIAN EL, CAPLAN LP: The accuracy of bedside neurological diagnoses. *Ann Neurol* 28:78, 19

DEMYER WE: *Technique of the Neurologic Examination: A Pro-grammed Text*, 4th ed. New York, McGraw-Hill, 1994.

DUBOWITZ LM, DUBOWITZ V, MERCURI E: *The Neurological Assessment of the Preterm and Full-term Newborn In*

GESELL A, AMATRUDA CS: in Knoblock H, Pasamanick B (eds): *Gesell and Amatruda's Developmental Diagnosis*, 3

Chapter 2

SPECIAL TECHNIQUES FOR NEUROLOGIC DIAGNOSIS

The analysis and interpretation of data elicited by a careful history and examination may prove to b
no more than corroborate the clinical impression. However, it happens more often that the nature o
possibilities may be reduced to two or three, but the correct one is uncertain. Under these circumst
The aim of the neurologist is to arrive at a final diagnosis by artful analysis of the clinical data aidec
strategy of laboratory study of disease should be based purely on therapeutic and prognostic consi
exigencies.

A few decades ago the only laboratory procedures available to the neurologist were lumbar punctu
the skull and spinal column, contrast myelography, pneumoencephalography, and electroencephal
technology, the physician's armamentarium has been expanded to include a multitude of neuroima
methods are so impressive that there is a temptation to substitute them for a careful, detailed histor
should be avoided; it certainly does not guarantee a diagnosis. In fact, in a carefully examined serie
findings [including magnetic resonance imaging (MRI)] clarified the clinical diagnosis in 40 patients
The physician should always keep in mind the primacy of the clinical method and judge the relevan
clinical findings. Hence the neurologist must be familiar with all laboratory procedures relevant to n

Below is a description of laboratory procedures that have application to a diversity of neurologic dis
complex or category of disease—e.g., audiogram (deafness); electronystagmogram, or ENG (vertig
(neuromuscular disease)—are presented in the chapters devoted to these disorders.

LUMBAR PUNCTURE AND EXAMINATION OF CEREBROSPINAL FLUID

pressure and the tugging of cerebral and dural vessels as the patient assumes the erect posture. T
lumbar puncture are considered further in Chap. 30. Bleeding into the spinal meningeal spaces ca
>13.5 or an International Normalized Ratio (INR) >1.3], have low platelet counts (<30,000 to 50,00
Purulent meningitis and disc space infections have been known to complicate lumbar puncture, the
particulate matter (e.g., talc) can produce a sterile meningitis. Diplopia, facial paresis, and tinnitus
rare occasions.

Examination Procedures

Once the subarachnoid space has been entered, the pressure and—in special cases—"dynamics"
obtained. The gross appearance of the fluid is noted, after which the CSF, in separate tubes, can b
micro-organisms; (2) protein and glucose content; (3) tumor cells, using a Millipore filter or similar t
and presence of oligoclonal bands; (5) pigments, lactate, NH_3, pH, CO_2, enzymes, and substances
cryptococcal antigen, mycobacteria, herpesvirus and cytomegalovirus DNA (by polymerase chain r

Pressure and Dynamics With the patient in the lateral decubitus position, the CSF pressure is me
space. In the normal adult, the opening pressure varies from 100 to 180 mmH_2O, or 8 to 14 mmHg
pressure above 200 mmH_2O with the patient relaxed and legs straightened reflects the presence of
mmH_2O or below indicates intracranial hypotension, generally due to leakage of spinal fluid or syste
sac and the patient in a sitting position, the fluid in the manometer rises to the level of the cisterna
recumbent position). It fails to reach the level of the ventricles because the latter are in a closed sy
manometer is influenced by atmospheric pressure. Normally, with the needle properly placed in the
a few millimeters in response to the pulse and respiration and rises promptly with coughing, straini

The presence of a spinal subarachnoid block can be confirmed by jugular compression. First one s
simultaneously, with enough pressure to compress the veins but not the carotid arteries (Queckens
rise in pressure of 100 to 200 mmH_2O and a return to its original level within 10 s after release. Fai
the needle is improperly placed. A rise in pressure in response to abdominal compression (or coug
subarachnoid block. Failure of the pressure to rise with compression of one jugular vein but not the
These tests are now rarely used, having been replaced by more precise and less hazardous imagi
Jugular compression should never be performed when an intracranial tumor or other mass lesion is

Gross Appearance and Pigments Normally the CSF is clear and colorless, like water. Minor degr
and water against a white background (by daylight rather than fluorescent illumination) or by lookin
imparts a hazy or ground-glass appearance; at least 200 red blood cells (RBC) per cubic millimeter
1000 to 6000 RBC per cubic millimeter imparts a hazy pink to red color, depending on the amount
sedimentation of the RBC. Several hundred or more white blood cells in the fluid (pleocytosis) may

A traumatic tap (in which blood from the epidural venous plexus has been introduced into the spina
interpreted to indicate a pre-existent subarachnoid hemorrhage. To distinguish between these two
taken at the time of the LP. With a traumatic tap, there is usually a decreasing number of RBC in th
pressure is usually normal, and if a large amount of blood is mixed with the fluid, it will clot or form
because the blood has been greatly diluted with CSF and defibrinated. With subarachnoid hemorrh
pink-red discoloration (erythrochromia) to the supernatant fluid; allowed to stand for a day or more,
centrifugation of bloody fluid from a traumatic tap will yield a colorless supernatant; only with large
fluid be faintly xanthochromic due to contamination with serum bilirubin and lipochromes.

