A Classification and Outline of Cerebrovascular Diseases II

A report by an ad hoc Committee established by the Advisory Council for the National Institute of Neurological and Communicative Disorders and Stroke, National Institutes of Health, Bethesda, Maryland 20014.

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# A Classification and Outline of Cerebrovascular Diseases II

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CLASSIFICATION AND OUTLINE OF CEREBROVASCULAR DISEASES II

Introduction*

The purpose of this “Classification and Outline of Cerebrovascular Diseases II” is: (1) to place in classified form the known types of cerebrovascular disease in such a manner that all or a portion of the classification will be of value to clinicians, pathologists, physiatrists as well as other disciplines; (2) to give practical meaning to the classification by defining the terms so they can be employed interchangeably by clinicians and investigators in various parts of the country; and (3) to set down in organized fashion the salient diagnostic criteria for a number of clinical entities.

Since the time of the deliberations of the first ad hoc Committee on Cerebrovascular Disease of NINDCS, there has been considerable expansion of the body of knowledge concerning cerebrovascular disease. It continues to be evident that in such a complex set of clinical-pathophysiological phenomena some standard reference language or set of definitions should be used or the literature of investigation will be uninterpretable. An example of this: Does the term “transient ischemic attack” include diffuse ischemia (syncpe) or is the term limited to episodes of focal ischemia? Follow-up data about the frequency of stroke in patients with the former are entirely different than follow-up data concerning patients with the latter! The second ad hoc Committee on Nomenclature of Cerebrovascular Diseases welcomed the opportunity to continue the task of producing a lucid, usable and acceptable classification and set of definitions and diagnostic criteria for standard use. As in the first “Classification and Outline of Cerebrovascular Diseases,” it is recognized that many of the statements on the following pages are only tentative ones and, as understanding of mechanisms and causes increases, some of these statements will need to be changed.

By the term “cerebrovascular” we denote all disorders in which there is an area of brain transiently or permanently affected by ischemia or bleeding, or in which one or more brain blood vessels are primarily involved in a pathological process, or a combination of the two. The former includes such phenomena as a sudden change in cardiac rhythm causing a decreased arterial perfusion pressure to the brain, the temporary redistribution of blood in “hemiplegic migraine,” etc., while under pathological process are included abnormalities of the blood vessel wall, stenosis or occlusion by thrombus or embolus, and altered permeability to plasma and blood cells.

The term “cerebrovascular” is now well established and designates this whole category of diseases. “Cerebrovascular” and “cerebral” are used in the original Latin sense, referring to the whole brain and not merely to the hemispheres of the forebrain.

Basis of the Classification

The Basis of the Classification has been changed as it is no longer possible to fill the spectrum of clinical and research needs with a classification which is limited to pathology. As the Committee reviewed the first “Classification and Outline of Cerebrovascular Diseases,” it was apparent that a different approach would be required in order to classify: (a) the temporal profile of development of a cerebrovascular abnormality, (b) such transient pathophysiological mechanisms as vasospasm, changes in cardiac rhythm, systemic hypotension, hypercapnia, etc., (c) anatomical parameters of the blood vessel system or of the brain and spinal cord, (d) neurological clinical phenomena whether history, physical examination, laboratory results, etc., and (e) the performance capabilities of the patient related to the placement requirements of the patient when changing care status, whether hospital to home, nursing home, etc.

Therefore, the revised Classification is based on division into six parts: Part I. Clinical Stage, Part II. Pathophysiological Mechanisms, Part III. Anatomy (blood vessels, brain), Part IV. Pathology (blood vessels, brain), Part V. Clinical Phenomena, and Part VI. Status of Patient (Performance and Placement). This division into six parts makes it possible to classify any cerebrovascular problem, at any point in time, and also makes it possible for individuals with some special interest or need concerning one or two parts to fulfill those selective needs. As is so often true in clinical medicine, sequential reclassification in Part I (Clinical Stage) will often be necessary as a patient’s problem proceeds through progressing cerebral infarction to completed cerebral infarction. Various combinations, of course, may occur. Part VI (Patient Status) may not be used until some time into a patient’s hospital course, and Part V (Clinical

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Phenomena) may be only partially completed in many instances. The idea of "thinking through" the entire classification, as the busy physician deals with a patient problem, is paramount as the Classification provides an orderly format for proceeding from clinical stage to pathophysiological mechanism — anatomical locus of the process — pathology — clinical phenomena including neurovascular examination and laboratory evaluation to the patient's capability status with or without need for special placement. In certain instances, a process or disease had to be listed under more than one major category, i.e., thrombosis under Pathophysiological Mechanisms and Pathology, and embolism in the same two subdivisions.

An attempt has been made to make the Classification, or some portion of it, serviceable to the various disciplines dealing with clinical service, training or research concerning the Cerebrovascular Diseases. In the interest of practicability and to assist the student, bold-faced type has been used for the common disorders. References to the location of items in the Outline are included in the Classification.

Part I. Clinical Stage

INTRODUCTION

The primary purpose of this section of the Classification is to provide a framework for the description of the current status of the patient in reference to the temporal profile of the disease without regard to other factors, such as etiology, pathology, neural deficit and the like, all of which are described in separate sections.

As the condition of the patient may be changing, sometimes rapidly, a static description presents certain problems, particularly as to the exact time when a patient is categorized. In general, a patient is placed into one of the categories at the time he is studied in sufficient detail so as to permit a reasonably certain clinical diagnosis. Because of the evolution of the deficit, it is likely that patients will be in different categories at different times; in these instances, a statement must be made to indicate the time of categorization. It is possible, also, that some deficits will be stable while other deficits are evolving; in these instances, appropriate comments will need to be made. By using more than one category, an accurate description of a complex temporal profile can be built (e.g., a patient who has transient ischemic attacks followed by a completed stroke with a permanent residual neurological deficit and who subsequently again has transient ischemic attacks would be classified as I.B.1., I.B.3., and later I.B.1.).

It is obvious that an infinite number of variations can be discerned among the patterns of cerebrovascular disease; thus, any classification is somewhat arbitrary. Furthermore, the purist may note some mixing of clinical description with pathological entities. The Committee recognized that the rate of change might be important, but to describe this fully would lead to too complex a Classification.

Part I. Clinical Stage

A. Asymptomatic

This category is for cerebrally asymptomatic individuals who are found to have evidence which may potentially be important as predisposing to future cerebrovascular disease. Such evidence is listed in detail in Part II. Some of the factors in this evidence have been termed the "Stroke Prone Profile."

B. Focal cerebral dysfunction

This category refers to focal brain dysfunction regardless of the nature of the vascular pathology (e.g., ischemic disease, intracranial hemorrhage, arteritis).*

1. Transient attacks (transient ischemic attacks)

These are episodes of temporary and focal cerebral dysfunction of vascular origin, which are variable in duration, commonly lasting from 2 to 15 minutes, but occasionally lasting as long as a day (24 hours). They leave no persistent neurological deficit. These attacks are usually called transient ischemic attacks (TIA) because their pathogenesis is believed to be ischemic. However, in rare instances, it is possible that such attacks may be associated with other types of vascular pathophysiology. (For complete description, see Outline of Cerebrovascular Diseases.)

Many factors might be included to describe the characteristics of the change or the rate of change in each individual attack or the pattern in many attacks. These factors are dealt with in I.B.2.

2. Actively changing neurological deficit

This category refers to a patient whose neurological deficit is actively changing in amount during the period of observation (specify duration from time of onset). The deficit may be getting more severe or less severe.

a. Improving

b. Worsening (also known as "stroke-in-evolution" or "progressing stroke")

3. Prolonged neurological deficit

This category refers to a relatively stable neurological deficit, that is, during the period of observation for categorization (specify duration

*Migraine syndrome. The aura of migraine (e.g., scintillating scotoma) is commonly attributed to focal cerebral ischemia owing to vasospasms. In some instances more severe neurological deficits precede the headache and may include hemiparesis, dysphasia and homonymous hemianopia and in the great majority of instances these deficits are transitory, only rarely persisting long enough to produce a cerebral infarct. The temporal profile may be classified under "Clinical Stage" while the word "migraine" is under I.A.5.b.
from time of onset) little or no change in the deficit is occurring.

a. Duration more than 24 hours and less than three weeks (sometimes referred to as a reversible ischemic neurological deficit or RIND)
b. Duration more than three weeks; often permanent (commonly known as "completed stroke")

C. General cerebral dysfunction
This category refers to general cerebral ischemia, which results from reduction in blood supply to the brain, yet is applicable also to lesions of other causes. This category does not imply the presence or absence of disease of the cerebral arteries.

1. Transient
   Brief episodes of general cerebral ischemia, the classic example of which is simple fainting. It is possible that loss of consciousness may result from ischemia of focal areas of the brain, areas whose integrity is required for maintenance of consciousness; in those instances in which a decision can be made that such episodes are in fact focal, the patient should be categorized under I.B.

2. Prolonged
   a. Acute onset, e.g., cardiac arrest
   b. Gradual or progressive onset

Part II. Pathophysiological Mechanisms

INTRODUCTION
Clinicians continuously ponder questions about mechanisms as they watch events unfold in the course of an illness. If the mechanisms (pathogenesis) of a focal abnormality of blood supply to the brain can be determined, corrective measures often can be instituted. There are processes which produce no specific tissue change which ultimately can be identified by the pathologist. These include such common items as thrombosis with lysis, embolism with fragmentation, vasospasm, hypotension, abnormalities of cardiac rhythm, and many others. Consideration of these factors is vital to the formulation of an outline of the mechanisms in a specific patient problem. In certain instances, it may be necessary to list more than one mechanism, e.g., cerebral embolism from a cardiac source, hypotension and myocardial infarction.

Section E, possible predisposing factors, is included to record the role of these abnormalities as "risk factors."

As the pathologist works with the problem of classifying autopsy findings, he may find it helpful to be able to use this section on Pathophysiological Mechanisms in instances where the clinical record contains such evidence as cholesterol retinal emboli and atrial fibrillation; while the autopsy findings include no cause for a hemorrhagic cerebral infarction. This section may stimulate more detailed search for evidence of some causes of stroke.

Part II. Pathophysiological Mechanisms

A. Primary abnormalities of cerebral circulation
   (specify transient or persistent)
   1. Thrombosis
      a. Lysis
      b. Recannulation
      c. Collateral flow
   2. Embolism
      a. Intraluminal source
      b. Cardiac source
      c. Other source
   3. Hemorrhage (specify — see Anatomy and Pathology)
   4. Compression
      a. Change of position of head, neck or arm
         1) Osteoarthritis of cervical vertebrae
         2) Atlas to axial joint
         3) Fibrous bands
         4) Kinks
         5) Loops
         6) Fracture
      b. Expanding mass
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c. External forces
   1) Manipulation
   2) Surgery
d. Cerebral edema
e. Acceleration
5. Vasospasm
   a. Post-traumatic
   b. Migraine
c. Post-intracranial hemorrhage (specify subarachnoid, intracerebral)
d. Post-subarachnoid hemorrhage
e. Hypertension (pheochromocytoma, acute renal disease, eclampsia)
f. Manipulation (e.g., surgery)
g. Embolism
   h. Drugs (e.g., Ergot, Sansert, etc.)
i. Other
6. Direction
   a. Reversal
   b. Shunts
7. Alteration in rate and/or volume (as seen in serial arteriogram)
   Specify decrease or increase
   a. Focal
   b. General
8. Dissection of arterial wall (specify artery)
   a. Transient disturbance of flow with subsequent restoration
9. Associated with arteriography

B. Abnormalities of general circulation
1. Hypotension — define
   (specify transient or persistent)
a. Cardiac abnormalities
   1) Abnormality of rate
   2) Abnormality of rhythm
   3) Conduction defects
   4) Myocardial impairment
      a) Infarct
      b) Myocarditis
      c) Myocardial degenerative disease
   5) Valvular disease including prosthesis
   6) Pericardial disease and/or effusion
b. Reflex
   1) Carotid sinus hypersensitivity
   2) Vasovagal
c. Shock (specify cause)
d. Endocrine (specify cause)
e. Blood loss
   1) Hemorrhage
   2) Pooling (specify region)
f. Orthostatic hypotension
g. Valsalva’s maneuver
h. Neurological disease
   1) Central nervous system (specify site)
   2) Peripheral neuropathy
   3) Autonomic nervous system
i. Iatrogenic
   1) Medication
   2) Post-sympathectomy
   3) Post-reconstruction vascular surgery (specify site)
j. Undetermined
2. **Hypertension** (specify transient or persistent) [See Appendix for grading of retinal changes — Wagener and Keith Classification]
   a. Unknown cause, including essential
   b. Endocrine
   c. Medication
   d. Renal
   e. Emotional
   f. Physical exertion
   g. Toxemia of pregnancy

C. **Alterations in blood**

1. **Viscosity**
   a. Dehydration
   b. Overhydration
   c. Other (specify)

2. **Cellular constituents**
   a. Erythrocytes
      1) Anemia
      2) Polycthyemia
      3) Hemoglobinopathy
         a) Sicklemia
         b) Hemoglobin C
   b. Leukocytes
   c. Thrombocytes (e.g., thrombocytosis, thrombocytopenia)

3. **Clotting defects**
   a. Hypercoagulability (specify cause when known, including medication)
   b. Hypocoagulability (specify cause when known, including medication)

4. **Proteins**
   a. Macroglobulins (specify type if known)
   b. Cryoglobulins
   c. Hyperfibrinogenemia
   d. Other

5. **Lipids**
   a. Cholesterol
   b. Triglycerides
   c. Lipoprotein
   d. Other

6. **Glucose**
   a. Hypoglycemia
   b. Hyperglycemia

7. **Blood gases**
   a. Oxygen
      1) Hypoxia
         a) Hypoventilation
            (1) Musculoskeletal disease
            (2) Pulmonary disease
            (3) Neurogenic
         b) Environmental deficiency
      2) Hyperoxia
   b. Carbon dioxide
      1) Hypercapnia
         a) Hypoventilation
            (1) Musculoskeletal disease
            (2) Pulmonary disease
            (3) Neurogenic
         b) Environmental CO₂ excess
      2) Hypocapnia
         a) Hyperventilation
            (1) Iatrogenic
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(2) Psychophysiological
(3) Neurogenic
c. Carbon monoxide
d. Nitrogen
e. Other gases
8. Electrolytes
   a. Hyponatremia
9. Hydrogen ion-content
10. Others including radiopaque material

D. Alteration of metabolic demands
1. Thermal changes
   a. Hypothermia
   b. Hyperthermia
2. Convulsion
3. Medications (specify, e.g., barbiturates, etc.)

E. Possible predisposing factors
1. Hypertensive disease
2. Diabetes mellitus
3. Cardiac disease
4. Hyperlipidemia
5. Genetic
6. Cigarette smoking
7. Hyperuricemia
8. Obesity
9. Drugs
10. Endocrine (? hypothyroidism, etc.)
11. Other
F. Unknown

Part III. Anatomy

INTRODUCTION

Anatomical structures are considered in two major subdivisions: (A) blood vessels (arteries and arterial anastomoses important for collateral circulation, arterial anomalies, arterioles, veins, venules and capillaries); and (B) central neural parenchyma (brain or spinal areas to which blood is supplied or from which it is drained; peripheral and autonomic nervous systems are not included).

The nomenclature of Nomina Anatomica (N.A.) 1966 and the Standard Nomenclature of Diseases and Operations (S.N.) 1961 are used as much as possible. More detailed anatomy of some vessels and neural structures can be found in N.A., e.g., thalamus, hypothalamus, brain stem, cerebellum, cranial nerves, spinal cord. S.N. lists left or right side only for certain structures. When these are of importance but not specifically designated, the small-case letters l. and r. will, in this Classification, indicate left and right, respectively. The small-case letter a. is used as an abbreviation for artery, aa. for arteries, v. for vein, vv. for veins, s. for venous sinus.

Although conventional anatomical subdivisions are used as much as possible, practical considerations have resulted in some exceptions, e.g., the subclavian artery is divided into three parts which are different from the three or four parts of some anatomy texts. All known branches of a specific artery are not included in the Classification but only those considered important in cerebrovascular and spinovascular disease or those known to have important anastomoses. A strict order of arterial origin is not always followed, e.g., in the case of branches of the anterior cerebral artery. Arterial anastomoses are not separately listed but may be described by indicating specific arterial systems and branches involved.

Under “Brain and spinal cord,” major subdivisions of “Hemisphere” and “Brain stem” have been introduced, as these anatomical terms are commonly used by clinicians and investigators working with cerebrovascular disease.
5) Central white matter (specify by lobe)
6) Internal capsule
   a) Anterior limb
   b) Genu
   c) Posterior limb
7) Thalamus
8) Inferior and medial hemisphere surfaces
   a) Hippocampal gyrus
   b) Corpus callosum
   c) Other (specify)
9) Corpus striatum
   a) Caudate nucleus
   b) Lentiform nucleus
      (1) Putamen
      (2) Globus pallidus
10) Hypothalamus
11) Insula
12) Other (specify)

b. Brain stem
1) Midbrain
   a) Tectum
   b) Tegmentum
   c) Cerebral peduncle
2) Pons
   a) Tegmentum
   b) Basis
3) Medulla oblongata
   a) Dorsal
   b) Ventral

c. Cerebellum
1) Vermis
2) Cerebellar hemisphere
3) Cerebellar nuclei
4) Superior cerebellar peduncle
5) Middle cerebellar peduncle
6) Inferior cerebellar peduncle
d. Cranial nerves
   1) Olfactory n. (I)
   2) Optic n. (II)
      a) Optic disk
      b) Prechiasmatic portion
      c) Optic chiasm
      d) Optic tract
   3) Oculomotor n. (III)
   4) Trochlear n. (IV)
   5) Trigeminal n. (V)
      a) Ophthalmic n.
      b) Maxillary n.
      c) Mandibular n.
      d) Gasserian ganglion
   6) Abducens n. (VI)
   7) Facial n. (VII)
   8) Acoustic n. (VIII) (vestibulocochlear n.)
      a) Cochlear division
      b) Vestibular division (labyrinthine division)
   9) Glossopharyngeal n. (IX)
10) Vagus n. (X)
11) Accessory n. (XI) (spinal accessory n.)
12) Hypoglossal n. (XII)
e. Cerebral ventricles
1) Lateral ventricle (specify r. or l.)
2) Third ventricle
   a) Choroid plexus
   b) Cerebral aqueduct (Sylvius)
3) Fourth ventricle

3. Spinal cord
   a. Gray matter (specify level)
   b. White matter (specify level)
   c. Dorsal roots (specify level)
   d. Ventral roots (specify level)

Part IV. Pathology

INTRODUCTION
Pathological alterations are considered in two major subdivisions: (A) blood vessels (arteries, arterioles, capillaries, venules, veins, and combined arterial, venous and capillary lesions); and (B) neural parenchymatous lesions.

