Introduction

The purpose of this Classification of Cerebrovascular Diseases III is to delineate the types of cerebrovascular disease in clinical and pathological terminology so that all or any portion of the classification may be used by clinicians, surgeons, pathologists, or physiatrists as well as by other groups interested in the subject. It is also intended to define clinical and pathological diagnostic terms for common use. The background material is intended as supporting information for the clinical and pathological classifications.

The second Ad Hoc Committee on Cerebrovascular Diseases developed a classification, published in 1975, that provided a clinical and pathological framework for cerebrovascular diseases at that time. The Classification and Outline of Cerebrovascular Diseases II was published near the beginning of an explosion in technological developments that have dramatically enhanced our capability to evaluate patients with these disorders. Technological advances will continue, but the present Classification of Cerebrovascular Diseases III and background material reflect the current state of our knowledge.

By the term “cerebrovascular diseases” we refer to all disorders in which there is an area of brain transiently or permanently affected by ischemia or bleeding and/or in which one or more blood vessels of the brain are primarily impaired by a pathological process. The terms “cerebrovascular” and “cerebral” are used in the original Latin sense, referring to the entire brain and not merely to the hemispheres of the forebrain. The term “stroke” is commonly used as a generic term to represent any one or all of a group of disorders, including cerebral infarction, intracerebral hemorrhage, or subarachnoid hemorrhage.

The background material supporting the Classification of Cerebrovascular Diseases III recognizes the potential impact of preventive measures on stroke occurrence. It also recognizes the enhancements of our capabilities to define anatomic lesions in the brain and in the blood vessels in living patients by technological developments.

The outline summarizes the classification of the clinical and pathologic disorders and the background material on prevention, clinical assessment, evaluation, status of the patient following stroke, and anatomy. The anatomy and pathology are sufficiently defined in the outline; therefore, the descriptive material, which follows the outline precisely, does not provide information for anatomy and pathology.

The document begins with the classification and then provides an orderly format for proceeding from prevention through clinical assessment and evaluation to the poststroke status of the patient and to the organization of the anatomical terms. It is intended that the classification, or the supporting material, should be useful to the various disciplines that are concerned with clinical service, training, or research in the cerebrovascular diseases.

Outline

I. Clinical Disorders
   A. Asymptomatic
   B. Focal Brain Dysfunction
      1. Transient Ischemic Attacks (TIAs)
         a. Carotid system
         b. Vertebralbasilar system
         c. Both
         d. Uncertain location
         e. Possible TIA
      2. Stroke
         a. Temporal profile
            1) Improving
            2) Worsening
            3) Stable stroke
         b. Types of stroke (for details see II. Pathology)
            1) Brain hemorrhage
            2) Subarachnoid hemorrhage (SAH)
            3) Intracranial hemorrhage from arteriovenous malformation (AVM)
            4) Brain infarction
               a) Mechanisms
                  (1) Thrombotic
                  (2) Embolic
                  (3) Hemodynamic
b) Clinical categories
   (1) Atherothrombotic
   (2) Cardioembolic
   (3) Lacunar
   (4) Other (see II. Pathology)
c) Symptoms and signs by site (distribution)
   (1) Internal carotid artery
   (2) Middle cerebral artery
   (3) Anterior cerebral artery
   (4) Vertebrobasilar system
       (a) Vertebral artery
       (b) Basilar artery
       (c) Posterior cerebral artery

C. Vascular Dementia
D. Hypertensive Encephalopathy

II. Pathology
A. Pathologic Alterations in Heart and Blood Vessels
   1. Arteries (and arterioles, when applicable)
      a. Congenital, developmental, and inherited lesions
         1) Aplasia of artery
         2) Hypoplasia of artery
         3) Anomaly of artery (fetal form)
         4) Redundancy (loops), dilatation, or elongation of artery
         5) Genetically determined defects in arteries
            a) Marfan's syndrome (arachnodactyly)
            b) Ehlers-Danlos syndrome
            c) Pseudoxanthoma elasticum
            d) Others
      6) Vascular malformations
         a) AVM
         b) Cavernous hemangioma
         c) Capillary telangiectasia
      b. Saccular aneurysm
         1) Unruptured
         2) Ruptured
      c. Atherosclerosis
         1) Stenosis
         2) Occlusion
         3) Ulceration of plaque
         4) Hemorrhage in plaque
         5) Dilatation, ectasia, fusiform aneurysm
      d. Hypertensive alterations
         1) Arteriolar sclerosis
         2) Hyaline medial degeneration with or without fibrinoid change
         3) Charcot-Bouchard microaneurysm
         4) Other
      e. Arterial embolism—Sources
         1) Heart
            a) Cardiac valvular disease (including mitral and aortic valve disease, prosthetic
               valve, mitral valve prolapse)
               (1) Septic
               (2) Nonbacterial thrombotic endocarditis
               (3) Libman-Sacks endocarditis
               (4) Other
            b) Cardiac atrium (including atrial fibrillation, myxoma, aneurysm)
            c) Cardiac ventricle (including myocardial infarction, aneurysm, dyskinesia of wall,
               ventricular assist device)
         2) Aorta and major branches
            a) Ulcerative plaque
b) Plaque plus thrombus
c) Fibromuscular dysplasia
d) Spontaneous arterial dissection
3) Paradoxical embolus
   a) Venous thrombosis
   b) Other
4) Tumor
5) Trauma-related embolism
   a) Fat
   b) Bone marrow
   c) Air
   d) Arterial dissection
f. Inflammatory arteritis
   1) Infectious
      a) Etiology
         (1) Bacterial
         (2) Fungal
         (3) Viral
         (4) Rickettsial
         (5) Other
      b) Pathogenesis
         (1) Meningoencephalitis
         (2) Septic emboli
         (3) Other
      c) Complications
         (1) Thrombosis
         (2) Hemorrhage
         (3) Scar
         (4) Pseudoaneurysm
   2) Noninfectious
      a) Etiology
         (1) Cranial (temporal) arteritis
         (2) Periarteritis nodosa
         (3) Other*
      b) Complications
         (1) Thrombosis
         (2) Hemorrhage
         (3) Scar
         (4) Pseudoaneurysm
g. Toxic, metabolic, and systemic disorders
   1) Thrombosis
      a) Dehydration
      b) Drugs (specify)
      c) Blood dyscrasias (specify type)
      d) Other
   2) Hemorrhage
      a) Anticoagulants (specify)
      b) Thrombolytic drugs
      c) Sympathomimetic amines (including illicit drugs)
      d) Heavy metals
      e) Blood dyscrasias
      f) Other
   3) Calcification or mineralization
      a) Hypoparathyroidism
      b) Hypervitaminosis D
      c) Mönckeberg's sclerosis (calcific medial arteriosclerosis)
      d) Mineralization (ferrugination, calcification, siderocalcification)
      (1) Infection (specify)