The fluid from a traumatic tap should contain one or two white blood cells (WBC) per 1000 RBC as
widely. With subarachnoid hemorrhage, the proportion of WBC rises as RBC hemolyze, sometimes
vagaries of this reaction are such that it, too, cannot be relied upon to distinguish traumatic from pr
which occurs in both types of bleeding.

The reason that red corpuscles undergo rapid hemolysis in the CSF is not clear. It is surely not due

are normal, the CSF protein should increase by about 1 mg per 1000 RBC provided that the same
content. Because of the irritating effect of hemolyzed RBC upon the lepto meninges, the CSF prote

The protein content of the CSF in bacterial menin-gitis, in which choroidal and meningeal perfusion
induce a less intense and mainly lymphocytic reaction and a lesser elevation of protein—usually 50
protein content is normal. Paraventricular tumors, by reducing the blood-CSF barrier, often raise th
mg/dL or even higher are found in exceptional cases of the Guillain-Barré syndrome and chronic in
or more usually indicate loculation of the lumbar CSF (CSF block); the fluid is then deeply yellow ar
combination of CSF abnormalities is called the *Froin syndrome*. Partial CSF blocks by ruptured dis
protein values are sometimes found in meningismus (a febrile illness with signs of meningeal irritati
(Chap. 30), in hyperthyroidism, or after a recent lumbar puncture.

The quantitative partition of CSF proteins by electrophoretic and immunochemical methods demons
weight of less than 150,000 to 200,000. The protein fractions that have been identified electrophore
beta$_2$, and gamma globulin fraction, the last of these being accounted for mainly by immunoglobulin
values of the different fractions are given in Table 2-1. Immunoelectrophoretic methods have also d
ceruloplasmin, transferrin, and hemopexin. Large molecules—such as fibrinogen, IgM, and lipoprot

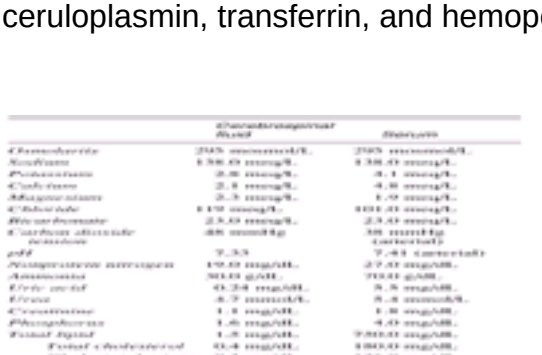


Table 2-1 Average values of constituents of normal CSF and serum

There are other notable differences between the protein fractions of CSF and plasma. The CSF alw
Although derived from plasma, this fraction, for an unknown reason, concentrates in the CSF, and t
because of its concentration by choroidal cells). Also, the CSF beta $_2$ or tau fraction (transferrin) is p
ventricular than in the spinal fluid. The gamma globulin fraction in CSF is about 70 percent of that in

At present only a few of these proteins are known to be associated with specific diseases of the ne
percent of the total CSF protein in diseases such as multiple sclerosis, neurosyphilis, subacute scle
meningoencephalitides. The serum IgG is not correspondingly increased, which means that this im
elevation of serum gamma globulin—as occurs in cirrhosis, sarcoidosis, myxedema, and multiple m
Therefore, in patients with an elevated CSF gamma globulin, it is necessary to determine the electr
changes in the CSF immunoglobulin pattern, particularly the demonstration of several discrete (olig
sclerosis.

The albumin fraction of the CSF increases in a wide variety of central nervous system (CNS) and c
the blood-CSF barrier, but no specific clinical correlations can be drawn. Certain enzymes that origi
kinase (CK-BB) but also enolase and neopterin, are found in the CSF after stroke or trauma and ha

Glucose Normally the CSF glucose concentration is in the range of 45 to 80 mg/dL, i.e., about two-

Acid-base balance in the CSF is of considerable interest in relation to metabolic acidosis and alkalo lower than that of arterial blood, which is 7.41. The PCO_2 in the CSF is in the range of 45 to 49 mm bicarbonate levels of the two fluids are about the same, 23 meq/L. The pH of the CSF is precisely r face of severe systemic acidosis and alkalosis. Measurements of acid-base balance in the CSF, wh in the lumbar CSF do not necessarily reflect the presence of similar changes in the brain, nor are th measurements of arterial blood gases.

The *ammonia content* of the CSF is one-third to one-half that of the arterial blood; it is increased in Reye syndrome; the concentration corresponds roughly with the severity of the encephalopathy. Th varies with changes in the serum level (high in gout, uremia, and meningitis and low in Wilson disea serum (see Table 2-1), and in uremia it rises in parallel with that in the blood. An intravenous injecti more slowly, exerting an osmotic dehydrating effect on the central nervous tissues and CSF. All 24 concentration of the total amino acids is about one-third that of plasma. Elevations of glutamine are phenylalanine, histidine, valine, leucine, isoleucine, tyrosine, and homocystine in the corresponding

Many of the *enzymes* found in serum are known to rise in CSF under conditions of disease, usually changes has proved to be a specific indicator of neurologic disease, with the possible exception of derived from granulocytes and are elevated in bacterial meningitis but not in aseptic or viral mening carcinomatous meningitis, as is carcinoembryonic antigen; the latter, however, is not elevated in ba are small and their measurement is difficult.