The etiological classification of the Standard Nomenclature of Diseases and Operations (S.N.) 1961 and the “Classification and Outline of Cerebrovascular Disease” (Neurology, Volume 8, Number 5, May 1958) are used whenever possible. Some additions, deletions and modifications have been made but the main portions of the Classification can be compared with the S.N. and the 1958 Classification. In the sections concerned with lesions of capillaries, the word petechiae is used since it connotes hemorrhages which are small enough to be recognized as presumably of capillary origin, although such hemorrhagic lesions may also be of arteriolar and venular origin.

The World Federation of Neurology code for grading atherosclerosis is in the Appendix.

Part IV. Pathology

A. Pathological alterations in vessels
1. Arteries (and arterioles, when applicable)
   a. Congenital, developmental and inherited lesions
      1) Absence of artery (aplasia)
      2) Hypoplasia of artery
      3) Anomaly of artery (fetal form)
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4) Anomaly of artery (unspecified)
5) Redundancy (loops), dilatation, elongation; congenital, of artery
6) Aneurysm, congenital
   a) Ruptured, aneurysm, congenital
7) Genetically determined defects in arteries
   a) Marfan’s syndrome (arachnodactyly)
   b) Ehlers-Danlos syndrome
   c) Pseudoxanthoma elasticum
   d) Others
b. Inflammatory lesions (arteritides)
   1) Infectious arteritides
      a) Septic embolism
      b) Syphilitic arteritis
      c) Pyogenic arteritis
      d) Tuberculous arteritis
      e) Infectious arteritides of other types
      f) Complications of infectious arteritides
         (1) Thrombosis
         (2) Hemorrhage
         (3) Scar
         (4) Aneurysm (mycotic aneurysm)
   2) Noninfectious arteritides
      a) Periarteritis nodosa
      b) Lupus erythematosus disseminatus
      c) Thrombotic microangiopathy (thrombotic thrombopenia, TTP)
      d) Rheumatic arteritis
      e) Cranial arteritis (temporal arteritis)
      f) Pulaseless disease (Takayasu’s disease)
      g) Noninfectious arteritides of other types (including allergic or hypersensitivity arteritis, etc.)
      h) Complications of noninfectious arteritides
         (1) Thrombosis
         (2) Hemorrhage
         (3) Scar
         (4) Aneurysm
   c. Trauma and physical agents
      1) Trauma to artery due to external forces, bony anomalies, fractures, dislocations, degenerative bone disease, fibrosis
         a) Local effects
            (1) Extramural hemorrhage
            (2) Intramural hemorrhage
               (a) With dissection
            (3) Thrombosis
            (4) Stenosis
         b) Remote effects
            (1) Fat embolization
            (2) Bone marrow embolization
            (3) Air embolization
      2) Trauma due to angiography
         a) Intramural hemorrhage
            (1) With dissection
         b) Extramural hemorrhage
         c) Rupture of atherosclerotic plaque
            (1) Atheromatous embolization
            (2) Thrombosis
         d) Embolization due to foreign materials (cotton fibers, etc.)
      3) Trauma due to cardiac catheterization and other intra-arterial diagnostic and therapeutic procedures
         a) Intramural hemorrhage
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b) Rupture of atherosclerotic plaque
   (1) Atheromatous embolization
 c) Thrombosis
d) Embolization due to foreign materials (cotton fibers, etc.)

4) Trauma due to surgery
   a) Reconstructive and reparative arterial surgery
      (1) Embolization by thrombotic fragments
      (2) Atheromatous or tissue embolization
      (3) Embolization due to foreign materials (cotton fibers, etc.)
      (4) Thrombosis
      (5) Thrombosis in arterial anastomosis
      (6) Thrombosis in arterial grafts
         (a) Natural grafts
         (b) Synthetic grafts
      (7) Aneurysm
      (8) Stenosis
   b) Other surgical procedures
      (1) Occlusion (ligation, clamp, etc.)
      (2) Rupture or accidental division
      (3) Intramural hemorrhage
         (a) With dissection
      (4) Rupture of atherosclerotic plaque
         (a) Atheromatous embolization
      (5) Thrombosis

5) Trauma of artery due to brain herniation
   (transventorial, subfalcial, foramen magnum, etc.)
   a) Thrombosis
   b) Hemorrhage

6) Radiation effects
   a) Post-irradiation thrombosis
   b) Post-irradiation scar (fibrosis)

d. Arterial lesions due to blood dyscrasias
   1) Thrombosis
      a) Polycythemia vera
      b) Secondary polycythemia (specify cause)
      c) Hemoglobinopathy (specify type)
      d) Other
   2) Hemorrhage
      a) Leukemia (specify type)
      b) Hemoglobinopathy (specify type)
      c) Hypoprothrombinemia
      d) Other

e. Arterial lesions associated with metabolic abnormalities
   1) Atherosclerosis [See IV.A.1.1.1]
      a) Familial hypercholesterolemia
      b) Other (including diabetes mellitus, hyperthyroidism, etc.)
   2) Thrombosis
      a) Dehydration
      3) Calcification
         a) Hypoparathyroidism
f. Arterial lesions associated with drug toxicity, idiosyncrasy and unknown effects
   1) Thrombosis
      a) Ergot derivatives
      b) Progestational agents (?)
      c) Methysergide (?)
      d) Others (specify)
   2) Hemorrhage
      a) Anticoagulants (specify)
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b) Sympathomimetic amines (specify)
c) Heavy metals (arsenic, etc.; specify)

3) Calcification
   a) Hypervitaminosis D

g. Arterial embolism due to cardiac disease and diseases of extracerebral vessels
   1) Cardiac arrhythmias (specify basic disease)
   2) Valvular disease (endocarditis)
      a) Septic (specify)
      b) Aseptic (specify)
   3) Myocardial infarction
   4) Atherosclerosis plus thrombosis
   5) Ulcerated atheroma
   6) Paradoxic embolism
      a) Congenital heart disease
      b) Systemic venous thrombosis
         (1) Aseptic
         (2) Septic
   7) Pulmonary venous thrombosis

h. Arterial lesions associated with neoplastic disease
   1) Thrombosis associated with neoplasm (specify)
      a) Intracranial neoplasm (primary or secondary)
      b) Extracranial neoplasm
   2) Hemorrhage associated with neoplasm (specify)
      a) Intracranial neoplasm (primary or secondary)
      b) Extracranial neoplasm
   3) Embolism associated with neoplasm (specify)
      a) Intracranial neoplasm (primary or secondary)
      b) Extracranial neoplasm

i. Arterial lesions due to unknown causes
   1) Atherosclerosis
      a) Atherosclerotic stenosis
         (1) Thrombosis
      b) Atherosclerotic occlusion
         (1) Thrombosis
      c) Ulceration of atherosclerotic plaque
         (1) Atheromatous embolization
      d) Atherosclerotic dilatation, ectasia or aneurysm (fusiform)
         (1) With rupture
      e) Atherosclerotic hemorrhage
      f) Intramural hemorrhage in plaque
      g) Calcification of atherosclerotic plaque
   2) Mönckeberg’s sclerosis (calcific medial arteriosclerosis)
   3) Mineralization (ferrugination, calcification, siderocalcific change) in parenchymal arteries (and arterioles) of central nervous system (CNS)
      a) Nonfamilial
      b) Familial calcification ("of basal ganglia")
      c) Other
   4) Fibromuscular dysplasia
   5) Thromboangiitis obliterans (Buerger’s disease) (?)
   6) Hemorrhagic dissection of arterial wall
      a) Cystic medial necrosis of aorta
      b) Dissection of other arteries

j. Arterial and arteriolar lesions associated with hypertension (specify type, etiology or associated disease)
   1) Atherosclerosis (arteriosclerosis) [See IV.A.1.i.1)]
   2) Hyaline medial degeneration with or without fibrinoid change
      a) With miiliary intracerebral aneurysm (?)
   3) Other degenerative changes (specify)
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2. Veins (and venules, when applicable)
   a. Congenital, developmental and inherited lesions
      1) Anomaly of veins (fetal form)
      2) Anomaly of veins (unspecified)
      3) Aneurysm of veins, congenital (phlebectasia)
         a) Ruptured aneurysm, congenital
   b. Inflammatory lesions (phlebitides)
      1) Infectious phlebitides
         a) Septic phlebitis
            (1) With thrombosis
         b) Tuberculous phlebitis
      2) Noninfectious phlebitides
         a) Thrombotic microangiopathy (thrombotic thrombopenia; TTP)
   c. Trauma and physical agents
      1) Trauma to vein due to external forces, bony anomalies, fractures, dislocations, degenerative bone disease, fibrosis
         a) Local effects
            (1) Extramural hemorrhage
            (2) Intramural hemorrhage
               (a) With dissection
            (3) Thrombosis
         b) Remote effects
            (1) Fat embolization
            (2) Bone marrow embolization
            (3) Air embolization
      2) Trauma due to angiography
         a) Intramural hemorrhage
            (1) With dissection
         b) Extramural hemorrhage
         c) Thrombosis
            (1) With embolization
         d) Embolization due to foreign materials (cotton fibers, etc.)
      3) Trauma due to intravenous procedures, diagnostic and therapeutic
         a) Intramural hemorrhage
         b) Extramural hemorrhage
         c) Thrombosis
            (1) With embolization
         d) Embolization due to foreign materials (cotton fibers, etc.)
      4) Trauma due to surgery
         a) Surgery involving veins
            (1) Embolization by thrombotic fragments
            (2) Tissue embolization
            (3) Embolization due to foreign materials (cotton fibers, etc.)
            (4) Thrombosis
            (5) Thrombosis in venous anastomosis
            (6) Thrombosis in venous grafts
               (a) Natural grafts
               (b) Synthetic grafts
         b) Other surgical procedures
            (1) Occlusion (ligation, clamp, etc.)
            (2) Rupture or accidental division
            (3) Intramural hemorrhage
            (4) Thrombosis
               (a) With embolization
      5) Trauma of veins due to brain herniation (transtentorial, subfalcial, foramen magnum, etc.)
         a) Thrombosis
         b) Hemorrhage
      6) Radiation effects
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a) Post-irradiation thrombosis
b) Post-irradiation scar (fibrosis)
d. Venous lesions due to blood dyscrasias
   1) Thrombosis
      a) Polycythemia vera
      b) Secondary polycythemia (specify cause)
      c) Hemoglobinopathy (specify type)
      d) Other
   2) Hemorrhage
      a) Leukemia (specify type)
      b) Hemoglobinopathy (specify type)
      c) Hypoprothrombinemia
      d) Other
e. Venous lesions associated with metabolic abnormalities
   1) Thrombosis
      a) Dehydration
   2) Calcification
      a) Hypoparathyroidism
f. Venous lesions associated with drug toxicity, idiosyncrasy and unknown effects
   1) Thrombosis
      a) Progestational agents (?)
      b) Others (specify)
   2) Hemorrhage
      a) Anticoagulants (specify)
      b) Heavy metals (specify)
   3) Calcification
      a) Hypervitaminosis D
g. Venous lesions associated with neoplastic disease
   1) Thrombosis associated with neoplasm (specify)
      a) Intracranial neoplasm (primary or secondary)
      b) Extracranial neoplasm
   2) Hemorrhage associated with neoplasm
      a) Intracranial neoplasm (primary or secondary)
      b) Extracranial neoplasm
   3) Embolism associated with neoplasm (specify)
      a) Intracranial neoplasm (primary or secondary)
      b) Extracranial neoplasm
h. Venous lesions due to unknown cause
   1) Phlebosclerosis
   2) Phlebolith
   3) Mineralization (ferrugination, calcification, siderocalcific change) in parenchymal vein (and
      venules of CNS)
      a) Nonfamilial
      b) Familial calcification (“of basal ganglia”)
      c) Other
   4) Varix
3. Capillaries
   a. Inflammatory lesions
      1) Infectious capillary purpura
      2) Noninfectious capillary lesions
         a) Thrombotic microangiopathy (thrombotic thrombopenia; TTP)
         b) Allergic capillary purpura
   b. Trauma and physical agents
      1) External trauma
         a) Petechiae
      2) Trauma by adjacent structures
         a) Petechiae
      3) Remote effects of external trauma
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4) Trauma due to angiography
   a) Petechiae due to radiopaque contrast medium
   b) Capillary thrombosis due to radiopaque contrast medium
   c) Petechiae due to atheromatous embolization
   d) Petechiae due to embolization by foreign materials (cotton fibers, etc.)
5) Trauma due to cardiac catheterization and other diagnostic and therapeutic intravascular procedures
   a) Petechiae due to atheromatous embolization
   b) Petechiae due to embolization by foreign materials (cotton fibers, etc.)
6) Trauma due to surgery
   a) Petechiae
7) Trauma of capillaries due to brain herniation (transmetentorial, subfalcral, foramen magnum, etc.)
   a) Petechiae
8) Heat stroke (sunstroke)
   a) Petechiae with heat stroke
9) Radiation effects
   a) Post-irradiation petechiae
   b) Post-irradiation thrombosis
   c) Post-irradiation fibrosis

c. Capillary lesions due to blood dyscrasias
   1) Thrombosis
      a) Polycythemia vera
      b) Secondary polycythemia (specify cause)
      c) Hemoglobinopathy (specify type)
      d) Other
   2) Petechiae
      a) Hemophilia
      b) Leukemia (specify type)
      c) Hemoglobinopathy (specify type)
      d) Hypoprothrombinemia
      e) Other

d. Capillary lesions associated with metabolic abnormalities
   1) Thrombosis
      a) Dehydration
   2) Petechiae (capillary purpura) due to metabolic disturbance (specify)
   3) Calcification
      a) Hypoparathyroidism
      b) Other
   4) Capillary proliferation
      a) Hyperoxia
      b) Hypoxia

e. Capillary lesions associated with drug toxicity, idiosyncrasy and unknown effects
   1) Thrombosis
      a) Progestational agents (?)
      b) Other (specify)
   2) Petechiae
      a) Allergic capillary purpura [See II.C.2.a.3]
      (specify drug or chemical)
      b) Anticoagulants (specify)
      c) Sympathomimetic amines (specify)
      d) Heavy metals (arsenic, etc., specify)
   3) Calcification
      a) Hypervitaminosis D

e. Capillary lesions due to cardiac disease and diseases of extracerebral vessels
   1) Petechiae with embolism [See II.A.2.c.]
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g. Capillary lesions associated with neoplastic disease
   1) Thrombosis associated with neoplasm [See II.A.8.]
   2) Petechiae (hemorrhage) [See II.A.8.]

h. Capillary lesions due to unknown cause
   1) Idiopathic brain purpura
   2) Mineralization (ferrugination, calcification, siderocalcific change) in capillaries of CNS
      a) Nonfamilial
      b) Familial calcification ("of basal ganglia")
      c) Other

i. Capillary lesions associated with hypertension (specify type, etiology, or associated disease)
   1) Hyalinization and fibrosis of capillary

4. Combined arterial, venous and capillary abnormalities
   a. Congenital, developmental and inherited lesions
      1) Arteriovenous fistula, congenital
      2) Arteriovenous fistula due to ruptured congenital aneurysm
      3) Vascular malformations (hamartomas, angiomas)
         a) Telangiectasis
         b) Cavernous (venous) angioma (hemangioma)
         c) Arteriovenous angioma
      4) Vascular malformations with the phakomatoses
         a) Lindau's disease
         b) Sturge-Weber's disease
         c) Others

b. Inflammatory lesions
   1) Infectious
      a) Arteriovenous aneurysm due to infection
      b) Calcification of vessels due to congenital rubella

c. Trauma
   1) Trauma due to external forces, bony anomalies, fractures, dislocations, degenerative bone disease, fibrosis
      a) Arteriovenous fistula, traumatic

d. Metabolic abnormalities
   1) Avitaminosis (specify type)

e. Lesions due to unknown causes
   1) Arteriovenous fistula due to ruptures
   2) Arteriosclerotic aneurysm

B. Pathological alterations in brain
   1. Infarction (pale, hemorrhagic and mixed)
      a. Without vessel stenosis or occlusion
      b. With arterial stenosis or occlusion due to:
         1) Congenital, developmental and inherited lesions of arteries [See IV.A.1.a.]
         2) Inflammatory lesions of arteries [See IV.A.1.b.]
         3) Trauma and physical agents [See IV.A.1.c.]
         4) Blood dyscrasias [See IV.A.1.d.]
         5) Metabolic abnormalities [See IV.A.1.e.]
         6) Drugs, etc. [See IV.A.1.f.]
         7) Cardiac and extracerebral vessel disease [See IV.A.1.g.]
         8) Neoplastic disease [See IV.A.1.h.]
         9) Unknown causes (chiefly atherosclerosis) [See IV.A.1.i.]
        10) Hypertension [See IV.A.1.j.]
        11) Other
      c. With venous stenosis or occlusion due to:
         1) Congenital, developmental and inherited lesions [See IV.A.2.a.]
         2) Inflammatory lesions [See IV.A.2.b.]
         3) Trauma and physical agents [See IV.A.2.c.]
         4) Blood dyscrasias [See IV.A.2.d.]
         5) Metabolic abnormalities [See IV.A.2.e.]
         6) Drugs, etc. [See IV.A.2.f.]

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7) Neoplastic disease [See IV.A.2.g.]
8) Unknown causes [See IV.A.2.h.]
d. With capillary lesions [See IV.A.3.]
e. With combined arterial, venous and capillary lesions [See IV.A.4.]