*Although common terminology employs the term "lupus vasculitis," a true inflammatory arteritis in the central nervous system in patients with systemic lupus erythematosus is exceedingly rare and therefore is not specifically indexed here.
h. Trauma or physical agents
   1) Mechanisms
      a) External forces, bony anomalies, fractures, dislocations, degenerative bone disease, fibrosis
      b) Intra-arterial interventional procedures
         (1) Angiography
         (2) Cardiac catheterization
         (3) Balloon occlusion
         (4) Transluminal angioplasty
         (5) Pump oxygenation
         (6) Embolization
         (7) Drug therapy
   c) Surgery or interventional procedures
      (1) Reconstructive and reparative arterial surgery
      (2) Other surgical procedures
         (a) Occlusion (ligation, clamp, etc.)
         (b) Rupture or accidental division
   d) Brain herniation (transientorial, subfalcial, foramen magnum, etc.)
   e) Radiation
      (1) Acute
      (2) Delayed

2) Pathologic consequences
   a) Vasospasm
   b) Compression
   c) Intramural hemorrhage
      (1) With dissection
   d) Rupture of atherosclerotic plaque
      (1) With atheromatous embolization
   e) Necrosis
   f) Thrombosis
   g) Fibrosis, hyalinization
   h) Stenosis
   i) Accelerated atherosclerosis
   j) Embolization due to foreign materials
   k) Pseudoaneurysm

i. Neoplastic disease
   1) Thrombosis
      a) Intracranial neoplasm (primary or secondary)
      b) Extracranial neoplasm
      c) Intravascular neoplasm
   2) Hemorrhage
      a) Intracranial neoplasm (primary or secondary)
      b) Extracranial neoplasm
   3) Embolism
      a) Intracranial neoplasm (primary or secondary)
      b) Extracranial neoplasm
   4) Intravascular malignant lymphomatosis (malignant angioendotheliosis)

j. Amyloid (congophilic) angiopathy
   1) Location
      a) Meningeal
      b) Parenchymal
   2) Secondary effects
      a) Hemorrhage
      b) Focal atrophy (scar)
      c) Infarct
   3) Associated factors
      a) Familial
b) Senile dementia of Alzheimer's type

c) Other

k. Miscellaneous disorders of arteries
   1) Fibromuscular dysplasia
   2) Thromboangiitis obliterans
   3) Hemorrhagic dissection of arterial wall
   4) Other

2. Veins (and venules, when applicable) and Venous Sinuses
   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
      4) Malformation
         a) AVM
         b) Dural AVM
         c) Venous angioma (varix)
   b. Inflammatory lesions of veins
      1) Infectious
         a) Etiology
            (1) Bacterial
            (2) Fungal
            (3) Viral
            (4) Other
         b) Pathogenesis
            (1) Meningoencephalitis
            (2) Trauma
            (3) Other
         c) Complications
            (1) Thrombosis
            (2) Hemorrhage
      2) Noninfectious
         a) Venous lesions due to toxic, metabolic, or systemic disorders
            (1) Thrombosis
               (a) Dehydration
               (b) Drugs (specify)
               (c) Blood dyscrasias (specify type)
               (d) Other
            (2) Hemorrhage
               (a) Anticoagulants
               (b) Heavy metals
               (c) Blood dyscrasias
               (d) Other
   c. Trauma
      1) Mechanisms
         a) Surgery involving veins
            (1) Embolization (specify)
            (2) Thrombosis
            (3) Thrombosis in venous anastomosis or grafts
         b) Other surgical procedures
            (1) Occlusion (ligation, clamp, etc.)
            (2) Rupture or accidental division
            (3) Intramural hemorrhage
            (4) Thrombosis
               (a) With embolization
            c) Brain herniation (subfalcial, foramen magnum, etc.)
               (1) Thrombosis
               (2) Hemorrhage
         d) Radiation
            (1) Postirradiation thrombosis
            (2) Postirradiation scar (fibrosis)
2) Pathologic consequences
   a) Compression
   b) Hemorrhage
   c) Thrombosis

d. Calcification or mineralization
   1) Hypoparathyroidism
   2) Hypervitaminosis D
   3) Infection
   4) Other

e. Associated with neoplastic disease
   1) Thrombosis
      a) Intracranial neoplasm (primary or secondary)
      b) Extracranial neoplasm
   2) Hemorrhage
      a) Intracranial neoplasm (primary or secondary)
      b) Extracranial neoplasm
   3) Embolism
      a) Intracranial neoplasm (primary or secondary)
      b) Extracranial neoplasm

f. Other

3. Capillaries
   a. Petechiae associated with inflammation
      1) Infectious
      2) Noninfectious
   b. Petechiae due to trauma and physical agents
      1) External trauma
      2) Remote effects of trauma
         a) Fat embolization
         b) Bone marrow embolization
         c) Air embolization
      3) Angiography
      4) Therapeutic intravascular procedures
      5) Surgery
      6) Brain herniation
      7) Heat stroke
      8) Radiation
   c. Capillary lesions associated with toxic, metabolic, or systemic disorders
      1) Petechiae
         a) Anticoagulants
         b) Drugs
         c) Heavy metals or toxins (specify)
         d) Metabolic disturbances
         e) Blood dyscrasias (including thrombocytopenia, disseminated intravascular coagulation, and thrombotic thrombocytopenic purpura)
         f) Other
      2) Thrombosis
         a) Dehydration
         b) Drugs
         c) Blood dyscrasias (see above)
         d) Other
      3) Calcification or mineralization
         a) Hypoparathyroidism
         b) Hypervitaminosis D
         c) Infection
         d) Other
   d. Miscellaneous capillary lesions
   e. Other

B. Pathologic Alterations in Brain and Spinal Cord

1. Infarct*

*Subcortical cystic infarcts <1.5 cm in diameter are generally referred to as "lacunes."
a. Etiology (see II. Pathology, A. Pathologic Alterations in Heart and Blood Vessels)
b. Anatomic site (specify)
c. Size
d. Type
   1) Nonhemorrhagic (bland)
   2) Hemorrhagic
e. Age
   1) Recent
   2) Organizing
   3) Cystic
f. Mechanisms
   1) Thrombotic (commonly occurs in association with atherosclerotic plaque but may be precipitated by intravascular clotting abnormality)
   2) Embolic (due to occlusion of artery distal to point where adequate collateral blood flow is available)
   3) Hemodynamic (most commonly occurs when there is severe stenosis or occlusion of proximal arterial supply to portion of brain and collateral compensatory blood flow is inadequate or when global cerebral perfusion is critically decreased)

2. Hemorrhage
a. Etiology (see II. Pathology, A. Pathologic Alterations in Heart and Blood Vessels)
b. Anatomic site
   1) Epidural
c. Size
d. Age
   1) Recent
   2) Organizing
   3) Cystic

3. Ischemic neuronal necrosis
a. Etiology
   1) Cardiac arrest
   2) Systemic hypotension
   3) Vascular lesion (see II. Pathology, A. Pathologic Alterations in Heart and Blood Vessels)
b. Anatomic site(s)
c. Age

4. Ischemic leukoencephalopathy*
a. Etiology
   1) Vascular lesions
   2) Systemic disorders
      a) Cardiac arrest
      b) Systemic hypotension
      c) Other
b. Pathology
   1) Dilated perivascular spaces (état crible)
   2) Demyelination (with axonal preservation)
   3) Atrophy with loss of axons and myelin
   4) Necrosis
      a) Well-circumscribed (infarcts)
      b) Partial, poorly circumscribed
c. Anatomic site(s) (including subependymal region)
d. Age