The catabolites of the *catecholamines* can now be measured in the CSF. Homovanillic acid (HVA), (5-HIAA), the major catabolite of serotonin, are normally present in the spinal fluid; both are five or levels of both catabolites are reduced in patients with idiopathic and drug-induced parkinsonism.

Finally, it may be said that with continued development of microchemical techniques for the analysi metabolic mechanisms of the brain, particularly in the hereditary metabolic diseases. Ultrarefined m many new catabolic products, the measurement of which will be of value in diagnosis and therapy. use for diagnostic purposes.

RADIOGRAPHIC EXAMINATION OF SKULL AND SPINE

For a long time, plain films of the cranium constituted a “routine” part of the study of the neurologic from this procedure is relatively small. Even in patients with head injury, where radiography of the s is found in only 1 out of 16 cases, at a cost of thousands of dollars per fracture and a small risk fron useful in demonstrating fractures, changes in contour of the skull, bony erosions and hyperostoses basal foramina. Plain films of the spine are able to demonstrate destructive lesions of vertebrae, fra

Sequential refinements of technique—such as pneumoencephalography, carotid and vertebral arte valuable information in special cases, but without question the most important recent advances in r development of computed tomography (CT) and magnetic resonance imaging (MRI). They represe person. A new field of bioneuropathology has been created.

Computed Tomography

In this procedure, conventional x-radiation is attenuated as it passes successively through the skull intensity of the exiting radiation relative to the incident radiation is measured, the data are integrate achievement in mathematical methodology, attributed to Hounsfield and others, permitted the astor reconstructed images of the cranium and its contents in any plane. More than thirty thousand 2- to levels of the cranium. The differing densities of bone, CSF, blood, and gray and white matter are di softened and edematous brain, abscess, and tumor tissue and also the precise size and position of significantly greater than that from plain skull films.

Magnetic Resonance Imaging

Magnetic resonance imaging, formerly referred to as nuclear magnetic resonance, also provides “s
advantages over CT of using nonionizing energy and providing better resolution of different structu
neurologic diseases, MRI is the procedure of choice.

MRI is accomplished by placing the patient within a powerful magnetic field, causing certain endog
in the longitudinal orientation of the magnetic field. Application of a brief (few milliseconds) radiofre
the atoms from the longitudinal to the transverse plane. When the RF pulse is turned off, the atoms
absorbed and then emitted results in a magnetic signal that is detected by electromagnetic receive
RF pulse must be repeated many times (a pulse sequence), the signals being measured after the a
matrix of data, which is subjected to computer analysis and from which an image is constructed.

Nuclear magnetic resonance can be accomplished with several isotopes, but current technology us
the most abundant isotope and yields the strongest magnetic signal. The image is essentially a ma
and chemical environment of the hydrogen atoms. Different tissues have different rates of proton re
contrast. The terms *T1* and *T2* refer to the time constants for proton relaxation; these may be altere
T1-weighted image, the CSF appears dark and the cortical border and cortical–white matter junctio
show the CSF as white. T2-weighted images highlight alterations in white matter such as infarction
inversion recovery) imaging is a technique that gives a high signal for parenchymal lesions and a lo
tissue, shows the earliest stages of infarction, and accentuates inflammatory demyelinating lesions

The images generated by the latest MRI machines are truly remarkable (Fig. 2-2*A*, Fig. 2-2*B* and F
and gray matter, one can identify all discrete nuclear structures and lesions within them. Deep lesio
the cervicomedullary junction are seen much better than with CT; the structures can be displayed i
structures (bony artifact). Demyelinative lesions stand out with greater clarity and infarcts can be se
disintegrated RBC—methemoglobin, hemosiderin, and ferritin—can be recognized, enabling one to
Similarly, CSF, fat, calcium, and iron have their own signal characteristics in different imaging sequ

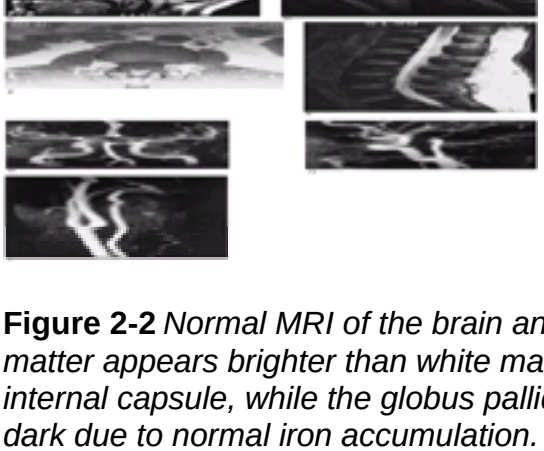


Figure 2-2 *Normal MRI of the brain and the cervical and lumbar spine.* A. *T2-weighted (SE 2500/9
matter appears brighter than white matter. The CSF within the lateral ventricles is very bright. The
internal capsule, while the globus pallidus is darker.* B. *T2-weighted (SE 2500/90) axial image at th
dark due to normal iron accumulation. The CSF within the lateral ventricles and subarachnoid spac
void”) from the rapidly flowing anterior, middle, and posterior cerebral arteries.* C. *T1-weighted (SE
appears brighter than gray matter and the corpus callosum is well defined. Subcutaneous fat and c
cervicomedullary junction are well delineated, and the region of the pituitary gland is normally demo
Note the bright signal intensity of the CSF, which provides a “myelographic” effect. Intervertebral di
demonstrate intermediate signal intensity, and the craniocervical junction is clearly defined.* E. *T1-w
level. Note that bone marrow appears brighter than disc material. The neural foramina are filled wit
of fat is present within the spinal canal posterior to the thecal sac. The CSF within the thecal sac is
demonstrated posteriorly. The facet joints are well seen.* F. *Gradient-echo sagittal image of the lum*

The MRI device is costly and requires special housing and cooling to contain its powerful magnetic proliferated and the technique has become indispensable for neurologic diagnosis. In most clinical directly to MRI after the clinical analysis.