2. Hemorrhage (intracranial, intracerebral, etc.)
a. Without vessel type identified
b. Of arterial origin
   (list as under Pathological Alterations in vessels, or use anatomical and etiological terms as follows)
   1) Anatomical site
      a) Intracerebral
         (1) With hypertension
         (2) Without hypertension
      b) Subarachnoid
      c) Subdural
      d) Intraventricular
   2) Etiology
      a) Congenital, developmental and inherited lesions of arteries [See IV.A.1.a.]
      b) Inflammatory lesions of arteries [See IV.A.1.b.]
      c) Trauma and physical agents [See IV.A.1.c.]
      d) Blood dyscrasias [See IV.A.1.d.]
      e) Metabolic abnormalities [See IV.A.1.e.]
      f) Drugs, etc. [See IV.A.1.f.]
      g) Cardiac and extracerebral vessel disease [See IV.A.1.g.]
      h) Neoplastic disease [See IV.A.1.h.]
      i) Unknown causes [See IV.A.1.i.]
      j) Hypertension [See IV.A.1.j.]
      k) Other
   c. Of venous origin
      1) Congenital, developmental and inherited lesions of arteries [See IV.A.2.a.]
      2) Inflammatory lesions [See IV.A.2.b.]
      3) Trauma and physical agents [See IV.A.2.c.]
      4) Blood dyscrasias [See IV.A.2.d.]
      5) Metabolic abnormalities [See IV.A.2.e.]
      6) Drugs, etc. [See IV.A.2.f.]
      7) Neoplastic disease [See IV.A.2.g.]
      8) Unknown causes [See IV.A.2.h.]
d. With capillary lesions [See IV.A.3.]
e. With combined arterial, venous and capillary lesions [See IV.A.4.]

Part V. Clinical Phenomena (History, Physical Examination, Laboratory Examination, Roentgen Examination, Other)

INTRODUCTION

Part V (Clinical Phenomena) is designed to accomplish classification or codification of material from the patient’s history, physical examination, laboratory examination and procedures which include angiography, cerebral blood flow determinations and many others. The physician caring for an individual patient may not wish to formally use this portion of the Classification but may find it valuable as a form of “check list” or “reminder” concerning symptoms, physical signs and many laboratory and special procedures. Epidemiologists and clinical investigators will find special aid from Part V for assistance in constructing protocols and forms for systematically recording clinical phenomena.

The subdivisions on Demography, Family History, and Past History are self-explanatory.

In using the subdivision “4. Present illness,” it is important to refer to the definitions in Part I (Clinical Stage) — definitions which clearly describe the meaning of such categories as Transient Attacks (TIA) and Actively Changing Neurological Deficit (progressing stroke). The definition of TIA includes comments concerning individual symptoms (i.e., vertigo, etc.) which are not to be designated as TIA. Although the arterial system involved is classified under Part III (Anatomy), the anatomical site of TIA, etc., is also included in Part V as a convenience to the person using the Classification.

The first two categories of subdivision “B.
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Physical examination" are traditional ones: "1. General" and "2. Neurological." However, the third category "Vascular" includes much new material which allows classification of the results of the neurovascular examination. In the "Outline" portion of the document is descriptive material concerning bruits, the findings from ophthalmoscopic examination and the technique of ophthalmodynamometry.

The subdivision "C. Laboratory examination" contains many standard types of test. In the appropriate portion of the "Outline" are comments concerning the relative importance of many of these tests. Certain arbitrary decisions are necessary, i.e., placing "Echoencephalography" and "Brain scan" in this category rather than under "E. Special procedures."

Because of the importance of cranial angiography, a special subdivision "D. Roentgen examination" is provided. Discussion of indications for angiography is included in the appropriate portion of the "Outline."

"E. Special procedures" provides a place for listing a number of items which will be done only in centers where special clinical investigations of cerebrovascular disease are done. Computerized axial tomography* (computerized tomography, computed tomography) is placed first; this appears to be an important noninvasive technique for the differential diagnosis of cerebral infarction, intracerebral hemorrhage and intracranial neoplasm, and is becoming available in more and more localities.

Part V. Clinical Phenomena (History, Physical Examination, Laboratory Examination, Roentgen Examination, Other)

A. History

1. Demographic
   a. Sex
   b. Age (specify date of birth) (day, month, year)
   c. Race, ethnic origin, and other related information
   d. Occupation and/or socioeconomic status
   e. Education
   f. Environment (urban or rural)

2. Family history
   a. Cerebrovascular disease
   b. Hypertension
   c. Diabetes
   d. Coronary artery disease
   e. Other vascular disease
   f. Other

3. Past history
   a. Hypertension
   b. Vascular disease
      1) Migraine
      2) Raynaud’s
      3) Arteriosclerosis obliterans
      4) Noninfectious arteritis (specify type)
      5) Familial hypercholesterolemia
      6) Syphilis
      7) Other (specify)
   c. Diabetes
   d. Heart disease
      1) Coronary
      2) Congenital
      3) Rheumatic
      4) Hypertensive
      5) Other (specify)
   e. Hematological disorders (specify)
   f. Metabolic disease (specify)
   g. Chronic alcoholism
   h. Obesity
   i. Smoking habits
   j. Drugs (specify)
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k. Pregnancy
l. Head injury
m. Cerebrovascular event(s) (specify according to definition in Part I) (do not include data thought to be dynamically related to patient's current problem)
n. Other neurological disease
o. Other past illness (specify)

4. Present illness
a. Transient attacks (transient ischemic attacks) [See I.B.1.]
   1) Number (specify)
   2) Date of first attack
   3) Date of most recent attack
   4) Frequency
      a) Becoming more frequent
      b) Becoming less frequent
      c) No change
   5) Duration (describe)
   6) Precipitating factors
      a) Position of body (describe)
      b) Change of position of head, neck or arm (describe)
      c) Exercise (describe)
      d) Hypotension
      e) Cardiac arrhythmia
      f) Emotion
      g) Other (specify)
   7) Content of typical transient ischemic attack (symptoms)
      The following list of symptoms is not meant to be inclusive of all neurological phenomena. The list may be used as a framework or check list. The content of a typical attack or of several attacks may need to be described in narrative form and an indication of sequence of occurrence of several symptoms and of their severity may need to be indicated (specify site, type and degree). These items will be applicable for all subsequent categories of clinical stage.
      a) Impaired consciousness
      b) Confusion
      c) Amnesia
      d) Visual disturbance
         (1) Unilateral
         (2) Bilateral
         (3) Homonymous
         (4) Diplopia
         (5) Variable
      e) Vertigo or dizziness
      f) Dysarthria
      g) Dysphasia — aphasia
      h) Auditory disturbance(s)
      i) Motor dysfunction
         (1) Mono
         (2) Hemi
         (3) Other
      j) Dysphagia
      k) Sensory disturbance
         (1) Mono
         (2) Hemi
         (3) Other
      l) Impaired equilibrium
      m) Headache
      n) Head noise (bruit, etc.)
      o) Impaired bladder function
      p) Impaired bowel function
      q) Convulsions

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1) Focal
2) Generalized
r) Other
8) Arterial system
a) Carotid
b) Vertebrobasilar
c) Mixed
d) Uncertain
b. Actively changing neurological deficit (progressing stroke)
1) Time of onset (specify date and hour)
2) Duration of change (progression) (specify time)
3) Predisposing factors [See II.E.]
4) Precipitating factors [See V.A.4.a.6]
5) Symptoms [See V.A.4.a.7]
6) Arterial system
c. Prolonged neurological deficit (reversible ischemic neurological deficit [RIND] and completed stroke)
Please make a distinction between RIND and long course as defined in I.B.3.
1) Time of onset (specify date and number of minutes or hours from first symptom to maximum neurological deficit)
2) Precipitating factors [See V.A.4.a.6]
3) Symptoms [See V.A.4.a.7]
4) Arterial system [See III]
a) Carotid
b) Vertebrobasilar
c) Mixed
d) Uncertain
B. Physical examination
1. General
a. Temperature
b. Respiration
1) Rate
2) Rhythm
3) Airway (tongue, secretion, cough, ventilation)
c. Pulse
1) Rate
2) Rhythm
d. Blood pressure Left________ Right________
Postural change (specify)
e. Cardiac status (describe if abnormal)
f. Bowel status (describe if abnormal)
g. Bladder status (describe if abnormal)
2. Neurological
a. State of consciousness (It is recognized that there are many ways of describing this item. It is important that the user of this Classification construct a standard system of description of state of consciousness, such as:)
1) Normal
2) Semi-stupor (lethargy — response slowed)
3) Stupor (appropriate response to verbal stimulus)
4) Deep stupor (purposeful response to noxious stimulus)
5) Semicoma (nonspecific response to pain)
6) Coma (above reflexes present — no response to pain)
7) Deep coma (absent DTRs, pupillary reactions and corneal reflexes — no response to pain)
b. Mental function
1) Normal
2) Impairment of intellect
a) Memory
b) Recall
c) Calculation
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d) Judgment
e) Orientation
f) Other

3) Speech and language
   a) Dysarthria
   b) Dysphasias (describe) ("aphasia" is often used to describe a speech defect which would more correctly be termed "dysphasia")
   c) Agnosia
      (1) Visual
      (2) Auditory
d) Dyspraxia
e) Dysgraphia
f) Dyslexia
g) Other (palilalia, etc.)

4) Disorders of emotion (affect) (describe)

5) Distortions of thought content (describe)

c. Cranial nerves and related functions
   1) Visual acuity
   2) Optic fundus — ischemic retinopathy [See V.B.3.d.6), etc.]
   3) Visual fields
   4) Pupillary reactions
   5) Ocular movements
      a) Muscle weakness (describe)
      b) Nystagmus
c) Gaze impairment
d) Internuclear ophthalmoplegia
e) Optokinetic responses
   6) Sensations on face including corneal reflex
   7) Muscles of mastication including jaw jerk
   8) Muscles of facial expression
      a) Supranuclear
      b) Nuclear and peripheral
   9) Auditory function
   10) Vestibular function
   11) Pharyngeal and laryngeal muscles including gag reflex
   12) Sternomastoid and trapezius muscles
   13) Tongue muscles including alternate motion rate (AMR)
   14) Miscellaneous
      a) Horner's
      b) Pseudobulbar palsy
c) Palatal myoclonus
d) Singultus

15) Other
d. Sensation (exclusive of special senses) (describe)
   1) Vibratory
   2) Two-point discrimination
   3) Sense of position
   4) Pain
   5) Light touch
   6) Temperature
   7) Stereognosis
   8) Deep pressure
   9) Double simultaneous stimulation
   10) Figure writing (graphesthesia)
   11) Localization of touch
   12) Texture, appreciation of weight, etc.

c. Reflexes
   1) Muscle stretch
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a) Biceps
b) Triceps
c) Brachioradialis
d) Hoffmann
e) Knee
f) Hamstring
g) Ankle
h) Other

2) Cutaneous
   a) Abdominal
   b) Other

3) Miscellaneous
   a) Plantar (Babinski)
   b) Grasp
c) Snout
d) Sucking
e) Other

f. Motor function
   1) Gait (describe)
   2) Posture (describe)
   3) Strength (grade each muscle if appropriate)
      a) Normal
      b) Mild impairment of strength (fair). Full range of motion against resistance
      c) Moderate impairment of strength (poor). Full range of motion antigravity
      d) Severe impairment of strength (combine polio classification “trace” and “poor”). Full range of
         motion with gravity eliminated
      e) No strength

4) Coordination (limb)
   a) Ataxia (faulty synergistic action throughout movement)
   b) Dysmetria (faulty in measuring to the object)
   c) Rebound phenomena

5) Praxis and alternate motion rate (AMR) (describe)

6) Adventitious movements (describe)

7) Other phenomena disturbing motor function (describe)
   a) Contractures
   b) Muscle tone

8) Evaluation of the function of groups of muscles
   a) Upper extremity (patient sitting)
      (1) Normal (4+) (No impairment of function)
      (2) Good (3+) (Mild impairment of function) Normal except for skilled acts and endurance
      (3) Fair (2+) (Moderate impairment of function) Shoulder abduction and extension of elbow +
         grasp and release of hand + supination-pronation of forearm through full range of motion
         but with impaired speed, coordination and strength
      (4) Poor (1+) (Severe impairment of function) Has shoulder abduction and extension but not
         through full range of motion
      (5) Bad (0) (No function)
   b) Lower extremity
      (1) Normal
      (2) Mild impairment of function
         a) Knee extension normal
         b) Hip and knee flexion good
         c) Dorsiflexion of ankle poor
         d) Walks well — lacks full skill and endurance
      (3) Moderate impairment of function
         a) Knee extension present
         b) Knee and hip flexion poor
         c) Dorsiflexion of ankle out
         d) Walks — much trouble
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(4) Severe impairment of function
   (a) Some range of motion (see above)
   (b) Cannot walk
(5) No function
   c) Trunk

3. Vascular (largely neurovascular)
   a. Inspection (specify abnormality)
   b. Palpation (specify abnormalities; tenderness, symmetry, thrills, etc.)
      1) Pulses (specify grade: 0 = absent to 4 = normal)
         a) Carotid
         b) Temporal
         c) Brachial
         d) Radial
         e) Femoral
         f) Popliteal
         g) Posterior tibial
         h) Dorsal pedis
         i) Other (specify)
   2) Carotid compression test*
   c. Auscultation — bruits [See Outline]
      1) Location (specify)
      2) Grade 1 to 6 (1 = barely discernible with stethoscope, 6 = audible without stethoscope)
      3) Pitch (high, medium, low)
      4) Quality (rough, soft, smooth, etc.)
      5) Duration (ss = short systolic, ls = long systolic, sd = systolic-diastolic, c = continuous)
   d. Ophthalmoscopy [See Outline]
      1) Retinal embolus
         a) Cholesterol
         b) Platelet-fibrin
         c) Mixed (red-white)
         d) Calcific
         e) Other
      2) Retinal hemorrhage (specify site)
      3) Hypertensive arterial changes
      4) Cotton-wool patches
      5) Microaneurysms
      6) Ischemic retinopathy
      7) Papilledema
      8) Retinal edema
      9) Other (specify)
   e. Ophthalmodynamometry (record as listed) [See Outline]
      1) Position of patient
         a) Sitting
         b) Lying
         c) Standing
      2) Value (systolic/diastolic)
         a) Right
         b) Left
   f. Neurological signs produced by position
      1) Head turning
      2) Standing up
      3) Sitting up
   g. Veins (specify pertinent abnormalities)

*The value of carotid compression tests relative to the information gained compared to the risk to the patient limits the use of the test to laboratories where careful monitoring of the patient (with ECG, EEG, etc.) is possible.
CLASSIFICATION AND OUTLINE OF CEREBROVASCULAR DISEASES II

C. Laboratory examination
1. Urinalysis (specify abnormality)
2. Blood
   Hemoglobin, red blood count, white blood count, hematocrit, viscosity, volume, cholesterol, lipids, serum enzymes, sugar, blood urea nitrogen, uric acid, creatinine, sedimentation rate, coagulation studies, platelet count, total thyroxine, serologic test for syphilis, L. E. cell preparation
3. Cerebrospinal fluid (specify abnormality)
4. Electrocardiography (specify abnormality)
5. Echoencephalography (specify abnormality)
6. Brain scan
   a. Static (specify isotope) (specify abnormality)
   b. Rapid serial scintigraphy (gamma camera)

D. Roentgen examination
1. Chest
   a. Normal
   b. Abnormal (specify)
2. Head
   a. Normal
   b. Abnormal (specify)
3. Angiography (cranial)
   a. Anesthesia
      1) General
      2) Local
   b. Media (specify)
   c. Site(s) of injection (specify)
      1) Catheter (specify type)
      2) Pump (type)
   d. Vessels visualized
      1) Extracranial (specify)
      2) Intracranial (specify)
   e. Abnormalities
      1) Extracranial (specify)
      2) Intracranial (specify)
   f. Complications
      1) Extracranial
         a) Cardiac
         b) Airway
         c) Hypotension
         d) Bleeding
         e) Other
      2) Intracranial
         a) Focal neurological deficit (describe)
         b) Convulsive events (describe)

E. Special procedures
1. Computerized axial tomography (computerized tomography, computed tomography) [See Outline]
2. Cerebral blood flow (specify method) (list results) (describe complications)
3. Retinal circulation time (specify method and results) (fluorescein)
4. Thermography and thermometry (specify method and results)
5. Retinal photography (specify method and results)
6. Tilt-table study (specify methods and results)
7. Phonocraniography (specify methods and results)
8. Electronystagmography
9. Doppler blood flow estimation (specify method, site, results)
10. Ocular plethysmography (specify method and results)
11. Cranial impedance plethysmography (rheoencephalography) (specify method and results)
12. Other
The patient must be able to recognize the demands is defined as the execution of set tasks. The design of a circum-
tances of the life-setting. The nature of the task, to select the appropriate ac-
aturally determined by environmental or situational variables that determine the placement status. Among

**Activities of daily living (ADL) — a battery of tasks designed to provide an index of ability to perform self-sustaining activities that meet personal requirements in each day's life-experience. These essential activities are basic in everyone's day, relatively independent of geographic locale, physical environment, or social situation. They include such common activities as dressing, washing, eating and traveling from place to place; they involve fundamental actions of body and limb movement, sitting balance, changing body position, standing, reaching, grasping, holding, and the like.**

**Avocational activities — those active pursuits of living not directly related to one's primary life work from which the individual gains significant participatory, creative or productive satisfaction. These are not essential requirements of everyday living but have value in bringing purpose and fulfillment to the individual, his family and circle of friends. They involve multifaceted behavior patterns and at times employ complex hierarchies of physical and mental activities. Avocational activities include such pursuits as hobbies, travel, recreation, socializing and the like.**

**Occupation — the primary life-work to which the individual devotes the major portion of time, skill, energy, thought and effort and from which the individual derives role and status, physical and mental satisfaction and economic support. In addition to recognized modes of remunerative employment, occupation includes homemaking, student life, retirement and other primary life-pursuits. In this Classification all such pursuits engaged in at the time of onset of stroke are considered the primary occupation.**

**PLACEMENT**

The goal of care is ultimate return of the recovered patient to an optimal physical, mental, social, vocational and economic condition consistent with the patient's remaining performance abilities and requirements for continued health maintenance. Suitable physical environment, living arrangements, and social, economic and health care support must be selected. Medical status, performance ability and conditions in available environmental settings are variables that determine the placement status. Among the critical considerations leading to optimal placement are the suitability of the architecture and surrounding environment, the kind and frequency of personal supervision and direction required, and the level of requirement for continuing medical-nursing care. Possibilities for placement vary widely but may include placement in full competitive employment, placement in sheltered or selected work situations, resumption of homemaker duties, retirement from the work market, independent living in personal residence with full management responsibility, home care with...
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family supervision, home care with outside assistance, domiciliary care, convalescent care, custodial care, or continued rehabilitation center or hospital care.