*Subcortical leukoencephalopathy (arterial and arteriolar sclerosis) often is referred to as "Binswanger's disease" when it is associated with hypertension and dementia.
Background Material

III. Risk Factors and Prevention
   A. Characteristics and Lifestyle
      1. Definite
         a. Cigarette smoking
         b. Alcohol consumption
         c. Drug abuse
         d. Age
         e. Sex
         f. Race
         g. Familial factors
      2. Possible
         a. Oral contraceptive use
         b. Diet
         c. Personality type
         d. Geographic location
         e. Season
         f. Climate
         g. Socioeconomic factors
         h. Physical inactivity
         i. Obesity
         j. Abnormal blood lipids
         k. Maternal mortality
   B. Disease or Disease Markers
      1. Definite
         a. Hypertension
         b. Cardiac disease
         c. TIA
         d. Elevated hematocrit
         e. Diabetes mellitus
         f. Sickle cell disease
         g. Elevated fibrinogen concentration
         h. Migraine and migraine equivalents
      2. Possible
         a. Hyperuricemia
         b. Hypothyroidism
   C. Asymptomatic Structural Lesions
      1. Physical Examination
         a. Bruit (cervical, orbital, cranial)
         b. Retinal emboli
         c. Blood pressure differences between arms
         d. Reduced pressure on oculoplethysmography
      2. Imaging
         a. Silent infarction or hemorrhage (magnetic resonance imaging [MRI], computed tomography [CT])
         b. AVM, aneurysm, hamartoma
         c. Atherosclerosis with arterial stenosis
         d. Fibromuscular dysplasia, dissection
   D. Multiple Factors in Combination

IV. Clinical Assessment
   A. History
   B. Physical Examination
      1. General
      2. Neurologic
      3. Vascular
a. Palpation
b. Auscultation

4. Ophthalmoscopy

V. Evaluation
A. Laboratory
1. Laboratory Studies Usually Ordered
   a. Urinalysis—sugar and protein contents, specific gravity, sediment microscopy
   b. Blood tests
      1) Serologic test for syphilis
      2) Complete blood count with differential and platelet counts
      3) Hemoglobin and hematocrit
      4) Erythrocyte sedimentation rate
      5) Blood sugar concentration
      6) Serum creatinine and blood urea nitrogen concentrations
      7) Serum cholesterol and triglyceride (with fractionation of cholesterol if elevated)
         concentrations
      8) Serum electrolyte concentrations
      9) Prothrombin time and activated partial thromboplastin time
   b. Laboratory Studies Ordered Selectively
      a. Urine—24-hour urine for metanephrine or vanillylmandelic acid concentrations (to rule
         out pheochromocytoma); porphobilinogen, lead concentrations, urine osmolality; tests for
         drug abuse
      b. Blood
         1) Serum protein concentration
         2) Coagulation studies
         3) Other blood studies—blood gases, liver function studies, thyroid function studies,
            calcium and catecholamine concentrations, hemoglobin electrophoresis, special
            serologic studies for collagen-vascular diseases, tests for human immunodeficiency
            virus
      c. Cerebrospinal fluid (CSF)
         1) Pressure
         2) Color
         3) Cell count and differential
         4) Protein content
         5) Glucose content
         6) Test for syphilis
         7) Bacteriologic and serologic studies
B. Neurophysiologic
1. Electroencephalography (EEG)
C. Cardiovascular
1. Cardiovascular Studies Usually Ordered
   a. Standard electrocardiography (ECG)
   b. Chest roentgenography
2. Cardiovascular Studies Ordered Selectively
   a. Long-term ECG monitoring
   b. Echocardiography (echo)
   c. Other
      1) Nuclear cardiology
         a) Myocardial infarct-avid scanning
         b) Myocardial perfusion imaging
         c) Radionuclide angiography
      2) Invasive investigations
         a) Cardiac catheterization and angiography
         b) Intracardiac electrophysiology
         c) Swan-Ganz catheterization
      3) Long-term blood pressure monitoring
D. Brain Imaging
1. CT
2. MRI
3. Other
E. Vascular
1. Noninvasive
   a. Indirect tests
      1) Periorbital circulation
         a) Directional continuous-wave Doppler sonography
         b) Photoplethysmography
      2) Orbital circulation
         a) Quantitative oculopneumoplethysmography
         b) Pulse-delay oculoplethysmography
         c) Ophthalmodynamometry
   b. Direct tests
      1) Cervical carotid artery
         a) B-mode ultrasonography
         b) Duplex ultrasonography
         c) Color duplex sonography
         d) Sequential ultrasonic arteriography
         e) Continuous-wave Doppler sonography
         f) Quantitative phonoangiography
      2) Intracranial vessels
         a) Transcranial Doppler sonography
   c. Utilization characteristics

2. Invasive — Angiography
   a. Intra-arterial
      1) Conventional
      2) Digital subtraction
      b. Intravenous
   c. Utilization characteristics

F. Cerebral Blood Flow and Metabolism

VI. Status of Patient Following Stroke
A. Cognitive Status
B. Communicative Capabilities
C. Functional Abilities
   1. Retrospective Evaluation
   2. Prospective Evaluation

VII. Anatomy
A. Blood Vessels
   1. Arteries
      a. Ascending aorta
      b. Aortic arch
         1) Brachiocephalic (innominate) a.
         2) Common carotid a. (specify r. or l.)
            a) External carotid a. (specify r. or l.)
               (1) Superior thyroid a.
               (2) Lingual a.
               (3) Facial (external maxillary) a.
               (4) Occipital a.
                  (a) Meningeal branch
               (5) Posterior auricular a.
               (6) Ascending pharyngeal a.
                  (a) Posterior meningeal aa.
               (7) Superficial temporal a.
               (8) Maxillary (internal maxillary) a.
                  (a) Middle meningeal a.
                  (b) Infraorbital a.
      b) Internal carotid a. (specify r. or l.)
         (1) Carotid sinus portion (Part I) (from bifurcation to end of bulbous dilatation)
(2) Cervical portion (Part II) (from end of sinus portion to entry into petrous bone)

(3) Petrous portion (Part III) (from entry into petrous bone to exit from petrous bone)
   (a) Caroticotympanic aa.
   (b) Other inconstant arterial branches

(4) Cavernous sinus portion (Part IV) (from exit from petrous bone through cavernous sinus course)
   (a) Hypophyseal aa.
   (b) Semilunar aa.
   (c) Anterior capsular a.