The technology of MRI is evolving constantly. The visualization of blood vessels in the brain (MR a traumatic discontinuities of peripheral nerves (Filler et al), and of developmental defects of the CN Diffusion-weighted imaging (DWI) is a new imaging procedure that takes only a minute to accompli stroke; it is also particularly useful in distinguishing between cerebral metastases and abscesses. E interpretation of signal characteristics and morphologic changes as well as new ways of using this t ("functional" MRI or fMRI). These functional images taken in normal patients during the performanc psychiatric disease are exposing novel patterns of cerebral activation and altering some of the trad MRI to quantitate the volume of anatomic structures offers the prospect of demonstrating neuronal adapting MRI techniques to study changes in blood flow during nervous and mental activity.

The uses of CT scanning and MRI in the diagnosis of particular neurologic disorders are considere

Angiography

This technique has evolved over the last 50 years to the point where it is relatively safe and an extr malformations, narrowed or occluded arteries and veins, arterial dissections, and angiitis. Since the been limited to the diagnosis of these disorders, and refinements in the former two techniques (MR on) threaten to eliminate even these applications of conventional x-ray angiography.

Following local anesthesia, a needle is placed in the femoral or brachial artery; a cannula is then th branches to be visualized. In this way, a contrast medium can be injected to visualize the arch of th extent of these systems through the neck and into the cranial cavity. Highly experienced arteriograp about 0.1 mm in lumen diameter (under optimal conditions) and small veins of comparable size.

Angiography is not altogether without risk. High concentrations of the injected medium may induce tip and embolize the artery. Overall morbidity from the procedure is about 2.5 percent, mainly in the complications at the site of artery puncture. Occasionally a frank ischemic lesion is produced, leavi the procedure should not be undertaken unless it is absolutely necessary. A cervical myelopathy is injection; the problem is heralded by pain in the back of the neck immediately after injection. Progre over the following hours. This same complication may occur at other levels of the cord with viscera

A refinement of radiologic technique—digital subtraction angiography—uses digital computer proce and intracranial arteries. The great advantages of this procedure are that the vessels can be visual accomplished with smaller catheters than those used in standard angiography. The arterial route is route.

Magnetic Resonance and Computed Tomographic (Spiral, Helical) Angiography

These are the newest noninvasive techniques for visualizing the main intracranial arteries and can artery stenosis. These techniques approach but have not yet reached the radiographic resolution o occlusive lesions, but they are very useful in gauging the patency of the large cervical and basal ve the CT technique requires the injection of intravenous dye but has the great advantage of showing bone. The use of these and other methods for the investigation of carotid artery disease (ultrasoun Chap. 34, on cerebral vascular disease.

Positron Emission Tomography

This technique, commonly known as PET, measures the cerebral concentration of systemically adr

Contrast Myelography

By injecting 5 to 25 mL of a radiopaque dye—e.g., iopamidol (Isovue)—through a lumbar puncture subarachnoid space can be visualized. The procedure is almost as harmless as the lumbar punctu concentrations of dye near the block can cause pain and regional myoclonus. Ruptured lumbar and encroaching on the spinal cord or roots, and spinal cord tumors can be diagnosed accurately. Ioph approved by the FDA but is now employed only in special circumstances (e.g., visualizing the uppe dye from below). If iophendylate is left in the subarachnoid space, particularly in the presence of bl the spinal cord and brain.

The CT body scan also provides excellent images of the spinal canal and intervertebral foramina in scanning a useful means of visualizing spinal and posterior fossa lesions (Fig. 2-2*E* and Fig. 2-2*F*) areas within the spinal canal, such as the lateral recesses and spinal nerve root sleeves. MRI provi as well as the vertebrae and intervertebral discs. It has largely replaced contrast myelography beca three planes) within the spinal cord with greater clarity.

ELECTROENCEPHALOGRAPHY

The electroencephalographic examination, for many years a standard laboratory procedure in the s supplanted by CT and MRI. Nevertheless, it continues to be an essential part of the study of patien used in evaluating the cerebral effects of many systemic metabolic diseases, in the study of sleep, anesthetized patients. For a few diseases, such as subacute spongiform encephalopathy (page 81 some detail, since it cannot suitably be assigned to any other single chapter.

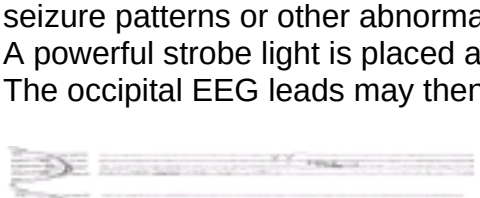
The electroencephalograph records spontaneous electrical activity generated in the cerebral cortex extracellular spaces of the brain, and these, in turn, reflect the summated effects of innumerable ex neurons. This spontaneous activity of cortical neurons is much influenced by subcortical structures, Afferent impulses from these deep structures are probably responsible for entraining cortical neuro alpha rhythm and sleep spindles (see further on).