Part VI. Status of Patient (Performance and Placement)

PERFORMANCE

Class I. No Significant Impairment — fully independent acts of daily living (ADL), pursues usual avocational activities, and returns to previous living site and occupation without modification.

Class II. Mildly Impaired — semidependent (requiring some assistance) in activities of daily living, and/or slight restriction of avocational activities, and/or able to return to previous occupation with some modification of the latter.

Class III. Moderately Impaired — semidependent (requiring lifting assistance) in activities of daily living, and/or considerable restriction of avocational activities, and/or unable to return to previous occupation.

Class IV. Severely Impaired — fully dependent in conduct of activities of daily living, and/or unable to participate in avocational activities, and/or unable to carry out any occupation.

PLACEMENT

Class A. No Limitation.

Class B. Mild Limitation — requires occasional supervision, and/or modified environment and/or occasional medical care.

Class C. Moderate Limitation — requires much supervision, and/or physical assistance or outside helpers and/or regularly available medical care.

Class D. Severe Limitation — requires constant or nearly constant attendance and/or immediately available medical-nursing care.

(Continued on p. 594)
Outline of Cerebrovascular Diseases

Vascular disorders affecting the brain have a variety of components:

1. The basic pathophysiological process which consists of: decreased perfusion pressure because of a cardiac or systemic circulatory problem, abnormalities of the blood (polycythemia, etc.) or abnormalities directly impairing the vessel's transport of blood (atherosclerosis, embolism, thrombosis, arteritis, etc.), singly or in a variety of combinations. As a result of these processes, the metabolic substrate for normal brain function is not supplied.

2. The pathophysiological change in brain parenchyma metabolism (commonly focal) which may be of short duration (as in a transient focal cerebral ischemic attack) and reversible, or may be infarction if impaired circulation persists, or may be hemorrhage if a vessel ruptures.

3. The neurological abnormality which results from the focal deficit in brain metabolism. The temporal profile of the neurological abnormality varies greatly — from a brief event (TIA) to permanent severe brain damage (completed stroke) producing hemiplegia, etc., or death.

The “Outline” is to assist all health professionals; it deals with the problems posed by individual patients and thus starts with “Clinical Stage.” The “Outline” contains a full definition of each category under “Clinical Stage” as well as mixing concepts from “Pathophysiological Mechanisms,” “Anatomy” and “Pathology” just as the physician must simultaneously mix these concepts as he attempts to solve the diagnostic and treatment dilemma presented by a patient.

Outline

Part I. Clinical Stage

The primary purpose of this section of the Classification is to provide a framework for the description of the current status of the patient in reference to the temporal profile of the disease without regard to other factors, such as etiology, pathology, neural deficit and the like, all of which are described in separate sections.

As the condition of the patient may be changing, sometimes rapidly, a static description presents certain problems, particularly as to the exact time when a patient is categorized. In general, a patient is placed into one of the categories at the time he is studied in sufficient detail so as to permit a reasonably certain clinical diagnosis. Because of the evolution of the deficit, it is likely that a patient will be in different categories at different times; in these instances a statement must be made to indicate the time of categorization. It is possible, also, that some deficits will be stable while other deficits are evolving; in these instances, appropriate comments will need to be made. By using more than one category, an accurate description of a complex temporal profile can be built (e.g., a patient who has TIAs followed by a completed stroke with a permanent residual neurological deficit and subsequently again has TIAs would be classified as I.B.1., I.B.3., and later I.B.1.).

It is obvious that an infinite number of variations can be discerned among the patterns of cerebrovascular disease; thus any classification is somewhat arbitrary. Furthermore, the purist may note some mixing of clinical description with pathological entities. The Committee recognized that the rate of change might be important but to describe this fully would lead to too complex a classification.

A. Asymptomatic

This category is for cerebrally asymptomatic individuals who are found to have evidence which may potentially be important as predisposing to future cerebrovascular disease. Such evidence is listed in detail in Part II. Some of the factors in this evidence have been termed the “Stroke Prone Profile.”

B. Focal cerebral dysfunction

This category refers to focal brain dysfunction regardless of the nature of the vascular pathology (e.g., ischemic disease, intracranial hemorrhage, arteritis).*

1. Transient ischemic attacks

These are episodes of temporary and focal cerebral dysfunction of vascular origin, rapid in onset (no symptoms to maximal symptoms in less than five minutes and usually less than a minute), which are variable in duration, commonly lasting from 2 to 15 minutes but occasionally lasting as long as a day (24 hours). The resolution or disappearance of each episode is swift (ordinarily a few minutes at most). A prolonged attack may take longer to clear. Each attack leaves no persistent neurological deficit. It is common practice to define these events as related to the carotid arterial system or the vertebrobasilar arterial system, meaning that the clinical locus of ischemia is in the customary distribution of one or the other of these arterial systems. There may be only one attack or there may be multiple attacks at varying intervals. One must recall that there are unusual instances which fall outside of this standard definition. This definition is constructed in arbitrary fashion in an attempt to provide a common basis for collecting groups of TIA patients. The contents of TIA are coded under Part V. Clinical Phenomena, 4. Present illness, a. Transient attacks.

*Migraine syndrome. The aura of migraine (e.g., scintillating scotoma) is commonly attributed to focal cerebral ischemia owing to vasoconstriction. In some instances more severe neurological deficits precede the headache and may include hemiparesis, dysphasia and homonymous hemianopia and in the great majority of instances these deficits are transitory, only rarely persisting long enough to produce a cerebral infarct. The temporal profile may be classified under “Clinical Stage” while the word “migraine” is under II.A.5.b.
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The typical history for a TIA in the carotid system is a swift (no symptoms to maximal symptoms in less than five minutes, usually less than two minutes) onset of:

1. Motor defect (weakness, paralysis, poor use, or clumsiness of one extremity or of both extremities on the same side).
2. Sensory defect (numbness including loss of sensation or paresthesias involving one or both extremities on the same side).
3. Aphasia (speech and/or language disturbance which may be only a minor defect or may be global and may or may not include difficulty in reading, writing, or performing calculations).
4. Loss of vision in one eye or in part of one eye when vision in both eyes was intact (amaurosis fugax).
5. Homonymous hemianopia.
6. Combinations of the above.

These clinical phenomena generally represent a decrease or absence of function. When there is a sensory event, it is commonly described as coming on all at once, that is, without a march.

The typical history of a TIA in the vertebrobasilar system is a swift (no symptoms to maximum symptoms in less than five minutes, usually less than two minutes) onset of:

1. Motor defect (weakness, clumsiness, or paralysis of any combination of extremities up to quadriplegia, sometimes changing from one side to another in different attacks).
2. Sensory defect (numbness, including loss of sensation or paresthesias in any combination of extremities including all four or involving both sides of the face or mouth. This is frequently bilateral trouble or the distribution may change from side to side in different attacks).
3. Loss of vision, complete or partial in both homonymous fields (bilateral homonymous hemianopia).
4. Homonymous hemianopia.
5. Ataxia, imbalance, unsteadiness, or dys equilibrium not associated with vertigo.
6. Either vertigo (with or without nausea and vomiting), diplopia, dysphagia, or dysarthria is not to be considered as a TIA when any of these symptoms occurs alone, but in combination with one another or with any of the above (numbers 1, 2, 3 and 4) the attacks should be considered a TIA.
7. Combinations of the above.

These clinical phenomena generally represent a decrease or absence of function. At times, the motor, sensory or visual defect constituting the content of a vertebrobasilar attack will be unilateral. It becomes difficult in such instances to make a distinction between whether the locus of ischemia is in the carotid arterial system or in the vertebrobasilar arterial system. In the list above, "drop attacks" is omitted. Fainting (syncope) is frequently confused with a "drop attack," so the latter should be included in the vertebrobasilar profile only when the patient's description of the "drop attack" is absolutely clear. The variety of manifestations included in the vertebrobasilar profile makes the potential pattern of symptoms considerably more variable and complex than that in the carotid system.

The diagnosis of TIA rests on the history of the attacks; the skill with which the history is taken and the interpretation of the history, except for those relatively few instances where the physician is with the patient at the time of the attack. The criteria for making the diagnosis will vary depending on whether an individual physician is working with an individual patient or whether the purpose is the screening of a population for TIA's. A problem is created, as in much of medical diagnosis, because of the relative weight or significance of some historical phenomena compared to other phenomena. The symptom "numbness" (mentioned above) is an example. If the question is, "Have you ever had a numb hand?" the answer from most adults will be "yes." This question is almost completely non-selective (non-diagnostic) and must be followed by a series of questions to establish the meaning and significance of the "numbness." In contrast, another phenomenon, when present, is simple and relatively much more significant than "numbness;" i.e., "Have you ever had painless blindness in one eye which came on very quickly (seconds) and lasted only a few minutes (5 to 20)?" is a question which, if answered "yes," is reasonably specific. Another similar but less specific question is "Have you ever had the sudden onset of a 5 to 20-minute duration attack of severe weakness of one side of the body (arm and leg)?" Another is "Have you ever suddenly lost the ability to speak (5 to 20 minutes' duration) or to understand the speech of others?" These questions illustrate the importance of understanding that different questions may have relatively different complexity and importance.

The matter of relative significance of different symptoms is important in the vertebrobasilar system as it is in the carotid system. For instance, if one asks the question, "Have you ever had any dizziness?," almost all adults will answer "yes." This question is almost completely non-selective (non-diagnostic) and if answered affirmatively must be followed by a series of direct and branching questions to establish the meaning and significance of the original phenomenon — "dizziness." A diagnosis of a TIA in the vertebrobasilar system should not be made on the basis of a history of a few minutes of vertigo as the only symptom. This is emphasized since vertigo is the most common symptom in the vertebrobasilar system; however, a diagnosis of vertebrobasilar TIA is made only when there is concurrently with vertigo (dizziness) an additional symptom or symptoms.

In some instances, patients with carotid system TIAs may have physical signs of appropriate arterial disease. These include diminished pulsation in the
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carotid artery, a bruit over the carotid artery or eye, emboli in the retinal vessels, or other signs of ischemic retinopathy and relative hypotension in the retinal artery as measured with the ophthalmodynamometer. These are only signs of arterial disease and may be present in the absence of a history of TIAs. In certain instances, bruits signifying compromise of flow in the innominate artery, either subclavian artery, or at the origin of either vertebral artery may be present; however, the absence or presence of such sounds does not weigh heavily in the diagnosis of vertebrobasilar TIA since it is again emphasized that the diagnosis is dependent upon the history of the attack, not upon morphological evidence of change in patterns of blood flow.

Certain symptoms may appear in a TIA in either arterial system. The most important of these are:
1. Dysarthria, if it occurs alone, and
2. Homonymous hemianopia, if it occurs alone.

The occurrence of certain symptoms in solitary fashion constitutes an attack which is an "uncertain TIA." An attack which consists solely of each of the following symptoms should be categorized as an uncertain TIA:
1. Vertigo alone
2. Dysarthria alone
3. Dysphagia alone
4. Diplopia alone

For the sake of clarity, the following symptoms, transient or prolonged, are not to be included as TIA:
1. Unconsciousness including syncope
2. Tonic and/or clonic activity
3. March of a sensory defect
4. Vertigo alone
5. Dysphagia alone
6. Dysarthria alone
7. Incontinence of bowel or bladder
8. Dizziness or wooziness alone
9. Loss of vision associated with alteration of consciousness
10. Focal symptoms associated with migraine
11. Scintillating scotomata
12. Confusion alone
13. Amnesia alone

The differential diagnosis of TIAs includes "hemiplegic" migraine, focal convulsive events (often due to neoplasm and producing either sensory or motor phenomena), Meniere's disorder, sensory phenomena associated with hyperventilation, and finally some unknown mechanism. The differentiation of "hemiplegic" migraine is a semantic and practical problem. In those instances where the aura of migraine is associated with a definitely focal neurological event, the latter may well be the result of transient focal cerebral ischemia but the implications are different than the usual TIA. To establish a diagnosis of the migraine association, there is ordinarily a positive family history, characteristic unilateral headache with nausea and sometimes vomiting, and onset of the attacks several decades ahead of the age at which TIAs commonly begin. Very careful history-taking ordinarily delineates the transient focal events associated with brain neoplasm from TIAs and this is also true of the other items in the differential diagnosis.

Many factors might be included to describe the characteristics of the change or rate of change in each individual attack or the pattern in many attacks. These factors are dealt with in I.B.2.

2. Actively changing neurological deficit
   This category refers to a patient whose neurological deficit is actively changing in amount during the period of observation (specify duration from time of onset). The deficit may be getting more severe or less severe.
   a. Improving
   b. Worsening (also known as "progressing stroke" or "stroke-in-evolution")

This category represents the common circumstance where focal ischemia is worsening and the process of infarction is beginning or extending. (In unusual instances slow onset bleeding may produce a similar temporal profile.) This subdivision is divided into those situations where the focal pathology is in the characteristic territory supplied by the carotid arterial system and where the supply is through the vertebrobasilar arterial system. As mentioned earlier, because of the evolution of the deficit, it is likely that patients will be in different categories at different times. For instance, a patient seen at 10 A.M. with mild left upper extremity weakness (history of onset at 9 A.M. the same day), re-examined at 11 A.M. and found to have paralysis of that extremity, would be classified as "progressing stroke," but if the deficit had disappeared at 11 A.M., the categorization would be "transient ischemic attack."

Description of the neurological findings most often characteristic of a focal deficit in specific arterial territories will be found under Part V.B.2.

3. Completed stroke (Prolonged neurological deficit)
   This category refers to a relatively stable neurological deficit, that is, during the period of observation for categorization (specify duration from time of onset), little or no change in the deficit is occurring.
   a. Duration more than 24 hours — less than three weeks (sometimes referred to as a reversible ischemic neurological deficit or RIND).
   b. Duration more than three weeks — often permanent (commonly known as "completed stroke").

THROMBOTIC INFARCTION

Thrombotic infarction is divided into those situations where there is infarction in the characteristic territory supplied by the carotid arterial system or where the supply is through the vertebrobasilar arterial system. Those infarctions occurring in the carotid arterial distribution often come on rather
Abruptly over a matter of many minutes to a very few hours. The neurological deficit may become maximum during sleep and, therefore, may be maximum when first discovered or may require several hours to a day to evolve. In a significant percentage of cases, there will have been warning TIAs. While there may be some head discomfort, violent headache is rare. There is relative preservation of consciousness and at times there may be rapid improvement. The cerebrospinal fluid (CSF) commonly remains clear and there is often associated evidence of atherosclerosis elsewhere in the coronary and peripheral vessels. The clinician searches for the presence of disorders commonly associated with atherosclerosis (hypertension, diabetes mellitus, gout, various forms of heart disease and xanthomatosis). The neurological examination reveals weakness or at times there may be rapid improvement. There is relative preservation of consciousness and at times there may be rapid improvement. The neurological examination reveals weakness or numbness limited to one side of the body. If the dominant hemisphere is involved, there may be aphasia or, which is more common, a dysphasia which will vary as far as its precise content is concerned. It is interesting to note that in this type of "stroke" the visual field defects will generally be homonymous, and it is rare for there to be simultaneous evidence of complete unilateral retinal ischemia and focal brain ischemia on the same side. As a clinician looks at the temporal profile of the disorder, he must attempt to decide whether the cerebral infarction is still progressing, as evidenced by an increasing neurological deficit, or whether the "stroke" is essentially completed, meaning that there is no further progression of the neurological deficit. This decision can be made only by relating the history of the preceding few minutes or hours to the existing findings or by picking up the temporal profile at the point where the patient is first seen and carefully re-examining the situation every few minutes or hours.

As in TIAs there may be, with the overt evidence of a focal neurological defect, signs which suggest general or local vascular disease. Hypertension and various forms of cardiac abnormality relate to the former, while bruits over the carotid bifurcation, retinal emboli, other evidence of ischemic retinopathy and a unilateral decrease of retinal artery pressure demonstrated by the use of the ophthalmodynamometer exemplify the latter. The differentiation of thrombosis in the vertebrobasilar system from intracerebral hemorrhage is not difficult, but there is a significant problem concerning the differential diagnosis of thrombosis in the carotid system and intracerebral hemorrhage. Table 1 lists the principal differences.

**Intracerebral Hemorrhage**

The absence of a history of TIAs is mentioned first since, if a history of such episodes is obtained, the diagnosis of intracerebral hemorrhage is almost eliminated from differential consideration. Symptoms associated with intracerebral hemorrhage generally come on during activity, and if the patient is sufficiently conscious to report his symptoms, the existence of head discomfort (sometimes described as very severe headache) is commonly related. There is a rapid evolution of focal neurological phenomena over many minutes or a few hours with hemiplegia being the most frequently observed neurological deficit. This rapid evolution, unfortunately, progresses to a state where there is involvement of consciousness going on quickly to coma. It is uncommon for a discrete paralysis of a function with normal mentation to be the physical picture. Usually the blood pressure is markedly elevated; in infrequent instances there may be only moderate hypertension. In 75% to 85% of patients with intracerebral hemorrhage, there will be grossly bloody CSF.

**Cerebral Infarction Secondary to Embolic Arterial Occlusion (Cardiac Source)**

It is now important to make the distinction between an embolus which comes from a relatively distant source, such as the heart or lungs, and the embolus or emboli which originate in an artery to the brain such as the carotid, vertebral or basilar arteries. This discussion has to do with the traditional source of embolus, principally the heart. The most characteristic component of the clinical picture of cerebral infarction secondary to embolic arterial occlusion (cardiac source) is the extraordinarily sudden development of focal neurological symptoms and signs. The patient is often struck down within seconds or at most a few minutes, meaning that there is progression to maximum neurological deficit in this very swift fashion. Frequently there is no complaint of pain along with the relative preservation of normal consciousness. There is an absence of history of antecedent TIAs, ordinarily a clear CSF, and, generally, a discrete and clearly focal neurological
constellation of physical abnormalities. Most important to this diagnosis is the demonstration of a source of emboli, usually in the heart. This “source” commonly consists of either a cardiac arrhythmia, valvular heart disease, or myocardial infarction. In a minority of instances, the basic pathology may be subacute bacterial endocarditis. Concurrent with the search for a source of emboli is the search for evidence of recent embolism in other organs: spleen, kidney, lungs, or extremities. Usually, the CSF is clear. Rapid improvement may take place.