(5) Intracranial portion (Part V) (from exit from cavernous sinus to trifurcation; also termed intradural portion or supraclinoid portion)
   (a) Ophthalmic a.
         [a] Nasal branches
         [b] Temporal branches
   (b) Posterior communicating a.
   (c) Anterior choroidal a.
   (d) Anterior cerebral a.
      [1] A1 segment (proximal horizontal portion from origin to anterior communicating a.)
         [a] Anteromedial (perforating) aa.
         [b] Recurrent a. of Heubner
         [c] Anterior communicating a.
      [2] A2–3 segment (from origin of anterior communicating a. to distal anterior cerebral a. bifurcation [ascending portion])
         [a] Medial striate a. or aa.
         [b] Orbital a.
         [c] Frontopolar a.
      [3] A4–5 segment (horizontal dorsal portion superior to corpus callosum)
         [a] Callosomarginal a.
            1. A4 (anterior frontal portion)
            2. A5 (posterior parietal portion)
         [b] Pericallosal a.
   (e) Middle cerebral a.
      [1] M1 segment (from origin to entry into sylvian fissure)
         [a] Lenticulostriate aa.
         [b] Orbitofrontal aa.
      [2] M2 segment (short insular segment to trifurcation or first bifurcation)
      [3] M3 segment (opercular segment of middle cerebral a. branches)
         [a] Anterior temporal a. or aa.
         [b] Ascending frontal a.
            1. Prerolandic aa.
            2. Rolandic aa.
         [c] Parietotemporal aa.
         [d] Posterior temporal a.
      [4] M4 segment (terminal segment)
         [a] Ascending parietal (postrolandic) a.
            1. Posterior parietal (including angular a.)

3) Subclavian a. (specify r. or l.)
a) Vertebral a.
   (1) Proximal portion (Part I) (from origin to entry into transverse foramen of sixth cervical vertebra or, in some cases, a lower or higher vertebra)
   (2) Intracranial portion (Part II) (from entry into C6 transverse foramen to exit from C2 transverse foramen)
      (a) Spinal aa. (specify segments)
      (b) Muscular aa.
   (3) Distal extracranial portion (Part III) (from exit from C2 transverse foramen, around posterior aspect of atlas [C1] to point of entry into dura)
      (a) Muscular aa.
   (4) Intracranial portion (Part IV) (from entry into dura to juncture with basilar a.)
      (a) Anterior spinal a.
      (b) Posterior inferior cerebellar a.
          [3] Lateral branch
      (c) Posterior meningeal a.
      (d) Posterior spinal a.

b) Basilar a.
   (1) Anterior inferior cerebellar a.
   (2) Internal auditory a.
   (3) Pontine aa.
   (4) Superior cerebellar a.
   (5) Posterior cerebral a.
      (a) P1 segment (mesencephalic a.) (from origin at basilar a. to posterior communicating a.)
          [1] Posteromedial (perforating) aa.
      (b) P2 segment (from posterior communicating a. to posterior margin of midbrain)
          [2] Posterolateral aa. (thalamic, thalamogeniculate aa.)
      (c) P3 segment (from posterior margin of midbrain to anterior limit of calcarine fissure)

b) Thyrocervical trunk
(1) Thyroid branches
(2) Cervical branches
d) Internal mammary a.
c) Thoracic aorta
   1) Intercostal aa.
      a) Spinal aa. (specify segments, e.g., artery of Adamkiewicz, which may be from T9–L1)
   2) Subcostal aa.
      a) Spinal aa. (specify segments) (anterior spinal a. listed above)
d) Abdominal aorta
   1) Lumbar aa.
      a) Spinal aa. (specify segments) (anterior spinal a. listed above)

2. Arterial Collateral Circulation
a. Extracranial–extracranial (specify arterial branches involved)
b. Extracranial–intracranial (specify arterial branches involved)
c. Intracranial–intracranial
   1) Circle of Willis (specify arterial branches)
   2) Leptomeningeal anastomoses of anterior, middle, and posterior cerebral aa. and superior, anterior inferior, and posterior inferior cerebellar aa. (see above)
d. Persisting embryonic arteries
   1) Otic a.
   2) Trigeminal a. (carotid–basilar anastomosis)
   3) Hypoglossal a.
3. Arterial Anomalies
   a. Aortic arch
      1) Common origin of left common carotid a. and brachiocephalic a.
      2) Origin of left common carotid a. from brachiocephalic a.
      3) Origin of left vertebral a. from aortic arch
      4) Origin of right subclavian a. distal to left subclavian a. (aberrant r. subclavian a.)
      5) Other
   b. Circle of Willis
      1) Posterior communicating aa.
         a) Bilaterally small, hypoplastic, or atretic
         b) Unilaterally small, hypoplastic, or atretic (specify r. or l.)
      2) Anterior communicating a.
         a) Absent
         b) Bifid or multiple and small
      3) Posterior cerebral a. origin from internal carotid a. (specify if r., l., or bilateral)
      4) Other
   c. Leptomeningeal branches of major cerebral aa.

4. Veins
   a. Internal cerebral vv.
      1) Septal vv.
      2) Choroid vv.
      3) Thalamostriate (striothalamic) vv.
      4) Internal cerebral vv.
      5) Great cerebral v. (v. of Galen)
   b. External cerebral vv.
      1) Superior cerebral vv.
      2) Superficial middle cerebral (superficial sylvian) v.
         a) Superior anastomotic (Trolard’s) v.
         b) Inferior anastomotic (Labbé’s) v.
      3) Inferior cerebral vv.
         a) Basal (Rosenthal’s) v.
         b) Deep middle cerebral (deep sylvian) v.
         c) Anterior cerebral vv.
         d) Striate vv.
   c. Cerebellar vv.
      1) Superior cerebellar vv.
      2) Inferior cerebellar vv.
   d. Dural venous sinuses
      1) Superior sagittal s.
      2) Inferior sagittal s.
      3) Transverse s.
      4) Sigmoid s.
      5) Straight s.
      6) Occipital s.
      7) Confluence of the sinuses (torcular herophili)
      8) Cavernous s.
         a) Sphenoparietal s.
         b) Superior ophthalmic v.
         c) Inferior ophthalmic v.
      9) Intercavernous (circular) s.
     10) Basilar plexus
     11) Superior petrosal s.
     12) Inferior petrosal s.
         a) Internal auditory vv.
   e. Emissary vv.
      1) Mastoid emissary v.
      2) Parietal emissary v.
      3) Plexus of hypoglossal canal
      4) Condylar emissary v.
      5) Plexus of foramen ovale
6) Plexus of internal carotid vv.
7) Occipital emissary v.
f. Diploic vv.
1) Frontal v.
2) Anterior temporal v.
3) Posterior temporal v.
4) Occipital v.
g. Internal jugular v.
h. External jugular v.
i. Subclavian v.
j. Brachiocephalic (innominate) v.
k. Superior vena cava
l. Spinal vv.
1) Anterior spinal v.
a) Sulcal vv.
2) Posterior spinal vv.
3) Vertebral venous plexuses
m. Inferior vena cava

B. Brain and Spinal Cord
1. Meninges
   a. Cranial dura mater (specify site according to lobes of brain or cranial bony fossae)
      1) Epidural space, cerebral
      2) Subdural space, cerebral
   b. Spinal dura mater
      1) Epidural space, spinal
      2) Subdural space, spinal
   c. Leptomeninges (arachnoid and pia mater)
      1) Subarachnoid space, cerebral
      2) Subarachnoid space, spinal