Electrodes, which are solder or silver-silver chloride discs 0.5 cm in diameter, are attached to the s conductive paste (or conductive paste alone). The electroen-cephalograph has 8 to 24 or more am the same time. The amplified brain rhythms are strong enough to move an ink-writing pen, which pr 30 Hz (cycles per second) on paper moving at a standard speed of 3 cm/s. The resulting electroen recorded as a number of parallel wavy lines, or “channels.” Each channel represents the electrical may be used as one recording site, but the channel still represents a bipolar recording). The chann compare the activity from one region of the cerebral cortex to that from the corresponding region of being replaced by a digital format, in which the digitized waveforms are viewed on the computer sc conventional EEG but has the great advantage of providing many more channels and requiring pra

Patients are usually examined with their eyes closed and while relaxed in a comfortable chair or be activity that is recorded under restricted circumstances, usually during the waking state, from sever segment of the person's life.

In addition to the resting record, a number of so-called activating procedures are usually carried ou

1. The patient is asked to breathe deeply 20 times a minute for 3 min. Hyperventilation, through seizure patterns or other abnormalities.
2. A powerful strobe light is placed about 15 in. from the patient's eyes and flashed at frequencie The occipital EEG leads may then show waves corresponding to each flash of light (photic dri



are suspected of having epilepsy and are already being treated for it, most physicians prefer to rec
drugs. Under special circumstances these drugs may be omitted for a day or two in order to increas

The proper interpretation of EEGs involves the recognition of the characteristic normal and abnorm
the patient), the detection of asymmetries and periodic changes in rhythm, and, importantly, the dif

Types of Normal Recordings

The normal record in adults shows somewhat asymmetrical 8- to 12-per-second 50-μV sinusoidal a
and wane spontaneously and are attenuated or suppressed completely with eye opening or mental
invariant for an individual patient, although the rate may slow during aging. Also, waves faster than
recorded from the frontal regions symmetrically. When the normal subject falls asleep, the alpha rh
sharp waves and sleep spindles) appear; if sleep is induced by barbiturate or benzodiazepine drugs
be normal. A small amount of theta (4- to 7-Hz) activity may normally be present over the temporal
(1- to 3-Hz) activity is not present in the normal waking adult.

During stroboscopic stimulation, an occipital response to each flash of light may normally be seen (
calcarine cortex 20 to 30 ms after the flash of light. The presence of such a response indicates that
contrary, the patient is either hysterical or malingering. The evoked visual responses (see further on
blindness than occipital driving, since the latter may be absent in normal persons. Spread of the oc
abnormal waves, provides evidence of abnormal excitability (Fig. 2-3*B* and Fig. 2-3*C*). Seizure pat
accompanied by gross myoclonic jerks of the face, neck, and limbs (photomyogenic or photomyocl
photoconvulsive response). Such effects occur with some frequency during periods of withdrawal fr
differentiated from purely myoclonic ones that are produced normally by contracting scalp muscles

Children and adolescents are more sensitive than adults to all the activating procedures mentioned
during the middle and latter parts of a period of overbreathing. This activity, referred to as “breakdo
frequency of the dominant rhythms in infants is normally about 3 Hz, and they are very irregular. W
regularity of these occipital rhythms; by the age of 12 to 14 years, normal alpha waves are the dom
the brain as expressed in the EEG). The records of infants and children are difficult to interpret bec
rigid classification using frequency criteria impossible (see Hahn and Tharp). Nevertheless, asymm
abnormal in children of any age. Also, normal patterns in the fetus, from the seventh month onward
described by Stockard-Pope et al and by deWeerd, are clearly indicative of a developmental disord

Types of Abnormal Recordings

The most pathologic finding of all is the replacement of the normal EEG pattern by “electrocerebral
recorded from the scalp, is absent. Artifacts of various types should be seen as the amplifier gains
connected to the machine. Acute intoxication with high levels of drugs such as barbiturates can pro
absence of nervous system depressants or extreme degrees of hypothermia, a record that is “flat” (
almost always a result of profound cerebral hypoxia or ischemia or of trauma and raised intracrania
reflexes, and spontaneous respiratory or muscular activity of any kind for 6 h or more—is said to be
there is no chance of neurologic recovery. The topic of brain death is discussed further in Chap. 1

Localized regions of absent brain waves may rarely be seen when there is a particularly large area
clot lies between the cerebral cortex and the electrodes. With such a finding, the EEG localization o
of the lesion is not disclosed. However, most such lesions are not large enough, relative to the reco
then record abnormal waves arising from functioning though damaged brain at the borders of the le

Two types of abnormal waves, already mentioned, are of lower frequency and higher amplitude tha
μV are called *delta* waves (Fig. 2-3*G* and Fig. 2-3*H*); those with a frequency of 4 to 7 Hz are called
usually reflects the effects of sedative drugs or, if focal, an immediately underlying skull defect (bon
sharp waves are transient high-voltage waveforms that have a pointed peak at conventional paper
waves that occur interictally in epileptics or in individuals with a genetic disposition to seizures are

have been seizures. The highly characteristic pattern of Creutzfeldt-Jakob disease is shown in Fig

Cerebrovascular Disease The EEG is now little used in the differential diagnosis of vascular hemi
distinguish an acute lesion in the distribution of the internal carotid or other major cerebral artery, w
contrast, with a lacunar infarction deep in the cerebrum or brainstem, the surface EEG is usually no
roughly 50 percent of patients with infarction in the territory of the middle cerebral artery have a nor
even in the week or two following the stroke. A persistent abnormality is associated with a poor pro
midbrain produce bilaterally synchronous slow waves, but those of the pons and medulla (i.e., belo
near-normal EEG pattern despite catastrophic clinical changes.