**Cerebral Infarction Secondary to Embolic Arterial Occlusion (Emboli from an Intrac-Arterial Source)**

When emboli consist of thrombus which has formed on an atherosclerotic plaque or of material from an ulcerated atherosclerotic plaque, the source is called “intra-arterial.” The temporal profile of the clinical events may be variable from quite swift (many seconds or a few minutes) to a minute and the headache is immediate or almost immediate. The suddenness of onset and the severity of pain, the suddenness means a second or two of time to a minute and the headache is immediately so intense that it alters the pattern of the patient’s activity. In some instances, there is an immediate or almost immediate disturbance of consciousness (including unconsciousness with recovery of consciousness in a few minutes). Often there is absence or a poverty of definite focal neurological signs. The somewhat frequent exception to this is the appearance of a partial oculomotor nerve palsy.

The characteristic clinical picture of “primary” subarachnoid hemorrhage begins with the extraordinarily sudden onset of severe headache. These two items, the suddenness of onset and the severity of pain, are commonly dramatic. The suddenness means a second or two of time to a minute and the headache is immediately so intense that it alters the pattern of the patient’s activity. In some instances, there is an immediate or almost immediate disturbance of consciousness (including unconsciousness with recovery of consciousness in a few minutes). Often there is absence or a poverty of definite focal neurological signs. The somewhat frequent exception to this is the appearance of a partial oculomotor nerve palsy. In a few minutes there is commonly nausea and may be vomiting. The patient may complain of a stiff neck or extension of the cranial discomfit to the posterior cervical region. On physical examination, particularly if the patient is not in extremis, there is a stiff neck on forward bending and there may be Kernig’s or Brudzinski’s signs as evidence of meningeal irritation. Subhyaloid (pre-retinal) hemorrhages may be detected within a few minutes of onset of the symptoms. Mandatory to the diagnosis is the presence of gross bleeding into the CSF. The clinical diagnosis cannot be substantiated without this finding. The precise source of the bleeding, as well as the genesis of the defect in the arterial wall, often are not predicted accurately on the basis of clinical examination. Subsequent arteriography has demonstrated that the most common defect is the rupture of a saccular aneurysm or in some situations there is no arteriographic evidence of any abnormality. Rarely, a neoplasm may be the source of bleeding but with the whole clinical picture the differential diagnosis can be made with a high degree of accuracy. If there are a history of previous convulsive phenomena, a patient in the second, third or fourth decades of life, a cranial bruit, and particularly a characteristic calcification which appears in x-rays of the head, the existence of an arteriovenous malformation is likely.

**Intracranial Hemorrhage from a Vascular Malformation**

While subarachnoid hemorrhage results from a defect in the wall of an intracranial vascular malformation and the onset of symptoms may be sudden, there is seldom the drama of swiftness and severity commonly associated with subarachnoid hemorrhage from other sources.

As mentioned above, there is generally a history of preceding convulsive phenomena and in some instances there is a story of focal cerebral symptoms. When subarachnoid bleeding with a mild focal neurological deficit and gross blood in the CSF is the sequence of events in an individual in the second, third or fourth decades, the rupture of an intracranial arteriovenous malformation should be suspected. In 20% to 25% of instances, a cranial bruit is present. Some subhyaloid (pre-retinal) hemorrhages may develop, and particular attention is paid to the detection of retinal angiomas since vascular malformations may occur in multiple sites (the retina as well as the brain). X-rays of the head may show calcification, which sometimes is characteristic evidence for the presence of an arteriovenous abnormality. As implied above, the CSF contains gross blood which is not due to a “mechanically bloody cerebrospinal fluid tap” and the observation of blood in the spinal fluid secondary to an intracranial or intraspinal pathology which has produced the bleeding.

**Extradural and Subdural Hemorrhage**

Intracranial bleeding due to head trauma is not ordinarily thought of as a “stroke.” However, it is included here because of the frequency with which the question of diagnosis of traumatic intracerebral hemorrhage arises in instances where the history is inadequate or where the patient has fallen and injured himself at the time of onset of the “stroke.” Acute extradural and subdural hemorrhage must always be considered in the patient who has had any kind of head injury.
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injury or an abrupt ictus under known circumstances, whether the spinal fluid is bloody or clear.

It is particularly important that acute intracerebellar hemorrhage be considered in the differential diagnosis when there is sudden onset of a severe focal cerebellar deficit with progression which often includes nausea and vomiting, cranial nerve palsies, drowsiness and subsequent further change in consciousness. Appropriate neurosurgical treatment may be lifesaving.

Chronic subdural hemorrhage may occur without a history of trauma, and the clinical picture of headache with some drowsiness, mental confusion and occasionally mild focal neurological deficit, especially in the elderly, may be incorrectly diagnosed as cerebral infarction. Acute extradural hemorrhage, acute subdural hemorrhage, and chronic subdural hematoma must be thought of constantly; there should be no hesitation in proceeding to place diagnostic burr holes in instances where subdural bleeding cannot be appropriately excluded.

OTHER CAUSES OF INTRACRANIAL HEMORRHAGE

Hematological disorders which may give rise to intracranial bleeding are leukemia, aplastic anemia and thrombocytopenic purpura. Any portion of the brain may be involved and frequently the lesions are multiple. When cerebral hemorrhage occurs, there is often evidence of previous abnormal bleeding elsewhere in the body (skin, mucous membrane, kidney, or bowel). Rarely, an intracerebral hemorrhage may be associated with chronic liver disease and intracranial bleeding is a rare complication of anticoagulant therapy.

Brain stem hemorrhage secondary to herniation of a portion of the temporal lobe through the tentorial notch is all too frequent a complication of an expanding supratentorial lesion such as neoplasm, abscess, primary intracerebral hemorrhage or massive cerebral infarction. This type of hemorrhage is in the midbrain and pons, and constitutes a serious complication of temporal lobe herniation, almost always resulting in irreversible coma and death. These hemorrhages are given the name of Duret, who was one of the first workers to observe and describe them.

Hemorrhage into primary and secondary brain tumors is an uncommon complication of these lesions. In rare instances, the first clinical phenomenon produced by the basic pathological lesion will be the sudden occurrence of a focal neurological deficit. In these unusual instances, the clinical picture is that of a stroke. In some situations, the clue that the bleeding is secondary and not primary is provided either by the known presence of neoplastic lesions or by the history of the gradual onset of a focal neurological lesion preceding the sudden change occasioned by the hemorrhage.

Septic embolism from the lesions of acute or subacute bacterial endocarditis may produce cerebral infarction with a considerable amount of hemorrhagic reaction in the infarct. A mycotic aneurysm, resulting from local inflammation and destruction of the wall of an artery at the site of arrested septic material, may rupture with severe bleeding resulting. At autopsy, no aneurysmal dilatation may be found, only the edge of the torn vessel. Mycotic aneurysms commonly are at the bifurcation of small vessels, close to or in the subarachnoid space.

HYPERTENSIVE ENCEPHALOPATHY

The use of this term is specifically reserved for a syndrome in which there is a stereotyped sequence of events of serious import and dramatic development. This syndrome occurs in persons with moderate or severe hypertension and is characterized by the increase in severity of the hypertension over a few hours' time, severe progressive headache often associated with nausea and/or vomiting proceeding to alterations of consciousness (apathy progressing to coma), and convulsions. There is generally severe hypertensive retinopathy and there may or may not be evidence of various degrees of the renal involvement so frequently associated with hypertension. The CSF pressure is increased but the fluid is commonly otherwise normal. This is the classical basic syndrome of hypertensive encephalopathy.

To these events and physical signs occasionally may be added the history of the development of focal neurological signs and these signs are present on examination. It is emphasized that the addition of these focal neurological signs (in order for the primary diagnosis of hypertensive encephalopathy to be seriously considered) must be in the context of being added onto the syndrome described above. Otherwise, the clinician will be repeatedly making a diagnosis (in the absence of the basic syndrome) of hypertensive encephalopathy when the disorder is simply some other type of "stroke."

VASCULAR MALFORMATIONS AND DEVELOPMENTAL ABNORMALITIES: ANEURYSM

The principal effect of saccular or berry aneurysms results from rupture of the lesion with the production of subarachnoid hemorrhage and brain damage. Fusiform, diffuse and globular aneurysms consist of varieties of enlargement of the entire circumference of the artery. Fusiform aneurysms are tortuous, relatively circumscribed dilatations most commonly involving the basilar artery or the internal carotid arteries within or near the cavernous sinus. The distinction between fusiform aneurysms and diffuse aneurysms is probably of little importance. Globular aneurysms are a group in which there is marked spherical dilatation with the parent vessel coming in one side and leaving from the other side of the lesion. Any of these aneurysms may produce symptoms by exerting pressure on neighborhood structures or by being the site of thrombosis.
TRIGEMINAL ENCEPHALOANGIOMATOSIS (STURGE-WEBER-DIMITRI DISEASE)

A localized degeneration of the cerebral cortex is found in association with a port-wine stain (capillary angioma) in the distribution of the first division of the trigeminal nerve in the face. The cerebral cortex adjacent to the angioma often contains calcium and produces a double contour opacity on x-ray of the head. These cerebral lesions may be associated with mental retardation, convulsive disorders and focal neurological deficits, although the lesions seldom bleed.

CONGENITAL ABNORMALITIES IN THE ANATOMICAL PATTERN OF CEREBRAL ARTERIES

Many variations in the relative size of the vessels making up the circle of Willis have been described. Deviations from the "normal pattern" are present in almost half of the specimens which have been examined. These deviations from "normal" are probably only of clinical importance when vessels are either absent or of threadlike size. Rarely, one or both internal carotid arteries are missing — occasionally one is only rudimentary in size. Carotid-basilar anastomosis via the cavernous sinus rarely occurs.

INFLAMMATORY DISEASE OF ARTERIES

The arteritides form clinical manifestations by producing varieties of metabolic encephalopathy or cerebral infarction.

INFECTIONS AND INFESTATIONS

Meningovascular syphilis is now a relatively uncommon cause of stroke. Neurosyphilis produces a chronic meningitis and the blood vessels of the brain and spinal cord, lying within the meninges, become involved. If the arteritic change involves the intima, thrombosis may result, producing cerebral infarction. These infarcts are characteristically small in size. If there has not been previous antiluetic therapy, the CSF will invariably show an increased cell count and an elevation of the protein level. False-positive tests for syphilis in the CSF are essentially nonexistent.

Pyogenic meningitis (influenza, staphylococcus, pneumococcus) and tuberculous meningitis occasionally cause cerebral arteritis and thrombosis. Generally, the primary diagnosis will already have been made; the initial clinical abnormality is rarely the sudden onset of a focal brain lesion.

Rare instances of arteritis may occur with typhus, schistosomiasis mansoni, mucormycosis, malaria and trichinosis. Papillary and arterial changes and perivascular inflammatory cells may be present in the nervous system in typhus and other rickettsial diseases. The abnormal chemistry underlying the confusional psychoses, convulsions and coma which may occur with these lesions is not understood.

Schistosomiasis mansoni infection may be associated with occlusion of small arteries and multiple cerebral infarcts. In rare instances, diabetes mellitus may be complicated by mucormycosis and occlusion of the internal carotid artery.

A clinical state called "cerebral malaria" may occur with malaria of the malignant or falciparum variety. There is acute onset of hyperpyrexia, convulsions, somnolence deepening to coma, and often death. If the patient lives, there may be focal brain involvement with hemiparesis, aphasia, etc. The cerebral infarction is caused by the occlusion of cerebral capillaries and arterioles by masses of red blood cells.

ARTERITIDES OF UNDETERMINED CAUSE

Cranial arteritis (temporal arteritis) uncommonly causes the sudden onset of a focal brain lesion. However, the differential diagnosis of this disorder is extremely important since there is effective treatment (steroids) which will prevent the unilateral or bilateral blindness which occurs in more than one-third of the cases. In this disorder the branches, mainly of the external carotid artery (especially the temporal arteries), are involved by subacute inflammation which may lead to thrombosis. There are generally some kind of cranial or scalp discomfort, lassitude and malaise, and without treatment almost always marked elevation of the erythrocyte sedimentation rate. In some instances, the syndrome of amaurosis fugax has been caused by cranial arteritis.

Systemic lupus erythematosus produces arteritic changes in the central nervous system (also at times the peripheral nervous system) which may cause a form of metabolic encephalopathy characterized by delirium, confusional states and convulsions with drowsiness, sometimes progressing to coma. In rare instances, the effective inflammatory pathology appears to be limited to one area so that a cerebral infarct occurs. More commonly, micro infarcts and sometimes petechial hemorrhages are multiple and widely scattered. In other instances where there is severe kidney involvement, intracerebral hemorrhage or hypertensive encephalopathy may occur. Very rarely embolic occlusion of large and small brain arteries secondary to verrucous endocarditis (Libman-Sacks) happens.

Rheumatic arteritis is an uncertain specific entity. In acute rheumatic fever or in chronic endocarditis of rheumatic origin, brain embolism may take place. Lupus erythematosus and rheumatic fever are closely interrelated, and it may be that lesions of the latter are actually those of the former.

Polyarteritis nodosa (panarteritis) directly involves the cerebral arteries in less than 10% of cases. The neurological picture produced is not commonly that of the acute onset of cerebral infarction, although such may happen. There is more likely to be the occurrence of a more general cerebral syndrome in which headache, confusional reactions and convulsions are present. The coexistence of mononeuritis...
CLASSIFICATION AND OUTLINE OF CEREBROVASCULAR DISEASES II

Multiplex may assist greatly in establishing a clinical diagnosis. Hypertension is commonly present and if there is renal involvement it may become so severe as to lead to hypertensive intracerebral hemorrhage or hypertensive encephalopathy.

Idiopathic granulomatous arteritis (Takayasu’s disease) of the aorta and its major branches, including the common and internal carotid arteries, occurs from time to time in all countries, although it was originally described in Japan. It is frequently referred to as the “pulseless disease” due to the occlusion of carotid and limb arteries produced. Along with various types of emboli and angioma, this disorder should be considered in the differential diagnosis of unexplained stroke in young adults.

Moyamoya (a Japanese expression for something hazy like a wisp of fog drifting in the air) disease is a rare one, which in its fully developed form is characterized by an angiographical picture showing occlusion or stenosis at the carotid bifurcation with a netlike cluster of vessels intracranially on the same side. In children, the disorder is commonly characterized by attacks of paroxysmal hemiplegia, while in the adult the onset may be sudden with seizures and subarachnoid hemorrhage.

Thromboangiitis obliterans (Winiwarter and Buerger) is not included; its existence as an entity is uncertain.

DURAL SINUS AND CEREBRAL VENOUS THROMBoses

In everyday practice, intracranial phlebothrombosis and thrombophlebitis are uncommon and do not often cause confusion in the differential diagnosis of stroke. With inflammation in these structures, blood may be dammed back into small venules and capillaries, obstructing venous drainage so that local brain ischemia occurs sometimes followed by cerebral edema and hemorrhagic infarction. Focal convulsive events may occur.

Vein and sinus thromboses were much more common in the pre-antibiotic era and were secondary to pyogenic infections of the mastoid, paranasal sinuses or face. The inflammatory process sometimes travels to the larger veins directly or may come about from the production of a local osteomyelitis, or by producing thrombophlebitis of small diploic vessels which carry the infection intracranially.

Thrombosis of cerebral veins may be seen shortly following childbirth or a surgical operation. The former may be due to hypercoagulability of the blood (hyperfibrinogenemia or increased blood platelet count).

The manifestations greatly depend upon the site and severity of the cerebral pathological process. Convolusions and/or hemiparesis may occur; the CSF may be bloody. Weakness involving only a leg or an arm (sparing the face) is likely due to a lesion adjacent to the sagittal sinus, the common site of infarction, if occlusion of the sagittal sinus has taken place. If the sagittal sinus is occluded, another clinical picture is that of bilateral neurological signs. With this and/or occlusion of the transverse sinuses, there may be an increase in intracranial pressure which can be associated with headache, choked disks and visual obscurations. The changes in the CSF are variable, from a mild increase in the white blood cell count and rise in the pressure of modest degree to bloody CSF under high pressure if extensive hemorrhagic infarction has taken place. In those instances where the venous thrombosis is secondary to a purulent meningitis, the characteristic CSF findings of the latter will be present.

THE CLINICAL NEUROLOGICAL PICTURE

The clinical neurological picture is determined by the site of the brain damage (infarction or hemorrhage). It is now common practice to divide the brain blood supply into two major categories: (1) the carotid system, and (2) the vertebrobasilar system.

Internal Carotid Artery

Occlusion of the internal carotid artery in the neck does not produce any characteristic clinical picture. In the presence of adequate intracranial collateral circulation, internal carotid artery occlusion may produce no symptoms or signs. At the other end of the spectrum, in instances where the collateral circulation is faulty, there may be infarction of a major portion of the ipsilateral hemisphere with contralateral hemiplegia, hemianesthesia, homonymous hemianopia and aphasia (if the dominant hemisphere is involved). Stupor may come quickly, particularly if there is brain swelling with compression of the brain stem. With occlusion of the internal carotid artery in the neck, there is very seldom a simultaneous onset of permanent blindness in the ipsilateral eye together with the contralateral hemiplegia, hemianesthesia, and aphasia. Likewise, occlusion of the internal carotid artery in the neck is seldom a direct cause of permanent ipsilateral blindness (without hemispheric signs). Much more common than the very severe syndrome outlined above is the circumstance where there is atherosclerotic occlusive disease in the internal carotid artery in the neck and an associated distal arterial occlusive event which produces only a fragment of the neurological defect which would occur if all the territory supplied by the carotid artery were infarcted. Thus, the neurological picture may range from a monoparesis to hemiparesis with or without a homonymous defect in vision, a variety of impairments of speech and language, different types of anosmia and a complete range of partial to full sensory abnormalities. A cervical internal carotid lesion is particularly to be suspected as a pathogenetic source of a sudden-onset hemispheric neurological defect when there have been antecedent characteristic TIAs (particularly amaurosis fugax) or a long systolic or systolic-diastolic bruit at the take-off of the ipsilateral internal carotid artery, ipsilateral decrease in retinal...
artery pressure or cholesterol or fibrin-platelet emboli in the appropriate eye.