2. Brain
   a. Hemisphere (specify r. or l.)
      1) Frontal lobe
         a) Frontal pole
         b) Precentral gyrus
         c) Superior frontal gyrus
         d) Middle frontal gyrus
         e) Inferior frontal gyrus
            (1) Opercular portion
            (2) Triangular portion
            (3) Orbital portion
         f) Orbital (orbitofrontal) gyri
         g) Gyrus rectus
      2) Temporal lobe
         a) Temporal pole
         b) Superior temporal gyrus
            (1) Transverse temporal (Heschl’s) gyri
         c) Middle temporal gyrus
         d) Inferior temporal gyrus
         e) Fusiform gyrus
         f) Hippocampal gyrus
         g) Uncus
      3) Parietal lobe
         a) Postcentral gyrus
         b) Superior parietal lobule
         c) Inferior parietal lobule
         d) Supramarginal gyrus
         e) Angular gyrus
      4) Occipital lobe
         a) Occipital pole
         b) Lateral occipital gyri
         c) Calcarine gyri (area striata)
d) Cuneus
5) Medial hemisphere surfaces
   a) Cingulate gyrus
   b) Corpus callosum
   c) Other (specify)
6) Insula
7) Central white matter (specify by lobe)
8) Internal capsule
   a) Anterior limb
   b) Genu
   c) Posterior limb
9) Thalamus
10) Corpus striatum
    a) Caudate nucleus
    b) Lentiform nucleus
       (1) Putamen
       (2) Globus pallidus
11) Hypothalamus
12) Other (specify)
b. Brainstem
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) Abducent n. (VI)
7) Facial n. (VII)
8) Acoustic (vestibulocochlear) n. (VIII)
   a) Cochlear division
   b) Vestibular (labyrinthine) division
9) Glossopharyngeal n. (IX)
10) Vagus n. (X)
11) Accessory (spinal accessory) n. (XI)
12) Hypoglossal n. (XII)
e. Cerebral ventricles
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1) Lateral ventricle (specify r. or l.)
2) Third ventricle
3) Cerebral (sylvian) aqueduct
4) Fourth ventricle
5) Choroid plexus

3. Spinal Cord
   a. Gray matter (specify level)
   b. White matter (specify level)
   c. Dorsal roots (specify level)
   d. Ventral roots (specify level)
   e. Dorsal root ganglion

Classification

I. Clinical Disorders
   A. Asymptomatic
      This category includes patients who have no cerebral or retinal symptoms of vascular disease.
   B. Focal Brain Dysfunction
      1. TIAs
         These are brief episodes of focal loss of brain function, thought to be due to ischemia, that can usually be localized to that portion of the brain supplied by one vascular system (left or right carotid or vertebrobasilar system) and for which no other cause can be found. Arbitrarily, by convention, episodes lasting <24 hours are classified as TIAs although the longer the episode the greater the likelihood of finding a cerebral infarct by CT or MRI. TIAs commonly last 2–15 minutes and are rapid in onset (no symptoms to maximal symptoms in <5 minutes and usually in <2 minutes). Fleeting episodes lasting only a few seconds are not likely to be TIAs. Each TIA leaves no persistent deficit, and there are often multiple attacks. There are unusual instances that fall outside this definition.

      Left carotid system TIAs typically have a rapid onset to maximum symptoms in <2 minutes of one or more of the following:
         a. Motor dysfunction (dyarthria, weakness, paralysis, or clumsiness of the right extremities and/or face)
         b. Loss of vision in the left eye (amaurosis fugax) or, rarely, the right field of vision (homonymous hemianopsia)
         c. Sensory symptoms (numbness including loss of sensation or paresthesia involving the right upper and/or lower extremity and/or face)
         d. Aphasia (language disturbance)

      Right carotid system TIAs produce similar symptoms on the opposite side, except that aphasia occurs only when the right hemisphere is dominant for speech.

      Vertebrobasilar system TIAs are characterized by the rapid onset of symptoms in <2 minutes of:
         a. Motor dysfunction (weakness, paralysis, or clumsiness of any combination of upper and lower extremities and face, left and/or right
         b. Sensory symptoms (loss of feeling, numbness, or paresthesia) involving the left, right, or both sides
         c. Loss of vision in one or both homonymous visual fields
         d. Loss of balance, vertigo, unsteadiness or disequilibrium, diplopia, dysphagia, or dysarthria are characteristic but are not to be considered as a TIA when any one of these symptoms occurs alone

      Dysarthria can accompany either carotid or vertebrobasilar TIAs.

      Most patients have TIAs that include motor symptoms. Sensory symptoms involving only part of one extremity or only one side of the face during a single attack not accompanied by other symptoms are difficult to interpret with certainty. It is common for amaurosis fugax (monocular blindness) to occur without other symptoms during the episode. Occasional patients have only episodes of aphasia. An attack that does not include either motor defect,
visual loss, or aphasia should be reviewed even more carefully before accepting TIA as the diagnosis. These clinical phenomena generally represent a decrease or absence of function. With repeated attacks, the symptoms may be varied or mostly stereotyped. Monocular visual loss is likely to last only a few minutes.

The diagnosis of TIA rests on the history of the attacks and the skill with which the history is taken and interpreted, except in those instances when the physician is present during an attack. Some symptoms, such as “numbness” and “dizziness,” are common and not always indicative of a TIA. The pattern, timing, and circumstances in which the symptoms occur are as important as the symptoms in making the diagnosis.

A bruit over a carotid artery is highly suggestive evidence of carotid stenosis. Emboli in retinal vessels and a difference of >15 mm Hg between the systolic blood pressures of the arms are both indicators of proximal large-vessel disease. However, these findings are often present in the absence of a history of TIAs.

One important and useful concept is the diagnosis of “possible TIA.” Many patients have some symptoms that can be seen in TIAs but insufficient evidence to make the diagnosis. The patient may have additional uncharacteristic symptoms, the symptoms may occur in unusual circumstances, or the description may be too vague to make the diagnosis of TIA. Instead of accepting or discarding the diagnosis prematurely, it is often helpful to use the diagnosis of possible TIA for these patients and seek further evidence by obtaining another history, talking to witnesses of the event, or reassessing the patient after a further attack.

The following symptoms are not characteristic of a TIA:
- Unconsciousness without other signs of posterior (vertebrobasilar) circulation symptoms
- Tonic and/or clonic activity
- Prolonged march of symptoms over several areas of the body
- Scintillating scotoma

The following symptoms are not to be considered as TIAs:
- March of a sensory deficit
- Vertigo alone
- Dizziness (or wooziness) alone
- Dysphagia alone
- Dysarthria alone
- Diplopia alone
- Incontinence of bowel or bladder
- Loss of vision associated with alteration of level of consciousness
- Focal symptoms associated with migraines
- Confusion alone
- Amnesia alone
- Drop attacks alone

The differential diagnosis of TIAs includes classic and “hemiplegic” migraine, seizures, transient global amnesia, Ménière’s syndrome, sensory phenomena associated with hyperventilation, and syncope or near-syncope due to hypotension. Other conditions that may mimic TIAs are hypoglycemia, narcolepsy, cataplexy, and periodic paralysis.

2. Stroke
Stroke is a generic term for a clinical syndrome that includes infarction, hemorrhage, and SAH.
Epidural and subdural hemorrhage are not ordinarily considered as stroke but must be considered in a patient who has had a head injury. Chronic subdural hemorrhage may occur without a history of trauma.
- Temporal profile
  1) Improving
  2) Worsening
Stroke patients can worsen for a variety of reasons, but the most common reason is progressing stroke or stroke-in-evolution. Close to half of all stroke patients show worsening during the first few minutes or hours after the onset of the stroke. In approximately one fourth of all patients, worsening occurs after the patient is hospitalized. The history often provides evidence of recent worsening, while repeated examinations are the best way to discover and document stroke progression.