Cerebral Trauma A brief episode of cerebral concussion in animals is accompanied by a transitory
evident by the time a recording can be made. Large cerebral contusions produce EEG changes sim
often give way to focal ones, especially if the lesions are on the superolateral surfaces of the brain,
months. Sharp waves or spikes sometimes emerge as the focal slow-wave abnormality resolves ar
EEGs may be of prognostic value in this regard. They may also aid in evaluating patients for subdu

Diseases That Cause Coma and States of Impaired Consciousness The EEG is abnormal in a
There is a fairly close correspondence between the severity of acute anoxic damage from cardiac a
associated with generalized theta activity, intermediate forms with widespread delta waves and the
“burst suppression,” in which the recording is almost isoelectric for several seconds, followed by hig

The term *alpha coma* refers to a unique EEG pattern in which an apparent alpha activity in the 8- to
than posteriorly. When analyzed carefully, this apparent alpha, unlike the normal rhythm, is found n
band. This is usually a transitional pattern after global anoxia; less often, alpha coma may be seen
coma are usually transitional patterns leading to severe generalized slowing and voltage reduction
waves are normal in configuration but usually of decreased frequency.

In altered states of consciousness, the more profound the depression of consciousness, in general
coma, the slow (delta) waves are bilateral and of high amplitude and tend to be more conspicuous
conditions as acute meningitis or encephalitis and disorders that severely derange blood gases, glu
impairment of consciousness accompanying the large cerebral lesions discussed above. In *hepatic*
with the degree of confusion, stupor, or coma. Characteristic of hepatic coma are paroxysms of bila
although such waveforms may also be seen with encephalopathies related to renal or pulmonary fa

An EEG may also be of help in the diagnosis of coma when the pertinent history is unavailable. Per
epilepticus in the absence of obvious convulsions (“spike-wave stupor,” epileptic fugue state). It ma
encephalopathy, intoxication with barbiturates or other sedative-hypnotic drugs, the effects of diffus

Diffuse Degenerative Diseases Alzheimer disease and other degenerative diseases that cause s
relatively slight degrees of diffuse slow-wave abnormality in the theta (4- to 7-Hz) range. More rapid
panencephalitis (SSPE), Creutzfeldt-Jakob disease, and to a lesser extent the cerebral lipidoses—
pathognomonic EEG changes consisting of periodic bursts of high-amplitude sharp waves, usually
normal EEG in a patient who is profoundly apathetic is a point in favor of the diagnosis of catatonia

Other Diseases of the Cerebrum Many disorders of the brain cause little or no alteration in the EE
examples, though as many as 50 percent of advanced cases will have an abnormal record of nons
Wernicke-Korsakoff disease, despite the dramatic nature of the clinical picture, cause little or no ch
confusional states that have been designated by some clinicians as hypokinetic delirium (Chap. 20
manic-depressive disorders or schizophrenia), intoxication with hallucinogenic drugs such as LSD,
either with no modification of the normal record or with only minor nonspecific abnormalities unless

Clinical Significance of Minor EEG Abnormalities

The gross EEG abnormalities discussed above are by themselves clearly abnormal, and any formu

Table 2-2 Main sensory evoked potential latencies from stimulus, milliseconds[a]

Visual Evoked Potentials For many years it had been known that a light stimulus flashing on the r
In the EEG, such responses to fast rates of stimulation are referred to as the occipital driving respo
that a visual evoked response could be produced by the sudden change of a viewed checkerboard
the pattern reversal, were easier to detect and measure than flash responses and more consistent
this type of stimulus, applied first to one eye and then to the other, could demonstrate conduction d
a disease of the optic nerve—even though in some instances there were no residual signs of reduc
nerve head, or changes in pupillary reflexes.

This procedure, called pattern-shift visual evoked responses (PSVER) or pattern-reversal visual ev
delicate tests of lesions in the visual system. Figure 2-4 illustrates the normal PSVER and two type
and duration of PSVER accompany the abnormally prolonged latencies, but they are difficult to qua
100 ms (thus the term “P 100”); an absolute latency over approximately 118 ms or a difference in la
involvement of one optic nerve (Table 2-2). Bilateral prolongation of latencies, demonstrated by sep
nerves, in the optic chiasm, or in the visual pathways posterior to the chiasm.