**Middle Cerebral Artery**

The most significant clinical subdivision of the internal carotid artery system is the middle cerebral artery (MCA) or what might be more appropriately called the middle cerebral arterial system. Occlusion of the first portion of the MCA is almost always associated with the production of a neurological defect. Such a lesion is more distal in the total carotid arterial system and occurs at a site where a chance for collateral supply via the circle of Willis is no longer present. Occlusion of the artery is said to characteristically produce a contralateral hemiplegia, hemihypesthesia or hemianesthesia, homonymous hemianopia and aphasia if the defect is in the dominant hemisphere. However, occlusive disease in the middle cerebral arterial system more commonly produces a portion or fragment of this total picture, the variations in the neurological findings providing a broad spectrum of abnormalities. If the infarct is very large, there is more likely to be brain edema with brain stem compression and stupor a few hours after the onset. Depending on the pattern of collateral blood supply and the actual artery or arteries occluded in the middle cerebral system, there will be such variations as: from hemiplegia to a mild paresis of one side of the face alone, mild weakness of one upper extremity alone, etc. If the focal impairment of blood supply is in the posterior portion of the middle cerebral system, there is more likely to be homonymous hemianopia or a partial homonymous visual field defect, aphasia or dysphasia. If penetrating branches of the MCA are the only vessels occluded, the defect may be only motor, or if arteries to sensory cortex are the only ones involved a cortical-type hypesthesia will result. It is emphasized that there is a wide spectrum of neurological abnormalities (including many variations on the parietal lobe syndrome) which may occur with occlusive disease in the middle cerebral arterial system.

**Anterior Cerebral Artery**

Occlusive events in the main stem of the anterior cerebral artery or its various branches also are associated with the production of a variety of clinical events. No solitary syndrome has been delineated as characteristic of occlusion; this is because of the variety of patterns of branches of the vessels and particularly the variety of patterns of collateral supply available. When there is paralysis or severe weakness of the opposite lower extremity with mild or no involvement of the opposite arm, a lesion in the distribution of the anterior cerebral artery is likely. Mental change, often subtle and mild but sometimes severe enough to be called dementia, dyspraxia or apraxia of the use of an extremity or in walking, grasping and sucking reflexes, and problems with maintenance of continence of bowel and bladder may be associated with infarction of the brain in this distribution.

**Verteobasilar Arterial System**

The vertebobasilar system supplies blood to the medulla,pons, cerebellum, mesencephalon, thalamus, occipital lobes, and even portions of the temporal-occipital and parieto-occipital junctions. The most common defects include abnormalities of motor function: weakness, clumsiness, or paralysis of any combination of extremities up to quadriplegia with appropriate pyramidal tract signs combined with unilateral or sometimes bilateral cranial nerve palsies, particularly oculomotor defects or signs of trigeminal nerve or facial nerve involvement. A so-called “crossed” defect (motor or sensory on one side of the face and the opposite side of the body) is evidence of a brain stem lesion until proved otherwise. Involvement of sensory function is common and this may be in any combination of extremities including all four or may be in both sides of the face or mouth. If the occipital lobes are the site of ischemia, there will be loss of vision, complete or partial in both homonymous fields (bilateral homonymous hemianopia). Ataxia, imbalance, unsteadiness or dysequilibrium not necessarily associated with vertigo may occur because of labyrinthine system or cerebellar system defects. Vertigo, particularly in such instances as the lateral medullary syndrome, is a very common complaint and when produced by brain stem infarction is usually associated with one of several types of nystagmus. Dysphagia and/or dysarthria may occur in combination with any of the abnormal neurological signs already mentioned. The most important evidence for a brain stem locus of the ischemia is the bilaterality of sensory or motor abnormalities coupled with definite evidence of cranial nerve (III to IX) involvement. Impairment of consciousness early in the course of events is unusual. If nystagmus alone is noted, it may be impossible to decide whether the lesion is in the vestibular-cerebellar system in the brain stem or in a peripheral portion of the vestibular system. However, if the nystagmus has added to it impaired ocular rotation or some evidence of an upper motor neuron defect in one or more extremities or some abnormality such as dysarthria or dysphagia, the anatomical site of the ischemia is identified as brain stem. The neurological syndrome in vertebobasilar system acute occlusive disease develops during hours or a few days with progression frequently in stuttering or stepwise fashion. These repeated steps of progression may occur over a period of 72 to 96 hours and there is, therefore, the possibility of presuming that the progression has been “completed” only to find that this is not true.

**Posterior Cerebral Artery**

As has already been mentioned for other main cerebral arteries, the posterior cerebral artery supply...
CLASSIFICATION AND OUTLINE OF CEREBROVASCULAR DISEASES II

actually constitutes a subdivision of the vertebrobasilar system. The neurological abnormality produced by occlusive disease in this system depends on the site of the occlusion or stenosis and on the effectiveness of the available collateral flow. If the occlusion is distal, a homonymous hemianopia or even quadrantanopia may result. There may be variations in the position of the “watershed” between the posterior portion of the middle cerebral system supply and the anterior portion of the posterior cerebral supply so that a lesion in the posterior cerebral artery on the dominant side may produce dyslexia, dyscalculia, and a variety of speech and language abnormalities, while on the nondominant side portions of a parietal lobe syndrome defect may occur. If the occlusion is more proximal in the posterior cerebral arterial system, there may be a contralateral hemiparesis (cerebral peduncle), third nerve palsy (oculomotor), or a contralateral thalamic syndrome (posterior thalamus). Cortical blindness and varieties of visual-verbal agnosia may be produced if both occipital lobes are affected because of impaired blood flow to the territories of both posterior cerebral arteries. The patient may be unaware that vision is affected or may have strange variations in behavior.

Vertebral Artery

Although it is now common practice to use the phrase vertebrobasilar system, it has been demonstrated that occlusion of a vertebral artery at or near the origin of the posterior inferior cerebellar artery will produce a lateral medullary syndrome (Wallenberg’s syndrome) which is characterized by the sudden onset of severe rotational vertigo, nausea and vomiting, dysphagia, ipsilateral cerebellar ataxia, ipsilateral Horner’s syndrome and involvement of pain and temperature sense on the ipsilateral face with contralateral loss or impairment of sensation for pain and temperature on the extremities and trunk. When there is severe stenosis or occlusion of a subclavian artery, there may be reversal of flow in the ipsilateral vertebral artery. This has not been consistently associated with any neurological symptoms or signs. When one vertebral artery is very small and the other unusually large, occlusion of the principal vertebral artery may be associated with symptoms and signs usually occurring with main stem basilar artery occlusive events.

C. General cerebral dysfunction

This category refers to general cerebral ischemia, which results from reduction in blood supply to the brain, yet is applicable also to lesions of other causes. This category does not imply the presence or absence of disease of the cerebral arteries.*

*In the present state of our knowledge, chronic dementia of the elderly without evidence (clinical or pathological) of neurological deficits of vascular origin is rarely caused by cerebral atherosclerosis alone. Although these patients are frequently diagnosed as having “arteriosclerotic dementia,” “cerebral atherosclerosis with dementia,” or “chronic brain syndrome with cerebral atherosclerosis,” their conditions are often confused with other toxic metabolic or degenerative diseases (e.g., Alzheimer’s disease or senile dementia).

A patient with focal neurological deficit of vascular origin, e.g., hemiparesis plus a defect in mentation, should be classified under I.B.

Part V. Clinical Phenomena (History, Physical Examination, Laboratory Examination, Roentgen Examination, Other)

A. History

The family history may contain significant information about hypertension, diabetes and cardiac disease. The past history of the patient may contain relevant material about these same items as well as other items, including previous stroke.

In reviewing the history of the present illness, the physician must probe for answers (direct or by inference) relevant to different sections of this Classification of Cerebrovascular Diseases. If the complaints have been transient, the circumstances of onset, including activity being performed, physical position of the patient, quickness of development of the symptoms, and duration as well as the listing of the complaints must be described. In the history there may be important information concerning the pathophysiological mechanism, i.e., cardiac source for emboli or intracarotid source for emboli if there has been amaurosis fugax. The story of rapid onset and progression of a focal neurological deficit with headache against a background of untreated hypertension may point the way to a diagnosis of intracerebral hemorrhage. Antecedent TIAs statistically strongly favor a diagnosis of cerebral infarction rather than cerebral hemorrhage. These are but a few examples of the fashion in which history-taking should be designed.
to bring out information concerning "Clinical Stage, Pathophysiological Mechanisms, Anatomy, and Pathology."

B. Physical examination
   1. General
   In the general physical examination the emphasis is directed toward detecting evidence of any pathology in the cardiovascular system — evidence which may be of great importance, such as severe arterial hypotension with a cardiac arrhythmia, or evidence which may be of minimal importance, such as a blood pressure level of 150/90 mm Hg. Attention should be given to heart rate, rhythm and size, blood pressure in both upper extremities, heart sounds, peripheral pulses and detection of congestive failure including dyspnea, venous engorgement, hepatomegaly, ascites, pedal edema and pulmonary congestion or edema.

   Skin lesions such as ecchymosis and petechia may suggest processes which produce similar lesions in the brain. Skin tumors as well as masses in other sites may be the origin of intracranial metastases.

   2. Neurological
   The neurological examination and the interpretation of the neurological findings often make feasible the diagnosis of the site of the nervous system lesion or lesions. Certain combinations of neurological signs almost establish a diagnosis, i.e., lateral medullary syndrome, bilateral homonymous hemianopia, etc. Often the accurate inspection of a patient with "confusion" will detect aphasia and the physician then knows that the neuropathology is focal rather than diffuse. The reader is referred to one or several of the many textbooks which describe the neurological examination.

   3. Vascular (largely neurovascular) examination
   In order to place special emphasis on the importance of certain abnormal physical signs, certain portions of the examination have been grouped together under this term. These include: (a) inspection of vessels, (b) palpation (including carotid sinus massage and carotid compression), (c) auscultation at cervical and cranial sites, (d) ophthalmoscopy (including inspection of the retina for emboli, cotton-wool patches, vascular occlusions, hemorrhage and ischemic retinopathy), and (e) ophthalmodynamometry.

      a. Inspection of vessels
      Cranial arteritis is an unusual cause of stroke. However, accurate diagnosis is vital to correct treatment and significant arterial change may be detected by thoughtful viewing of superficial temporal arteries, coupled with palpation.

      b. Palpation
      Palpation of cervical-cerebral vessels should be done gently. Minor differences in pulse between sides are difficult to interpret and it may be impossible to distinguish a pulse coming from the first portion of the internal carotid artery or from the external carotid artery. Patients suspected of having atherosclerosis of cervical vessels may have ulcerated plaques or early thrombus forming; both are situations where manipulation of the arteries could dislodge emboli. Similarly carotid compression tests and carotid massage have inherent danger in patients with cervical-cerebrovascular disease. These important dangers include dislodging emboli, temporarily decreasing carotid flow and production of a significant change in cardiac rhythm. If the "sick sinus" syndrome is suspected, massage should be performed only with continuous ECG, EEG and blood pressure monitoring as well as with personnel and equipment available for the care of a cardiac emergency.

      c. Auscultation
      Auscultation of the cervical vessels often provides important evidence concerning the pattern of blood flow. A bell-type stethoscope is most easily applied in the supraclavicular fossa and over the eyes without using physical pressure which may produce artifactual noise. The bell of the stethoscope is first placed over the aortic valve and then moved (1 cm or less at a time) superiorly. This progressive movement of the stethoscope is necessary to distinguish transmitted carotid sounds from sounds arising in the innominate, subclavian, common carotid or internal carotid arteries. A neutral position (patient sitting or lying with face straightforward) is less likely to create sounds difficult to interpret than a variety of twisted neck positions. If respiratory (tracheal) sounds obscure auscultation, the patient is requested to "stop" breathing for a few seconds; then "start" breathing is the instruction. Bruits should be graded for loudness, the scale being 1 (least) to 6 (loudest); i.e., 1/6 is barely audible, 6/6 is the loudest. The timing (systolic, diastolic, systolic-diastolic), duration (short, medium, long) and quality (rough, soft, smooth, etc.) should be described. A bruit of 1/6 loudness is of little significance, while one of 2/6 to 3/6 loudness, long systolic-diastolic duration, and timing of fairly high pitch over the origin of the internal carotid artery means high-grade carotid stenosis until proved otherwise. A soft (1/6 to 2/6) diastolic sound, varying with slight change in neck position, is commonly an unimportant venous hum. Soft, sometimes almost continuous cervical bruits are fairly common in children and ordinarily do not indicate the presence of significant pathology. By carefully recording the description of bruits and correlating this with arteriographical and other findings, the examiner will quickly learn to correctly interpret such sounds.

      If the patient's history suggests the presence of a neoplasm or arteriovenous malformation, auscultation over the cranial vault and orbits should be done. When there is a complaint of a rhythmic head noise, particular attention is directed to auscultation of the locus of the sound. It may be necessary to wet the hair of the patient to eliminate artifactual noise. Auscultation of the orbit is performed by instructing the patient to close the eyes, placing the bell of the stethoscope over the eye and having the patient open the eyes to...
eliminate artifactual muscle sounds. Soft bruits over the cranial vault of children are of little importance. Loud bruits may be caused by angiomas, arteriovenous shunt and, rarely, brain neoplasms. A continuous, almost machinery-like murmur or bruit over the orbit is most commonly caused by a carotid cavernous arteriovenous shunt. Noises heard over the orbit have been of little help in establishing the site and severity of lesions of the internal carotid artery.

d. Ophthalmoscopy

Ophthalmoscopy provides an opportunity to directly inspect small blood vessels, blood vessels which are a direct continuation of the internal carotid arterial system. In office and hospital practice relatively little use is made of this simple, safe method of acquiring important data concerning the cerebral-cerebral portion of the circulation. The retina should be inspected for arterial or venous occlusion, emboli (cholesterol, platelet-fibrin, calcific, mixed, foreign body), hemorrhages, cotton-wool patches, venous stasis, microaneurysms, changes associated with arterial hypertension, papilledema and ischemic retinopathy.

1) Retinal emboli

In the last two decades the importance of detecting (with the ophthalmoscope) a retinal embolus or emboli has been demonstrated. The most common emboli are made up of cholesterol crystals. These appear as shiny orange-yellow plaques often situated at the bifurcation of retinal arterioles. The plaque may appear to be wider than the arteriole; one sees the outer dimension of the column of red blood cells rather than the wall of the arteriole. Pressure on the eye often changes the position of the embolus slightly — the material may appear to glint or change shade, a characteristic sometimes referred to as a heliographic reflection. The blood flow in the arteriole is often seemingly unimpeded by these bright orange-yellow plaques. These emboli may move distally and often disappear in a few days. The presence of one or more cholesterol retinal emboli indicates that there is or has been an ulcerated atheromatous carotid (internal) lesion until proved otherwise.

Another important type of embolus in retinal vessels consists of gray-white material, thought to consist of blood platelets and fibrin. These emboli may be long and seen to move through an arteriole but are commonly stationary; pressing on the eye does not move the embolus, and there is no heliographic reflection. Blood does not appear to flow past these emboli; there may be infarction of the retina. Special studies show that some of these emboli have a high lipid content. In many instances the source of these emboli is an atheromatous lesion at the origin of the internal carotid artery. Particles of calcium are another type of retinal embolus. These are white, generally short, and stationary. Calcium emboli commonly come from heart valve lesions.

Septic emboli, talc and cornstarch emboli as well as others may be seen in the retina but are less common than those already described.

e. Ophthalmodynamometry

Ophthalmodynamometry is a procedure for measuring the arterial systolic and diastolic pressures in the main retinal branch or branches of the ophthalmic artery. The convex foot-plate of the instrument is applied to the conjunctiva over the insertion of the lateral rectus muscles in a horizontal manner so that the instrument points directly toward the opposite eye. When measurements are being made in the patient’s right eye, the instrument is held in the observer’s left hand and the ophthalmoscope is held in the right hand. To measure pressure in the left retinal artery, the observer holds the ophthalmodynamometer in the right hand and the ophthalmoscope in the left. When the instrument is in position, the observer must bring the central artery on the disk into focus through the ophthalmoscope. The instrument then is pressed gradually against the eye to raise the intraocular pressure sufficiently to exceed the diastolic level of the blood pressure in the retinal artery. The diastolic pressure is that level which produces the first collapsing pulsation of the artery. At this point a finger is applied to the brake on the instrument and the reading is taken from the scale. The ophthalmodynamometer is reapplied and several more readings are taken to insure accuracy. The systolic pressure is obtained by increasing the force of application of the instrument still further. The visible arterial pulsation gradually diminishes as the pressure increases, and when pulsation ceases, the reading on the instrument is the systolic blood pressure.

Ophthalmodynamometry is ordinarily useless unless the arterial pressures are measured in both eyes. It cannot be performed unless the patient is cooperative. It is helpful to instill a mydriatic; this should not be done if there is glaucoma. The test should not be done soon after cataract extraction, recent retinal detachment, etc.

The clinical significance of the retinal arterial pressures is dependent on comparing the values in the two eyes. A difference of 15% to 20% almost always is a sign of stenosis or occlusion of the internal carotid artery ipsilateral to the lower pressure. The arterial pressure may be equal and/or normal in the presence of unilateral carotid stenosis or occlusion because of the development of collateral blood supply. Immediately following acute occlusion of an internal carotid artery the ipsilateral retinal arterial pressure drops. Return of the pressure to that of the contralateral eye depends on the speed with which collateral circulation develops. A marked decrease in the retinal arterial pressure (brachial arterial pressure remaining normal) when the patient moves from the supine position to the upright position (ocular orthostatism) is important evidence of carotid occlusive disease.