Worsening can be designated as smooth worsening, when it is gradual; step-like worsening, when the neurologic deficit increases rapidly on one or more occasions with intervening periods of no change; or fluctuating worsening, when at least one episode or period of worsening is preceded by improvement.

3) Stable stroke
This category refers to a patient with stroke who has shown little change in deficit over a period of time, which should be specified (e.g., stroke with stable deficit for 72 hours). This term is preferred to the older term “completed stroke,” which has had two conflicting interpretations in common use. One meaning was intended to indicate that a stroke had stopped worsening, but the term has also been interpreted as indicating maximal impairment such as hemiplegia and hemianesthesia and therefore, presumably the stroke could not worsen. Some strokes last >24 hours but clear up in 1-3 weeks (sometimes referred to as a reversible ischemic neurologic deficit [RIND]).

b. Types of stroke
Determination of stroke type can be crucial to rational treatment and prediction of outcome. With the use of CT and MRI and lumbar puncture, bleeding into and around the brain can be diagnosed and these types of stroke separated from the more common infarction. Subtypes of infarction can be diagnosed or suspected on the basis of the presence of other disease, the presentation and findings on examination, and the findings on brain imaging.

1) Brain hemorrhage
Approximately 10% of all strokes are due to brain hemorrhage. Hypertension, especially uncontrolled hypertension, is the leading condition associated with brain hemorrhage. Other predisposing conditions include ruptured aneurysm; AVM; cavernous angioma; drug abuse with cocaine, amphetamines, or alcohol; blood dyscrasia; anticoagulant therapy; amyloid angiopathy; and brain tumor.

The clinical features of brain hemorrhage vary depending on the location and severity of the bleeding. It is unlikely to be preceded by TIAs. The process is usually acute, frequently with severe headache and a decreased level of consciousness. Usually, the blood pressure is elevated at the time of the initial examination, even if there was no preexisting hypertension. (The clinical state can be graded according to the level of consciousness.)

The most common locations of hypertensive bleeding are the basal ganglia, thalamus, lobe of a hemisphere, cerebellum, or pons. Deep hemispheric hemorrhages typically produce a contralateral hemiparesis and hemisensory deficit with aphasia in those patients with the dominant hemisphere involved. Aphasia can occur in cases of hemorrhage into the thalamus of the dominant hemisphere. The presence of oculomotor findings such as forced downgaze or upgaze palsy, unreactive miosis, and convergence paralysis are characteristic of thalamic hemorrhage and help to differentiate it from putaminal hemorrhage.

Patients with lobar hemorrhage into the cortex or subcortical white matter less frequently have a history of hypertension than those with deep hemorrhage. In elderly persons, amyloid angiopathy is a common cause of lobar hemorrhage. Headache is a common feature. Disturbance of the level of consciousness also occurs less often and is seen later in the clinical course. The neurologic deficits are more variable than in deep hemorrhage and depend upon the location and size of the hematoma.

Cerebellar hemorrhage usually occurs in one of the hemispheres, originating in the region of the dentate nucleus. Disequilibrium, limb ataxia, nausea, and vomiting are
common early features. Patients with cerebellar hemorrhage frequently complain of headache and dizziness. The examination usually demonstrates a combination of signs indicative of cerebellar and pontine dysfunction. Peripheral facial palsy, nystagmus, miosis, decreased corneal reflex, and abducens palsy are the most common brainstem and cranial nerve findings.

Primary hemorrhage into the brainstem usually has devastating effects, but occasionally a small hemorrhage occurs, producing limited dysfunction compatible with functional survival. The neurologic deficit resulting from hemorrhage depends on the level of brainstem involvement, with the pons being the most common site.

Symptoms and signs may not distinguish brain hemorrhage from other stroke types, even though many patients with brain hemorrhage have untreated hypertension, and a large proportion present with obtundation and focal deficit. CT reliably shows intraparenchymal hemorrhage. Since the widespread use of CT, it has become evident that a number of patients with a small brain hemorrhage will present with little headache or obtundation and a deficit indistinguishable from that of infarction. Thus, the only way to reliably diagnose hemorrhage is with the routine use of CT. A small percentage of patients with ischemic infarction will present with severe headache, rapid obtundation, and other clinical features indistinguishable from those of brain hemorrhage. Patients with brain hemorrhage rarely show any improvement in neurologic deficit during the first 24 hours.

2) SAH
The characteristic clinical picture of primary SAH (in which the initial bleeding is into the subarachnoid space) begins with the sudden onset of a severe headache. The suddenness of the onset and the severity of the pain are usually dramatic. The headache commonly reaches a severe intensity in a matter of seconds to a minute and is so severe as to alter the patient’s pattern of activity. Often there is a rapid alteration of level of consciousness (including unconsciousness with recovery in a few minutes). Vomiting at onset is frequent. Patients with SAH may be younger and less likely to have hypertension and other underlying disease before the onset of the stroke than patients with other types of stroke.

<table>
<thead>
<tr>
<th>Grade</th>
<th>Symptoms and signs</th>
</tr>
</thead>
<tbody>
<tr>
<td>0</td>
<td>Unruptured aneurysm—no history or other manifestation of subarachnoid hemorrhage</td>
</tr>
<tr>
<td>1</td>
<td>Asymptomatic or minimal headache and slight nuchal rigidity</td>
</tr>
<tr>
<td>1a</td>
<td>No acute meningeal or brain reaction, but fixed neurologic deficit</td>
</tr>
<tr>
<td>2</td>
<td>Moderate to severe headache, nuchal rigidity, no neurologic deficit other than cranial nerve palsy</td>
</tr>
<tr>
<td>3</td>
<td>Drowsiness, confusion, or mild focal neurologic deficit</td>
</tr>
<tr>
<td>4</td>
<td>Stupor, moderate to severe hemiparesis; may also include early decerebrate rigidity and vegetative disturbances</td>
</tr>
<tr>
<td>5</td>
<td>Deep coma, decerebrate rigidity, moribund appearance</td>
</tr>
</tbody>
</table>

The clinical grade (Table 1) at the time of initial evaluation is a good index of short-term prognosis. There are usually no focal findings on examination, but the likeliest single finding is a partial oculomotor nerve palsy. Most patients have a stiff neck on bending the head forward or other signs of meningeal irritation (Kernig’s or Brudzinski’s signs). Subhyaloid ( preretinal) hemorrhage may be found on funduscopic examination. CT almost always shows blood in the subarachnoid space on the day of the hemorrhage, but there is a diminishing chance of finding blood with each day after the onset. In patients suspected of SAH who have a CT scan that does not show blood, lumbar puncture should be done to confirm the diagnosis. In SAH, the CSF will be bloody, and the supernatant will be xanthochromic within a few hours after the hemorrhage.