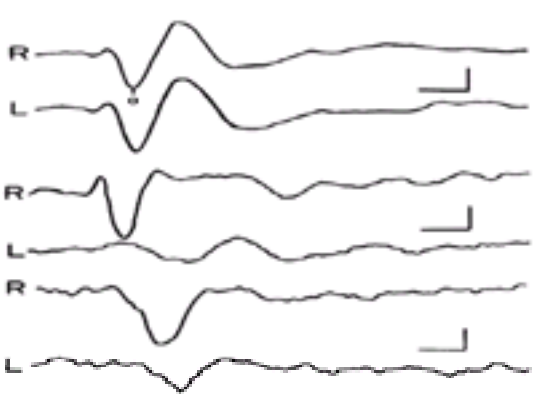


Figure 2-4 *Pattern-shift visual evoked responses (PSVER). Latency measured to first major positiv
are normal. Middle tracings: PSVER from the right eye is normal but the latency of the response fro
tracings: PSVER from both eyes show abnormally prolonged latencies, somewhat greater on the le
permission from Shahrokhi et al.)*

As indicated above, PSVER is especially valuable in proving the existence of active or residual dise
who were known to have had retrobulbar (optic) neuritis showed that among 51 such patients, only
found similar abnormalities of PSVER in about one-third of multiple sclerosis patients who had no h
of abnormal PSVER in a patient with a clinically apparent lesion elsewhere in the CNS may usually

A compressive lesion of an optic nerve will have the same effect as a demyelinative one. Many oth
amblyopias, ischemic optic neuropathy, and the Leber type of hereditary optic neuropathy—show a
involving structures anterior to the retinal ganglion cells may also produce increased latencies. Imp
well with the amplitude of the PSVER. By presenting the pattern-shift stimulus to one hemifield, it is
radiation, or one occipital lobe, but with less precision than that provided by the usual monocular te

Brainstem Auditory Evoked Potentials The effects of auditory stimuli can be studied in the same

recording the evoked potentials (for the upper limb) over Erb's point above the clavicle, over the C-
limb) over the lumbar and cervical spines and the opposite parietal cortex. The impulses generated
computer can be traced through the peripheral nerves, spinal roots, and posterior columns to the n
lemniscus to the contralateral thalamus, and thence to the sensory cortex of the parietal lobe. Dela
indicates peripheral nerve disease; delay from Erb's point (or lumbar spine) to C-2 implies an abno
posterior columns; the presence of lesions in the medial lemniscus and thalamoparietal pathway ca
parietal cortex (Fig. 2-6). The normal waveforms are designated by the symbol P (positive) or N (ne
from stimulus to recording (e.g., N 11, N 13, P 13, P 22, etc.). As shorthand for the polarity and app
cervicomedullary junction is termed “N/P 13,” and the cortical potential from median nerve stimulati
19–P 22.” The corresponding cortical wave after tibial or peroneal nerve stimulation is called “N/P 3

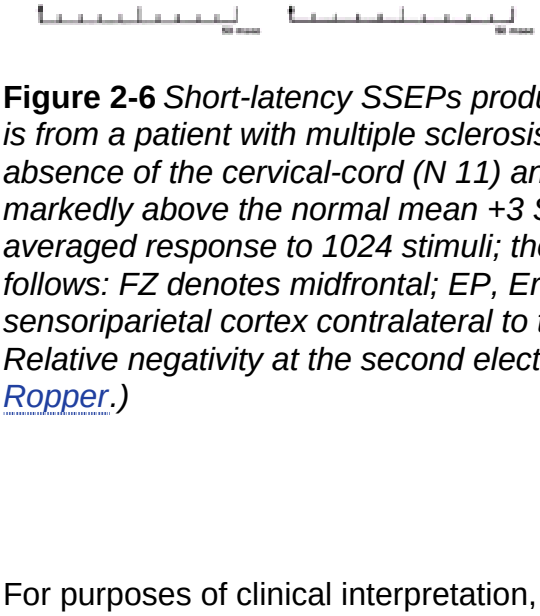


Figure 2-6 *Short-latency SSEPs produced by stimulation of the **median nerve** at the wrist. The set
is from a patient with multiple sclerosis who had no sensory symptoms or signs. In the patient, note
absence of the cervical-cord (N 11) and lower-medullary components (N/P 13), and the latency of t
markedly above the normal mean +3 SD for the separation from the brachial plexus. Unilateral stim
averaged response to 1024 stimuli; the superimposed trace represents a repetition to demonstrate
follows: FZ denotes midfrontal; EP, Erb's point (the shoulder); C2, the middle back of the neck over
sensoriparietal cortex contralateral to the stimulated limb.*
*Relative negativity at the second electrode caused an upward trace deflection. Amplitude calibratio
Ropper.)*

For purposes of clinical interpretation, the SEPs are assumed to be linked in series, so that interwa
the generators of the two peaks involved (Chiappa and Ropper). Normal values are shown in Table
are to be found in the monograph by Chiappa. This test has been most helpful in establishing the e
brainstem in disorders such as the Guillain-Barré syndrome, ruptured lumbar and cervical discs, mu
the clinical data are uncertain. The counterpart also pertains—namely, that obliteration of the cortic
reflects profound damage to the somatosensory pathways in the hemisphere or to the cortex itself.
waves after cardiac arrest is a powerful predictor of a poor clinical outcome. Likewise, the persisten
profound damage that only a limited clinical recovery is to be expected.

Evoked potential techniques have also been used in the experimental study of olfactory sensation (

Transcranial Motor Cortex Stimulation

It is now possible, by using single-pulse high-voltage magnetic stimulation, to directly activate the m
lack of conduction in descending motor pathways. This technique, introduced by Marsden and asso
(presumably Betz cells) and the fastest-conducting axons. Cervical cord stimulation is believed to a
cortical and cervical activation of hand or forearm muscles represents the conduction velocity of the
who have applied the technique to 20 hemiplegic patients with cerebrovascular lesions, found that

risk than does a craniotomy and open biopsy. In choosing to perform a biopsy in any of these clinic
definitive diagnosis—one that would permit successful treatment or otherwise enhance the manage

CHAPTER REFERENCES

ALTENMÜLLER EO, GERLOFF C: Psychophysiology and the EEG, in Niedermeyer E, Lopes DaSilva F (eds): *Electro* ed. Baltimore, Williams & Wilkins, 1999, pp 637–665.