A suction ophthalmodynamometer requiring a
source of electric power is available. It is safe and highly accurate, but unhandy.

f. Neurological signs produced by position

If the patient describes phenomena which are precipitated by certain positions of the head and/or torso, it is wise to carefully try to reproduce the symptoms and simultaneously search for physical signs of dysfunction. Thus, extension of the neck may be associated, in the patient’s history, with the onset of “dizziness;” have the patient reproduce the motion and observe for nystagmus. The history provides a clue to the existence of orthostatic hypotension. These are examples of a variety of such phenomena.

C. Laboratory examination

There are a number of laboratory tests which are of aid in the diagnosis and management of cerebrovascular disease, but none of them can approach in general usefulness a careful history and physical examination.

1. Urinalysis
   a. Sugar
      Under usual circumstances, there is no glucose in the urine. This is a crude but very useful screening test for abnormal glucose metabolism.
   b. Protein
      Normally, on qualitative evaluation, there is no protein in the urine. This is a crude but valuable screen for either abnormal substances coming through the kidney or for abnormal kidney function.
   c. Sediment microscopy
      This examination may give valuable information about such things as the presence of vasculitis and inflammatory disease of the kidney as well as being a crude screen of renal function in patients with hypertensive vascular disease.
   d. Other
      A wide variety of tests for substances including those for vanilmandelic acid, porphobilinogen, cyanide-nitroprusside test for homocystinuria, aldosterone, lead, metanephrines, etc., may be done in selected instances but are only rarely of help in the management of a stroke patient.

2. Blood
   a. Test for syphilis, such as VDRL, etc.
      In the 1970s, meningovascular lues, or luetic vasculitis, is not a common cause of stroke; however, a blood screening test for syphilis should still be included in the laboratory profile.
   b. Red blood count, white blood count, blood hemoglobin, hematocrit, and differential blood count
      These tests frequently give valuable information concerning the presence of hemocoagulation secondary to dehydration, the presence of polycythemia vera which in turn may be associated with thrombosis or with hemorrhage, and other forms of polycythemia. The differential blood count may suggest the existence of or the beginning of an inflammatory process and the peripheral blood film provides the opportunity to estimate the number and types of platelets.

   c. Erythrocyte sedimentation rate (ESR)
      While it is relatively uncommon for a form of vasculitis (temporal arteritis, giant cell arteritis, lupus erythematosus, polyarteritis nodosa, etc.) to be directly a cause of stroke, such an etiology may be of tremendous importance and the diagnosis may be immediately suggested by the marked elevation of the ESR which is so commonly present in acute vascular (collagen) disorders such as those referred to.

   d. Fasting blood sugar or casual blood sugar
      This is an excellent screening test for the detection of diabetes mellitus and, in certain patients who are acutely ill, hypoglycemia of a variety of origins may be significant.

   e. Creatinine or urea
      Either is a reasonably good screening test for renal disease, especially when coupled with urinalysis. If either creatinine or urea is normal in the blood and urinalysis is normal, significant renal disease generally is not present.

   f. Cholesterol — triglycerides
      Slight or moderate elevations of cholesterol and/or triglycerides are probably of little significance at the time a stroke actually occurs. However, elevated blood cholesterol has been identified as a likely risk factor for occlusive cerebrovascular disease in persons under 50 years of age. Elevation of triglycerides has been indicated as a risk factor in the development of coronary heart disease. Thus, one or both substances may be important to the prevention of further focal cerebral ischemia in a person with TIAs or in a patient who has had a completed cerebral infarct. When there is marked elevation of the values, as in familial hyperlipidemia, the risk of cerebral infarction is definitely increased.

   g. Prothrombin time
      In certain selected instances anticoagulant therapy may be initiated early in the course of occlusive cerebrovascular disease; in such instances it is important to have the results of this test for “baseline” purposes.

   h. Uric acid
      There is little direct importance of mild to moderate hyperuricemia with the practical management of an acute progressing stroke or even a completed stroke. However, hyperuricemia has been associated with atherosclerosis of the coronary, peripheral and cerebral arteries and, therefore, may be somewhat distantly related to the profile of “risk factors for stroke” and may be obtained in the work-up for this reason.

   i. Other
      Here included are numerous tests which may be important in the care of selected patients but are not considered in the usual list for essentially all patients thought to have some form of cerebrovascular disease. These include: acid-base balance, bilirubin, bromsulphalein dye retention, calcium, catecholamine, chloride, clot retraction, whole blood coagulation
time, plasma coagulation time, partial thromboplastin time, plasma fibrinogen, euglobulin lysis time, serum osmolality, arterial oxygen saturation, oxygen tension (air), potassium, protein electrophoresis, sodium, free and/or total thyroxine, enzymes, partial pressure of carbon dioxide and pH. It may be that tests of platelet adhesiveness and ability to aggregate may become clinically important.

3. Cerebrospinal fluid

This examination should be performed when the clinician has a serious problem in establishing the differential diagnosis of the intracranial pathology — bleeding, focal ischemia or inflammatory disease. The fluid is ordinarily obtained by lumbar puncture and the examination should not be considered "routine" in the work-up of the patient. The amount of fluid withdrawn depends upon the question to be asked; a cubic milliliter will be adequate to demonstrate gross bleeding, while the amount needed will be much greater if the search is for the etiology of a meningitis which is increasing the number of lymphocytes in the fluid. In some instances the clinician may be reluctant to do a lumbar puncture even though there is no papilledema because of clinical evidence of increased intracranial pressure with the apparent brain pathology limited to one hemisphere. If there is a macroscopic amount of blood in the CSF, it is not possible (without analysis of the history and a neurological examination) to know whether the bleeding comes from a primary subarachnoid site or from an intracerebral source. The observations made depend on the clinical situation.

a. Pressure

Information about the CSF pressure is of only relative value in the differential diagnosis. The Queckenstedt maneuver (compression of the jugular veins) should not be performed for the differential diagnosis of intracranial lesions. In certain instances the examiner will not wish to withdraw enough fluid to even measure the pressure in the usual manometer.

b. Color

Normally the CSF is crystal clear and colorless. If there is gross blood and the intensity of color decreases after a few drops of fluid appear, the bleeding is due to trauma to a blood vessel. If the fluid is xanthochromic immediately after it is withdrawn or within a very few minutes, the xanthochromia has been produced by something other than a traumatic lumbar puncture. A trace of xanthochromia may be quickly detected by putting a cubic centimeter or less of CSF in a Wassermann tube and comparing the appearance with a tube containing water. The line of vision should be directed down the length of the tube and the background should be white. The color may be produced by high CSF protein content or icterus but is commonly due to intracranial bleeding (rarely intraspinal bleeding). If the CSF has a "ground-glass" appearance, there are generally more than 400 white blood cells present per cubic milliliter of fluid.

c. Cells

The CSF should be examined for red blood cells and white blood cells. In certain instances yeast, neoplastic cells and bacteria may be detected. It should be noted that in the usual setting in which CSF examination is performed in a patient strongly suspected of having cerebrovascular disease the appearance of the fluid and the microscopic examination of the fluid give the answer to the question most commonly asked: "Has there been bleeding into the subarachnoid space?"

d. Protein

Modest elevation of the CSF protein is common in patients with various categories of cerebrovascular disease and is not of particular assistance in establishing an accurate differential diagnosis. In this general diagnostic situation the electrophoretic fractionation of the proteins is only rarely of assistance in differential diagnosis.

e. Glucose

Unless there is massive subarachnoid hemorrhage, CSF glucose level is not commonly altered by various categories of cerebrovascular disease. When attention is to be paid to the glucose determination, the blood and CSF glucose levels should be drawn simultaneously to determine whether the normal ratio (CSF glucose approximately two-thirds the blood glucose) is present.

f. Test for syphilis

A positive test for syphilis in the CSF is highly significant; either such a result is a laboratory error or the chances are very great that the patient has luetic involvement of the nervous system, active or inactive. Although meningovascular syphilis is less common now as a cause of stroke than a few decades ago, this etiology continues to occur.

g. Enzymes

Glutamic oxalacetic transaminase (GOT), lactic dehydrogenase (LDH), and creatine phosphokinase (CPK) in the CSF have been studied but are not significantly helpful in the diagnosis of various categories of cerebrovascular disease or in the distinction between cerebrovascular disease and brain neoplasms. Therefore, these tests ordinarily are not performed.

h. Gases and pH

CSF pH and P02 are not ordinarily done as a portion of the differential diagnostic studies or for the clinical management of the patient. The tests have been done for investigative purposes.

4. Electrocardiogram

Because of the full documentation of the active interrelationship between various forms of cardiac pathology, including disturbances of rhythm, and cerebral ischemia (both diffuse and focal), an electrocardiogram should be obtained in essentially all patients suspected of any category of cerebrovascular disease. If there is no history of any cardiovascular symptoms or pathology, if heart size, sounds and
CLASSIFICATION AND OUTLINE OF CEREBROVASCULAR DISEASES II

5. Electroencephalogram

In the usual stroke patient (typical TIAs, most instances of progressing stroke and almost all cases of completed stroke) the electroencephalogram adds little significant information and is not necessary as a portion of the work-up of the patient. Commonly, in vertebrobasilar disease the electroencephalogram shows no focal abnormality. It has been said that serial electroencephalograms may very well portray accurately the favorable or unfavorable progression of the brain lesion in stroke. However, the clinician can almost always get this same information by spending three or four minutes with the patient one or more times a day.

In selected instances an electroencephalogram may reveal multiple focal abnormalities, thus giving potential evidence concerning the presence of multiple metastatic lesions.

During carotid arterial surgery and cardiac surgery, the electroencephalogram is of value in monitoring brain function. This is only true if the personnel are knowledgeable about the effect of anesthesia on the electroencephalogram. In rare instances where the clinician is unable to establish a differential diagnosis by the procedures described in the preceding pages, the combination of electroencephalography and echoencephalography may be helpful in the first few hours following the onset of the focal neurological abnormality.

If, within 36 hours of the onset, the electroencephalogram shows a focal abnormality and the echoencephalogram reveals a shift of 2 mm or more of the midline away from the side of the EEG focus, the statistical chances are that the lesion is an intracerebral hemorrhage or other expanding mass. However, a shift developing after 36 hours from the time of onset may be due to edema associated with infarction.

In medical centers where computerized axial tomography (e.g., EMI scanner, ACTA scanner, etc.) is available, there is very little need for echoencephalography other than its use in the evaluation of a patient in the emergency room.

Composite B-mode ultrasonography using radar techniques is being developed. The cervical portion of the carotid arteries has been scanned by several investigators but the method is still not feasible for clinical use.

Doppler techniques, including the use of a Doppler flowmeter, are under research and may be developed for clinical use.

7. Isotope brain scan

The static brain scan has become an established procedure for the detection of intracranial neoplasms. However, in medical centers where computerized axial tomography (e.g., EMI scanner, ACTA scanner, etc.) is available, there is now very little need for the static brain scan in the differential diagnosis of focal brain lesions. Non-neoplastic lesions, such as subdural hematomas, abscesses, cerebral infarcts or intracerebral hemorrhages, may at times produce focal abnormalities in the static brain scan. Brain scanning equipment is now widely available; however, it is unfortunate that in many instances clinical personnel competent to use and evaluate the scans for neurological differential diagnosis are not available.

The duration of time elapsing between the injection of the radionucleotide and the scanning is of paramount importance. Arteriovenous malformations are most commonly detected if the examination is done within 30 minutes of the injection, while cerebral infarcts are
more likely to be detected if the scanning is delayed two to four hours after the injection of the radioactive material. Commonly, cerebral infarcts are not identifiable by brain scanning until the third to eighth day following the occurrence of the lesion, and if a very definite focal abnormality is found within the first 24 hours from time of onset, brain neoplasm should be suspected.

Cerebral infarcts of about one inch or less in diameter do not visualize in a brain scan and, therefore, go undetected by this test. It is well established that a single abnormal brain scan confirms the existence of a focal brain lesion but gives no significant differential information concerning the pathology of the lesion.

Although this special procedure is safe, it is seldom necessary in making a differential diagnosis, adds considerable expense to the work-up of a patient, and has not in any way thus far replaced angiography.

Likewise, dynamic rapid serial scintigraphy using a gamma camera, thus giving a serial display of the images, provides much less precise information of clinical significance than is available through angiography. For instance, it is commonly impossible to determine whether a carotid artery is occluded or whether there is a severe degree of stenosis. Likewise, modest amounts of stenosis are generally missed by this test and ulceration of an atherosclerotic plaque in the internal carotid artery in the neck cannot be detected. Large and small vessel occlusive disease intracranially is commonly missed by this method.

D. Roentgen examination
1. Radiographs of the chest
Standard radiographs of the chest give valuable information concerning heart size and configuration, aortic pathology and pulmonary pathology, including infection and neoplasia.
2. Radiographs of the head
Plain radiographs of the skull give significant positive diagnostic information in relatively few patients with clinically typical occlusive cerebrovascular disease — cerebral infarction. However, common practice almost dictates such films be obtained. Occasionally evidence of cranial trauma or an intracranial mass lesion may be detected. Calcification of the carotid artery in the region of the sella is very common but does not give significant statistical evidence concerning the presence or absence of occlusive disease of a carotid artery at the place where the calcification is noted.
3. Angiography (cranial)
An important special procedure is cervical-cerebral angiography. There are numerous angiographical techniques for visualizing cervical-cerebral vasculature. The most important item, however, has to do with the skill of the personnel and their total familiarity with the method they are using. It is now fully apparent that in almost every patient where angiography is indicated, entire cerebral-cerebral circulation should be visualized with technically first-rate films. It is still the practice in some institutions to get films that show only a portion of the cervical part of the circulation, and in some instances the films of the intracranial vasculature are technically so poor that no significant information is available from them. Films of all portions of the cervical-cerebral circulation are necessary for the primary diagnosis as well as to ascertain whether a second or even more lesions may be present. If any kind of surgical intervention is being planned, it is important that the surgeon know the state of the collateral circulation and this can be obtained or evaluated only if fine films of all portions of the cervical-cerebral circulation are available.

Indications for angiography are:
1. Differential diagnosis of the brain pathology. Even with careful attention to all the items listed under history, general examination, neurological examination, and arteriographic examination and additional tests, there still remain about 5% of patients whose diagnosis is uncertain. In such instances, cervical-cerebral angiography is the best method of making a distinction between vascular occlusive disease, an intracranial expanding mass such as a hemorrhage, abscess or brain tumor, cerebral infarction and subdural hematoma, as well as demonstrating aneurysms and arteriovenous malformations.
2. Transient focal ischemic attacks — particularly the carotid system. In such instances cervical-cerebral angiography should be performed if there are one or more than one of: amaurosis fugax, bruit over the beginning of the internal carotid artery, retinal emboli, unilateral decrease in retinal artery pressure or ischemic retinopathy. If none of these are present, the likelihood of finding a lesion accessible to the surgeon is very small.
3. Selected instances of vertebrobasilar TIAs.
In some situations, it may be difficult to make a clinical distinction between the carotid and the vertebrobasilar system. If the TIAs are characteristic of those coming from the vertebrobasilar system, there is little merit to doing extensive angiography.
4. Very early progressing stroke or very frequent TIAs in the carotid system with, as a part of the history, amaurosis, an appropriate bruit, retinal emboli, etc.
5. Many patients with subarachnoid hemorrhage and some patients with intracerebral hemorrhage.
An uncertain indication is a long systolic or systolic-diastolic loud internal carotid artery bruit in patients who are being scheduled for major general surgery. If there is prolonged hypotension or very severe blood loss, the carotid stenosis may decrease blood supply to a focal region of brain to a critical level of ischemia. Recent observations suggest that such patients do not have an increased risk of stroke; therefore, arteriography is not necessary.
Contraindications are advanced forms of systemic disease. Acute myocardial infarction, allergy to contrast media, etc., must be considered contraindications or relative contraindications to cervical-cerebral angiography. If the cerebrovascular disease has produced a situation where there is cardiorespiratory depression, coma, or extraordinarily severe neurological deficit, angiography is not indicated.

E. Special procedures
1. Computerized soft tissue tomography (e.g., currently abbreviated to CT, EMI scanner, ACTA scanner, etc.)

Computerized tomography equipment employs a narrow beam of x-rays to scan a patient's head in a series of slices. The rays pass through the head and are detected by two sensing devices which always point toward the x-ray source. Both the x-ray tube and detector scan across the patient's head linearly and multiple readings of x-ray transmissions through the head are made during each traverse in the EMI scanner. At the end of each scan, the system is rotated 1° and the process is repeated. This continues for 180 scans when 28,000 readings will have been taken. These readings are then processed in a minicomputer, which calculates absorption values of the material within the slice from the 28,000 simultaneous equations. From these calculations a three-dimensional picture or matrix is built which in essence displays differential x-ray "densities" inside the head in a way never before possible. The matrix for each "brain slice" is displayed on a cathode ray viewing unit and a numerical print-out is also produced. The former is photographed by a Polaroid camera.

The system is about 100 times more sensitive than conventional x-ray systems and enables small variations in tissue density to be differentiated. The skin area irradiated is confined to a narrow band along the edge of the slice and the dosage is approximately equivalent to a conventional x-ray picture.

The cerebral ventricles are accurately visualized and there is good display of the subarachnoid spaces. Brain atrophy or any abnormality which alters ventricle size, configuration or position can be detected.

Extravasated blood has a density much greater than brain. A small focal hemorrhage is easily visualized and blood in the ventricles is easily detected.

Nonhemorrhagic cerebral infarcts can be seen; within a few hours of onset, edema around the infarct may be apparent. A very hemorrhagic infarct may be difficult to differentiate from a hemorrhage; the latter often has a rounded shape.

Brain tumors commonly can be distinguished from cerebral infarcts or intracerebral hemorrhage.

Circulating blood is not displayed; intracranial aneurysms and arteriovenous malformations will not be seen unless they contain clotted blood or calcium.

Subdural hematomas may be difficult to visualize, particularly when they are bilateral and do not produce shift of the ventricles.

It is apparent that computerized tomography will revolutionize the differential diagnosis of intracranial lesions.

2. Cerebral blood flow measurements

A variety of methods for measuring cerebral blood flow have been devised in the last 25 years. None of the currently used methods are accurate, reproducible and noninvasive. The methods have been used primarily in research and have provided information about cerebrovascular physiology. The most accurate techniques involve catheterization of the internal carotid artery and/or jugular vein with attendant risks. At the present time, these methods are not significantly helpful in clinical practice.