SAH is usually due to a ruptured saccular aneurysm. The aneurysm can sometimes be viewed on CT or MRI, but usually an arteriogram demonstrating all the intracranial
vessels is necessary to demonstrate the lesion. Vasospasm and infarction in the arterial distribution of the vasospasm are common causes of disability after the first 48 hours following the onset of SAH.

Other causes of nontraumatic SAH include AVMs and neoplasms. In 10–15% of patients, no source can be demonstrated on complete angiography, and the prognosis is generally more favorable in such patients.

3) Intracranial hemorrhage from an AVM
SAH, intracerebral hemorrhage, or a combination of both may occur from AVMs. It is characteristic that the hemorrhage has less pronounced symptoms and may be less severe than with ruptured aneurysms. There may be a history of seizures and sometimes focal cerebral symptoms and signs. In some patients, a bruit over the head may be present; in others, preretinal hemorrhage or retinal angiomas are present.

4) Brain infarction
Patients with infarction generally have a medical history that includes one or more risk factors for stroke. That is, patients are unlikely to have been completely healthy before the stroke. Although many of the patients have hypertension, diabetes, and heart disease, previous TIAs and strokes are also common. Severe headache and vomiting are unusual at the onset of brain infarction. The deficit generally comes on rapidly and may continue to worsen over hours or days. Patients present with focal neurologic symptoms and signs, for example, hemiparesis and sensory impairment, with carotid-distribution infarctions.

a) Mechanisms of ischemic infarction
(1) Thrombotic
Thrombotic infarction usually occurs when a thrombus is superimposed on an atherosclerotic plaque. In some circumstances thrombotic infarction may be precipitated by an abnormality of blood clotting.

(2) Embolic
Embolic infarction is due to occlusion of an artery by an embolus distal to a point where adequate collateral blood flow is available.

(3) Hemodynamic
Hemodynamically determined infarction most commonly occurs when there is severe stenosis or occlusion of the proximal arterial supply to a portion of the brain and collateral compensatory blood flow is inadequate while global cerebral perfusion is critically decreased (for example, with decreased cardiac output).

b) Clinical categories
Infarction is commonly considered to be atherothrombotic, cardioembolic, or lacunar. Perhaps 30–40% of patients with infarction cannot easily be classified clinically as having one of these types and are best labeled as having infarction of unknown type. Even if the usual differentiating criteria are met, the assumed diagnosis is not certain.

(1) Atherothrombotic
This type of infarction occurs with atherosclerosis involving selected sites in the extracranial and major intracranial arteries. There are two main ways in which atherosclerosis produces infarction. First, the plaque may enlarge to seriously compromise the lumen of a blood vessel, but more often this happens with a superimposed thrombus. When a vessel occludes, a stagnation clot may form above the original occlusion and propagate distally.

The second mechanism by which an atherosclerotic plaque causes infarction is embolism of thrombus or plaque fragments (artery-to-artery embolus). A history of TIAs and cervical bruit is more frequently found in persons with atherothrombotic infarction than in those with other types of stroke. Clinical diagnosis rests on finding evidence of arterial stenosis or occlusion thought to be due to atherosclerosis at one or more sites.

The infarct may be small and indistinguishable from a cardiac embolic infarct.
Cardioembolic

Although many authors have commented on the exceptionally rapid onset of this type of stroke, such is not uniformly the case. The onset begins with a focal deficit and worsening may occur. The basis for the clinical diagnosis is the demonstration of a cardiac-transcardiac source of embolus and no evidence of other causes of stroke. Cardiac conditions that may produce emboli include intermittent or continuous atrial fibrillation or flutter, recent myocardial infarction, congestive heart failure, and mitral or aortic valve disease. When the source is transcardiac by way of a right-to-left cardiac shunt (paradoxical embolus), the source of the clot is usually a peripheral venous thrombus. The diagnosis of cardioembolic infarction may be suggested by evidence of multiple brain or systemic infarcts.

Clinical presentations that are often due to cardioembolism include isolated homonymous hemianopsia and isolated aphasia. By brain imaging, it is sometimes possible to see an infarct with some hemorrhage in it. Most cardioembolic infarcts involve the cortex and are commonly in the distribution of branches of the middle cerebral artery.

Lacunar

Although this is a pathological term, it is commonly used as a clinical category for the small lesions that result from involvement of deep, small, penetrating arteries. These arteries tend to branch at 90° from the main intracerebral arteries and supply the deep white and gray matter of the cerebral hemispheres (e.g., lenticulostriate arteries) and the brainstem. Since the arteries have poor collateral connections, obstruction of blood flow by arterial disease, thrombus, or embolus leads to infarction in the limited distribution of one of these arteries. Over time, the infarct becomes cystic and filled with fluid surrounded by normal tissue, hence the name “lacune” or lake. Clinical diagnosis usually rests on brain imaging or the clinical syndrome indicating the anatomic location. Brain imaging shows a small lesion (<1.5 cm in greatest diameter) in a location compatible with the deficit. The clinical syndrome may include one of the “classic” lacunar presentations, that is, pure motor hemiparesis, pure sensory stroke, ataxic hemiparesis, or the dysarthria clumsy hand syndrome, even in the presence of a normal brain image on repeated occasions. Other possible causes of such a syndrome include a small hemorrhage or a cortical infarct. Prognosis is generally good for recovery of function.

Larger lesions, sometimes called giant lacunes, involve the distribution of multiple penetrating arteries and may be associated with disease of the larger intracranial arteries.

c) Symptoms and signs by site

The clinical picture is determined by the site and size of brain damage (from either infarction or hemorrhage). The usual practice is to think of the brain blood supply as being divided into two major categories: the carotid system and the vertebrobasilar system.

(1) 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. It may result in anything from a TIA to infarction of a major portion of the ipsilateral hemisphere. The mechanism may be hemodynamic if collateral blood flow is inadequate. Carotid occlusion or stenosis may also cause cerebral infarction by artery-to-artery embolism or by propagation of an occluding clot distally into the stem of the middle cerebral artery.
The neurologic picture can range from monoparesis to hemiparesis with or without a homonymous defect in vision, impairment of speech or language, various types of agnosia, and a range from partial to full hemisensory impairments. Although transient monocular blindness is commonly associated with internal carotid artery stenosis or occlusion, permanent monocular blindness is rare with carotid occlusion.

When infarction in the middle cerebral artery territory is preceded by TIAs in the same vascular distributions or when there is a high-pitched bruit over the origin of the internal carotid artery, the stroke is most likely due to internal carotid artery occlusive disease near its origin.

(2) Middle cerebral artery
The middle cerebral artery begins at the bifurcation of the internal carotid artery. Occlusion of the first portion of the middle cerebral artery (MI segment) almost always produces a neurologic deficit. Most occlusions here are due to emboli. Since the occlusion is distal to the circle of Willis, the opportunity for collateral circulation is restricted to anastomotic blood flow from the anterior and posterior cerebral arteries on the surface of the brain. When this fails, occlusion of the MI segment results in a severe deficit including hemiplegia, hemisensory deficit, homonymous hemianopsia, contralateral gaze palsy, and, if the infarct is in the dominant hemisphere, aphasia. However, occlusion of the middle cerebral artery stem may result primarily in a motor deficit due to the so-called giant lacune that occurs when there is adequate collateral circulation on the brain surface. If the occluding clot breaks up and travels distally, branches may be occluded, with resulting partial syndromes depending on the part of the hemisphere involved.