AMERICAN ELECTROENCEPHALOGRAPHIC SOCIETY: Guidelines in electroencephalography, evoked potentials, a

BARNETT GH, ROPPER AH, JOHNSON KA: Physiological support and monitoring of critically ill patients during magn

BARROWS LJ, HUNTER FT, BANKER BQ: The nature and clinical significance of pigments in the cerebrospinal fluid.

BERARDELLI A, INGHILLERI M, MANFREDI M, et al: Cortical and cervical stimulation after hemispheric infarctions.

BIGNER SH: Cerebrospinal fluid (CSF) cytology: Current status and diagnostic applications. *J Neuropathol Exp Neuro*

BLUME WT: *Atlas of Pediatric Electroencephalography*. New York, Raven Press, 1982.

CHIAPPA KH (ed): *Evoked Potentials in Clinical Medicine*, 3rd ed. Philadelphia, Lippincott-Raven, 1997.

CHIAPPA KH, ROPPER AH: Evoked potentials in clinical medicine. *N Engl J Med* 306:1140, 1205, 1982.

CHIMOWITZ MI, LOGIGIAN EL, CAPLAN LR: The accuracy of bedside neurological diagnoses. *Ann Neurol* 28:78, 19

DALY DD, PEDLEY TA (eds): *Current Practice of Clinical EEG*, 2nd ed. New York, Raven Press, 1990.

DAWSON GD: A summation technique for the detection of small evoked potentials. *Electroencephalogr Clin Neuroph*

DEWEERD AW: *Atlas of EEG in the First Months of Life* . New York, Elsevier, 1995.

DEN HARTOG-JAGER WA: *Color Atlas of CSF Cytopathology*. New York, Elsevier-North Holland, 1980.

DUFRESNE JJ: *Cytopathologie de CSF*. Basel, Ciba Foundation, 1973.

FILLER AG, KLIOT M, HOWE FA, et al: Application of magnetic resonance in the evaluation of patients with peripheral

FISHMAN RA: *Cerebrospinal Fluid in Diseases of the Nervous System*, 2nd ed. Philadelphia, Saunders, 1992.

GILMAN S: Imaging of the brain. *N Engl J Med* 338:812, 889, 1998.

GOLDENSHOHN ES, WOLF S, KOSZER S, LEGATT A (eds): *EEG Interpretation*, 2nd ed. New York, Futura, 1998.

GREENBERG JO (ed): *Neuroimaging: A Companion to Adams and Victor's Principles of Neurology*, 2nd ed. New York

HAHN JS, THARP BR: Neonatal and pediatric electroenceph-alography, in Aminoff MJ (ed): *Electrodiagnosis in Clinic*

HOROWITZ AL: *MRI Physics for Radiologists*, 2nd ed. New York, Springer, 1992.

HUGHES JR: *EEG in Clinical Practice*, 2nd ed. Woburn, MA, Butterworth, 1994.

HUK WN, GADEMANN G, FRIEDMAN G: *MRI of Central Nervous System Diseases*. Berlin, Springer Verlag, 1990.

KANAL E, GILLEN J, EVANS JA, et al: Survey of reproductive health among female MR workers. *Radiology* 187:395,

LATCHAW RE (ed): *MRI and CT Imaging of the Head, Neck, and Spine*, 2nd ed. St. Louis, Mosby–Year Book, 1991.

MARSDEN CD, MERTON PA, MORTON HB: Direct electrical stimulation of corticospinal pathways through the intact s

MODIC MT, MASARYK TJ, ROSS JS, et al: *Magnetic Resonance Imaging of the Spine*, 2nd ed. St. Louis, Mosby–Yea

Chapter 3

MOTOR PARALYSIS

Definitions

The term *paralysis* is derived from the Greek words *para*, “beside, off, amiss,” and *lysis*, a “loosenir of function, either sensory or motor. When applied to motor function, *paralysis* means loss of volun any point from the cerebrum to the muscle fiber. A lesser degree of paralysis is spoken of as *pares* partial or complete loss of function. The word *plegia* comes from a Greek word meaning “to strike,” meaning as *paralysis*. All these words are used interchangeably, though generally one uses *paraly* *paresis* for partial loss.

THE LOWER MOTOR NEURON

Anatomic and Physiologic Considerations

Each spinal and cranial motor nerve cell, through the extensive arborization of the terminal part of i more muscle fibers; together, they constitute *the motor unit*. All variations in the force, range, rate, a motor units called into action and the frequency and sequence of firing of each motor unit. Feeble m movements recruit many more units of increasing size. When a motor neuron becomes diseased, a irritability, i.e., the axon is unstable and capable of ectopic impulse generation, and all the muscle fi other units. The result of contraction of one or several such units is a visible twitch, or *fasciculation* spontaneous muscle action potential. Simultaneous or sequential spontaneous contractions of mult *myokymia*. If the motor neuron is destroyed, all the muscle fibers that it innervates undergo profoun interruption of a motor nerve, the individual denervated muscle fibers begin to contract spontaneou *fibrillation*. Inability of the isolated fiber to maintain a stable membrane potential is a more likely exp intact skin, but it can be recorded as a small, repetitive, short-duration potential in the EMG (Chap