By numerous invasive techniques measurement of jugular venous oxygen tension, jugular venous lactate concentration, and arterial-jugular venous oxygen differences may be carried out. The data provided have occasioned much discussion but are not of significant value in the clinical care of patients with stroke.

3. Retinal circulation time

Ten percent fluorescein is injected into an antecubital vein and the time elapsing between the injection and the arrival of the dye in each retina is determined by ophthalmoscopic examination, using special filters. The test is cumbersome (three people are needed; one to observe each retina and one to inject the fluorescein) and does not give enough unique information to make it worthwhile. Development of appropriate devices for simultaneously photographing the retina might make the test more practical.

4. Thermography and thermometry

Thermography (as relating to stroke) measures the temperature (by recording infrared emissions from the skin) particularly over the mesial supraorbital area of the forehead which is supplied by terminal branches of the ophthalmic artery which originates from the internal carotid artery. Sometimes occlusion or very severe stenosis of the internal carotid artery is associated with reduced skin temperature in this particular area. The equipment is expensive and the test requires several minutes. Because of the expense and general cumbrousness of the equipment, the test has not become popular.

Thermistor recordings of temperature from various points across the forehead give similar information to thermography. This testing technique has not been of enough practical value, particularly in replacing other methods, to be useful.

A variety of thermochromic liquid crystals and paints which detect small changes in temperature have been investigated but have not become useful practically because of difficulties in their application compared to the value of the information obtained.

5. Retinal photography

It is possible to photograph the retina in color
and picture the changes of vessel occlusion, retinal infarction, hypertensive disease, diabetes, hemorrhage and the various types of emboli. However, the procedure is so cumbersome and expensive that it is not practical for screening or in the diagnosis of relatively large numbers of patients.

6. Tilt-table study

Lowering of the effective arterial perfusion pressure to the brain by tilting the patient to an upright position on a tilt-table has been suggested as a way to induce transient focal cerebral ischemic attacks. Because it is ineffective in almost all instances as well as requiring special equipment, the procedure is seldom used.

7. Phonocranioangiography

The recording and/or transmission of bruits is possible; the equipment and methods are not currently satisfactory for large-scale application of this method.

8. Electronystagmography

This is an electrical technique for recording nystagmus and other eye movements. It is possible to record nystagmus when the patient's eyes are closed. Therefore, the test is of value in recording caloric nystagmus which may be absent when the eyes are open but present when the eyes are closed. However, thus far there is no significant differential information about cerebrovascular disease obtained by this technique. Further study of electronystagmography may produce methods which make it of some clinical usefulness.

9. Doppler blood flow estimation

Continuous sonic energy has been used in an (noninvasive) attempt to map the morphology of extracranial arterial blood flow, i.e., carotids, supraorbital vessels, etc. Thus far the method has not produced satisfactory accurate results. Further refinement of the equipment may make this technique a useful one.

10. Ocular plethysmography (pulse propagation measurements)

The technique involves carrying light into the skin of the face; change in intensity of light backscattered from the skin is converted by a photocell to voltage for polygraphical recording during each cardiac cycle. By recording the electrocardiogram simultaneously, the interval from electrical ventricular systole (the R-wave) until the arrival of the opacity pulse wave at the site monitored can be measured. It is possible that further development of this instrumentation may produce a useful technique for the screening of patients with carotid occlusive disease.

11. Cranial impedance plethysmography (rheoencephalography)

This is a term commonly though incorrectly used in referring to a measurement of the impedance of the head to the passage of an electrical current which is applied externally. Although this technique has been under study for over a decade, it has not appeared to be of significant aid in the evaluation of stroke.

Part VI. Status of Patient (Performance and Placement)

Introduction

At many points in the natural history of stroke, it is desirable to estimate the performance ability of the patient. In this section, a classification of performance ability and placement potential is presented. Such assessment of the status of the patient serves as a significant guide to the course the disease process has taken, the management effort required, and the selection of a life-situation to which the patient can appropriately return. Ability of the patient to perform satisfying and productive activity in a supportive environment is a result of many intrinsic and extrinsic factors. Intrinsic factors may include such elements as the degree of general and focal cerebral damage, the residual neurological function, the integration of adaptive and substitutive patterns of function, the pre-existing level of development of intellect and skill, and developed attitudes and emotions. Extrinsic factors are even more varied and numerous and include such influences as the nature of the physical environment and living arrangements, the degree of family involvement and support, interpersonal relationships, the social and economic resources, and even attitudes of the family, friends, potential employers and the community at large. Prescription for continuing management in and outside of the health care facility must be based on information derived in detail from all available clinical and social sources. Classification of performance and placement provides a gross characterization of what life-tasks the patient is able to do and how he will be able to live.

Performance

In the context used in this Classification, performance is defined as the execution of set tasks. The design of a task can be structured in formal testing or can be variably determined by environmental or situational circumstances of the life-setting. The nature of the task determines the action to be taken by the patient. The patient must be able to recognize the demands made on him by the task, to select the appropriate action to accomplish the task and then to successfully carry out the selected action. Because the broad range of life-situations sets innumerable variations of task requirement, only generally recognized descriptions of performance that are of key significance in the care of the stroke patient form the basis of this Classification. It is further recognized that performance in a test setting varies according to test conditions and social situations and may not necessarily predict performance in other social contexts such as in the house, at work, or in the community at large. These considerations color but do not negate the value of classification by test and observation.

For purposes of this Classification, three descriptors of life-situations are selected as indices of performance status. These are: (a) activities of daily living
Activities of daily living (ADL) — a battery of tasks designed to provide an index of ability to perform self-sustaining activities that meet personal requirements in each day's life-experience. These essential activities are basic in everyone's day, relatively independent of geographical locale, physical environment, or social situation. They include such common activities as dressing, washing, eating, and traveling from place to place; they involve fundamental actions of body and limb movement, sitting balance, changing body position, standing, reaching, grasping, holding, and the like.

Avocational activities — those active pursuits of living not directly related to one's primary life work from which the individual gains significant participatory, creative, or productive satisfaction. These are not essential requirements of everyday living but have value in bringing purpose and fulfillment to the individual, his family and circle of friends. They involve multifaceted behavior patterns and at times employ complex hierarchies of physical and mental activities. Avocational activities include such pursuits as hobbies, travel, recreation, socializing and the like.

Occupation — the primary life-work to which the individual devotes the major portion of time, skill, energy, thought and effort and from which the individual derives role and status, physical and mental satisfaction, and economic support. In addition to recognized modes of remunerative employment, occupation includes homemaking, student life, retirement and other primary life-pursuits. In this Classification all such pursuits engaged in at the time of onset of stroke are considered the primary occupation.

Placement

The goal of care is ultimate return of the recovered patient to an optimal physical, mental, social, vocational and economic condition consistent with the patient's remaining performance abilities and requirements for continued health maintenance. Suitable physical environment, living arrangements, and social, economic and health care support must be selected. Medical status, performance ability and conditions in available environmental settings are variables that determine the placement status. Among the critical considerations leading to optimal placement are the suitability of the architecture and surrounding environment, the kind and frequency of personal supervision and direction required, and the level of requirement for continuing medical-nursing care. Possibilities for placement vary widely but may include placement in full competitive employment, placement in sheltered or selected work situations, resumption of homemaking duties, retirement from the work market, independent living in personal residence with full management responsibility, home care with family supervision, home care with outside assistance, domiciliary care, convalescent care, custodial care, or continued rehabilitation center or hospital care.

Part VI. Status of Patient (Performance and Placement)

Performance

Class I. No Significant Impairment — fully independent acts of daily living (ADL), pursues usual avocational activities, and returns to previous living site and occupation without modification.

Class II. Mildly Impaired — semidependent (requiring some assistance) in activities of daily living, and/or slight restriction of avocational activities, and/or able to return to previous occupation with some modification of the latter.

Class III. Moderately Impaired — semidependent (requiring lifting assistance) in activities of daily living, and/or considerable restriction of avocational activities, and/or unable to return to previous occupation with some modification of the latter.

Class IV. Severely Impaired — fully dependent in conduct of activities of daily living, and/or unable to participate in avocational activities, and/or unable to carry out any occupation.

Placement

Class A. No Limitation.

Class B. Mild Limitation — requires occasional supervision, and/or modified environment and/or occasional medical care.

Class C. Moderate Limitation — requires much supervision, and/or physical assistance or outside helpers and/or regularly available medical care.

Class D. Severe Limitation — requires constant or nearly constant attendance and/or immediately available medical-nursing care.
CLASSIFICATION AND OUTLINE OF CEREBROVASCULAR DISEASES II

Appendix

I. Clinical Stage
   A. Asymptomatic
   B. Focal cerebral dysfunction
      1. Transient attacks (transient ischemic attacks, TIA)
      2. Actively changing neurological deficit (progressing stroke)
      3. Prolonged neurological deficit (completed stroke)
   C. General cerebral dysfunction
      1. Transient
      2. Prolonged
         a. Acute onset
         b. Gradual progression

II. Pathophysiological Mechanisms
   A. Primary abnormalities of cerebral circulation (specify transient or persistent)
      1. Thrombosis
      2. Embolism
      3. Hemorrhage (specify — See Anatomy and Pathology)
      4. Compression
      5. Vasospasm
      6. Direction of flow
      7. Alteration in rate and/or volume
      8. Dissection of arterial wall
      9. Associated with arteriography
   B. Abnormalities of general circulation (specify transient or persistent)
      1. Hypotension (specify cause)
      2. Hypertension
   C. Alterations in blood (specify type)
   D. Alterations of metabolic demand (specify type)
   E. Possible predisposing factors
      1. Hypertensive disease
      2. Diabetes mellitus
      3. Cardiac disease
      4. Hyperlipidemia
      5. Other (specify)
   F. Unknown

III. Anatomy
   A. Blood vessels
      1. Arteries
         a. Ascending aorta
         b. Aortic arch
            1) Brachiocephalic a. (innominate a.)
            2) Common carotid a. (specify r. or l.)
               a) External carotid a. (specify r. or l.)
               b) Internal carotid a. (specify r. or l.)
                  (1) Ophthalmic a.
                  (2) Posterior communicating a.
                  (3) Anterior choroidal a.
                  (4) Anterior cerebral a.
                     (a) Anterior communicating a.
                  (5) Middle cerebral a.
            3) Subclavian a. (specify r. or l.)
               a) Vertebral a. (specify r. or l.)
                  (1) Posterior inferior cerebellar a.
               b) Basilar a.
                  (1) Anterior inferior cerebellar a.

In several places in this condensed version of the Classification, the symbols used are changed from the complete Classification. This is to make possible the use of the common items.
CLASSIFICATION AND OUTLINE OF CEREBROVASCULAR DISEASES II

(2) Internal auditory a.
(3) Pontine aa.
(4) Superior cerebellar a.
(5) Posterior cerebral a. (specify r. or l.)

c. Thoracic aorta
d. Abdominal aorta

2. Arterial collateral circulation
3. Arterial anomalies
4. Veins

B. Brain and spinal cord

1. Meninges
   a. Cranial
      1) Epidural
      2) Subdural
      3) Subarachnoid

2. Brain
   a. Hemisphere (specify r. or l.)
      1) Frontal lobe
      2) Temporal lobe
      3) Parietal lobe
      4) Occipital lobe
      5) Central white matter (specify by lobe)
      6) Internal capsule
      7) Thalamus
   b. Brain stem
      1) Midbrain
      2) Pons
      3) Medulla oblongata
   c. Cerebellum
d. Cranial nerves (specify by number and r. or l.)
e. Cerebral ventricles (specify by name and r. or l.)

3. Spinal cord

IV. Pathology

A. Pathological alterations in vessels

1. Arteries
   a. Congenital, developmental and inherited lesions
      1) Congenital aneurysms
      2) Congenital aneurysm, ruptured
   b. Inflammatory lesions (arteritides)
      1) Infectious
      2) Noninfectious
         a) Cranial arteritis (temporal arteritis)
   c. Trauma and physical agents
      1) Trauma to artery due to external forces
      2) Trauma due to angiography
      3) Trauma due to catheterization and other intra-arterial procedures
      4) Trauma due to surgery
   d. Arterial lesions due to blood dyscrasias
   e. Arterial lesions associated with metabolic abnormalities (including familial hypercholesterolemia, diabetes mellitus, etc.)
   f. Arterial lesions associated with drug toxicity, drug idiosyncrasy and unknown drug effects
      1) Anticoagulants
      2) Other
   g. Arterial embolism due to cardiac disease and diseases of extracerebral vessels
      1) Cardiac arrhythmias (specify basic disease)
      2) Valvular disease
      3) Myocardial infarction
   h. Arterial lesions associated with neoplastic disease
CLASSIFICATION AND OUTLINE OF CEREBROVASCULAR DISEASES II

i. Arterial lesions due to unknown causes
   1) Atherosclerosis
   2) Atherosclerotic stenosis
   3) Atherosclerotic occlusion

j. Arterial lesions associated with hypertension
2. Veins (specify) [See complete Classification]
3. Capillaries (specify) [See complete Classification]
4. Combined arterial, venous and capillary abnormalities
   a. Congenital, developmental and inherited lesions
      1) Arteriovenous fistula, congenital
      2) Arteriovenous fistula due to ruptured congenital aneurysm
      3) Vascular malformations (hamartomas, angiomas)

B. Pathological alterations in brain
1. Infarction (pale, hemorrhagic and mixed)
   a. Without vessel stenosis or occlusion
   b. With arterial stenosis or occlusion associated with: (list as under Pathological alterations in vessels, arteries — IV.A.1.)

2. Hemorrhage
   a. Without vessel type identified
   b. Of arterial origin (list as under Pathological alterations in vessels, arteries or use terms as follows)
      1) Anatomical site
         a) Intracerebral
            (1) With hypertension
            (2) Without hypertension
         b) Subarachnoid
         c) Subdural
         d) Intraventricular
      2) Etiology (specify)
   c. Of venous origin (specify)
   d. With capillary lesions [See IV.A.3.]
   e. With combined arterial, venous and capillary lesions [See IV.A.4.]

V. Clinical Phenomena (History, Physical Examination, Laboratory Examination, Roentgen Examination, Other)

A. History
1. Demographic
2. Family history
3. Past history
4. Present illness
   a. Transient ischemic attacks [See I.B.1.]
   b. Actively changing neurological deficit [See I.B.2.]
   c. Prolonged neurological deficit (RIND and completed stroke) [See I.B.3.]

B. Physical examination
1. General
2. Neurological
3. Vascular (neurovascular)

C. Laboratory examination
1. Urinalysis (specify abnormality) [See Outline p 606]
2. Blood (specify abnormality) [See Outline p 606]
3. Cerebrospinal fluid (specify abnormality) [See Outline p 607]
4. Electrocardiography (specify abnormality) [See Outline p 607]
5. Electroencephalography (specify abnormality) [See Outline p 608]
6. Echoencephalography (specify abnormality) [See Outline p 608]
7. Brain scan (specify abnormality) [See Outline p 608]

D. Roentgen examination
1. Chest
2. Head
3. Angiography (cranial) [See Outline p 609 for indications]
CLASSIFICATION AND OUTLINE OF CEREBROVASCULAR DISEASES

E. Special procedures [See Outline pp 610-611 for discussion]

1. Computerized axial tomography (computerized tomography, computed tomography) [See Outline]
2. Cerebral blood flow (specify method) (list results) (describe complications)
3. Retinal circulation time (specify method and results) (fluorescein)
4. Thermography and thermometry (specify method and results)
5. Retinal photography (specify method and results)
6. Tilt-table study (specify method and results)
7. Phonocraniography (specify method and results)
8. Electronystagmography
9. Doppler blood flow estimation (specify method, site, results)
10. Ocular plethysmography (specify method and results)
11. Cranial impedance plethysmography (rheoencephalography) (specify method and results)

VI. Status of Patient (Performance and Placement)

Performance

Class I. No significant impairment
   Fully independent acts of daily living (ADL), pursues usual avocational activities, and
   returns to previous living site and occupation without modification.

Class II. Mildly impaired
   Semidependent (requiring some assistance) in ADL, and/or slight restriction of avocational
   activities, and/or able to return to previous occupation with some modification of the latter.

Class III. Moderately impaired
   Semidependent (requiring lifting assistance) in ADL, and/or considerable restriction of
   avocational activities, and/or unable to return to previous occupation and must seek selec-
   tive occupation.

Class IV. Severely impaired
   Fully dependent in conduct of ADL, and/or unable to participate in avocational activities,
   and/or unable to carry out any occupation.

Placement

Class A. No limitation

Class B. Mild limitation
   Requires occasional supervision, and/or modified environment, and/or occasionally
   medical care.

Class C. Moderate limitation
   Requires much supervision, and/or physical assistance or outside helpers, and/or regularly
   available medical care.

Class D. Severe limitation
   Requires constant or nearly constant attendance and/or immediately available medical-
   nursing care.

The World Federation of Neurology Code for Grading Atherosclerosis

Grade 1+: Opacity involving only a small part of the vessel circumference. No lumen narrowing.

Grade 2+: (A) A diffuse thin plaque that does not involve the entire vessel circumference with minimal lumen
   narrowing. (B) A small thick plaque that produces less than 25% lumen narrowing.

Grade 3+: (A) A diffuse thin plaque involving the entire circumference of the vessel with mild lumen narrow-
   ing. (B) A localized thick plaque producing 25% to 50% lumen narrowing.

Grade 4+: (A) A thick plaque involving the entire circumference of the vessel with moderate or marked lumen
   narrowing (pipestem). (B) A localized thick plaque resulting in more than 50% lumen narrowing.

Wagener and Keith Classification of Retinal Vascular Disease

Group I: Retinal changes are minimal and consist of mild narrowing and mild sclerosis of arterioles.

Group II: Changes are somewhat more advanced; patient's general health is good.

Group III: Pronounced abnormality of small retinal vessels and small artery may be obstructed; there are
   definitely localized narrowed areas in the retinal vessels, hemorrhages and exudates. May have mild alteration of
   vision.

Group IV: Similar to Group III but with florid hemorrhages, retinal edema and sometimes swelling of the
   nerve head. Visual impairment is present and neurological symptoms are common.