Severe stenosis of the middle cerebral artery can also be associated with infarction of the portions of the hemisphere supplied by this artery or its major branches.

(3) Anterior cerebral artery
The most common syndrome associated with occlusion of the anterior cerebral artery is weakness of the opposite leg, often most prominent distally and sometimes associated with weakness of the proximal muscles of the upper extremity. Sensory involvement of the same area may accompany the weakness. Apraxia, particularly of gait, and cognitive impairment may occur.

(4) Vertebrobasilar system
The vertebrobasilar system supplies blood to the medulla, pons, cerebellum, mesencephalon, thalamus, occipital lobe, and even portions of the temporooccipital and parietooccipital junctions.
(a) Vertebral artery
Severe stenosis or occlusion of the left subclavian artery or the brachiocephalic (innominate) artery may cause a reversal of blood flow in the vertebral artery on that side. This usually does not cause symptoms. When one of the vertebral arteries is atretic or terminates in the posterior inferior cerebellar artery and the other vertebral artery occludes, there may be infarction of the brainstem. Occlusion of a vertebral artery or the posterior inferior cerebellar artery may produce a lateral medullary infarct. This syndrome is characterized by the sudden onset of severe vertigo, nausea, vomiting, dysphagia, ipsilateral cerebellar ataxia, an ipsilateral Horner's syndrome, and decreased pain and temperature discrimination on the ipsilateral side of the face and the contralateral extremities and trunk.
Basilar artery
Occlusion of the basilar artery may result in infarction of the brainstem or sometimes TIAs or, rarely, no symptoms. Usually occlusion or severe stenosis of the basilar artery results in signs implicating bilateral brainstem impairment, whereas syndromes due to stenosis or occlusion of a branch artery involve structures on only one side of the brainstem. In addition to the larger (circumferentially) branches to the cerebellum, the basilar artery also has many short branches that supply the brainstem. Syndromes involving these branches can be localized by knowledge of the detailed anatomy of the brainstem and cerebellum. Branch artery defects may be “crossed,” that is, involve sensory or motor deficits on one side of the face and the opposite side of the body. Dizziness and vertigo may be prominent symptoms, and nystagmus is a frequent finding. Involvement of specific cranial nerves, such as the oculomotor nerve, also helps localize the lesion in the brainstem. Occlusion of the terminal portion of the basilar artery may result in occlusion of the penetrating arteries, resulting in bilateral thalamic and upper midbrain infarction. Like all infarctions, strokes involving the distribution of the basilar artery may evolve over several days.

Posterior cerebral artery
The basilar artery ends in the two posterior cerebral arteries. Occlusion is often due to an embolus and most often causes a homonymous visual field defect, usually hemianoptic or quadrantanoptic. Additional problems, such as dyslexia and dyscalculia, occur due to dominant hemisphere involvement. Involvement of the nondominant hemisphere may produce a parietal lobe syndrome. A hemisensory deficit may occur with thalamic infarction and occasionally leads to a “thalamic” syndrome of unremitting hemibody pain. When both posterior cerebral arteries are occluded, cortical blindness occurs and changes in behavior are often seen.

C. Vascular Dementia
Although there is no question that cognitive impairment occurs with destruction of large amounts of brain from one large infarct or from multiple smaller infarcts, there is controversy over the frequency with which vascular disease is the main cause of dementia. Since the prevalence of dementia, like that of stroke, increases with advancing age, it is not surprising that dementia is frequently associated with infarcts. When there are multiple infarcts, vascular dementia is a likely possibility, but a single small infarct is unlikely to be the cause of dementia. At present, there seems to be little evidence to support the concept that dementia is due to chronic ischemia without brain infarction.

D. Hypertensive Encephalopathy
This syndrome is now relatively rare, but it is mentioned here to distinguish it from a stroke (usually an intracerebral hemorrhage) that is associated with arterial hypertension. This syndrome occurs primarily in patients with chronic, poorly controlled hypertension and only rarely in patients with new-onset hypertension. In either case, blood pressure rises rapidly and the patients complain of headache, become obtunded, and develop seizures and sometimes transient neurologic deficits. On examination, the level of consciousness is relatively more depressed than the severity of neurologic signs. Papilledema is usually present along with flame hemorrhages and retinal exudates. In hypertensive encephalopathy, diastolic blood pressure is usually >130 mm Hg, and there is no gross bleeding into or around the brain on imaging or as detected by lumbar puncture. The CSF pressure is usually increased. These patients generally respond well to decreasing their blood pressure. In other types of stroke, the neurologic signs are often preeminent. Brain imaging and lumbar puncture will often clarify the diagnosis.

II. Pathology (see outline)

Background Material

III. Risk Factors and Prevention
The primary purpose of this section is to identify persons who are likely to have a stroke. This offers the opportunity to treat the predisposing factors that place these individuals at high risk and to prevent stroke from occurring.

A. Characteristics and Lifestyle
This category concerns those risk factors that are characteristics of the individual by birth, genetics, environment, or apparent choice.

1. **Definite**
   - Cigarette smoking and certain patterns of alcohol consumption have only recently been established as risk factors for stroke. In addition to drug abuse, these risk factors are individually controlled, and the risk can be decreased or removed by stopping use.
   - Age, male sex, race, and familial factors are definite risk factors for stroke that at first appear to be unalterable, but they may be markers for other factors that are treatable. For example, the heredofamilial predisposition to stroke appears to be genetic, but in some families this genetic susceptibility is to factors (such as hypertension and heart disease) that are treatable. Mortality rates for stroke are higher for blacks than for whites in the United States, and the rate for the Japanese in Japan are among the highest in the world. For the Japanese, there is evidence that racial variation is affected by the environment. In Japan, the mortality rates for stroke have been very high for most of this century and exceed those for heart disease, but the rates are lower for Japanese persons living in Hawaii and the United States mainland.

2. **Possible**
   - Although not proven to be risk factors for stroke, oral contraceptive use, diets high in animal fats, physical inactivity, obesity, and abnormal blood lipids can be altered in high-risk individuals. Other risk factors that have been considered but not established and are not easily controllable are personality type, geographic location, season, climate, socioeconomic factors, and early maternal mortality. Continuation of studies to establish the degree of risk for these factors are necessary.

**B. Disease or Disease Markers**

1. **Definite**
   - A number of diseases have been established to be major risk factors for stroke. Hypertension is foremost, and control of hypertension decreases the risk of stroke. Cardiac disease, in its many manifestations, is also treatable. Particularly important are myocardial infarction, valvular heart disease and prostheses, congestive heart failure, arrhythmias (particularly atrial fibrillation), left ventricular hypertrophy, ECG abnormalities, and endocarditis. Other treatable diseases or disease markers include TlAs (see IV. Clinical Assessment) and elevated hematocrit. Diabetes mellitus is a definite risk factor for stroke, but there is no evidence that treatment alters the risk. An elevated fibrinogen concentration is also a risk factor. Migraine and migraine equivalents are risk factors for stroke, but their mechanisms are not understood and the value or lack of value of specific treatment in the prevention of stroke is unknown.

2. **Possible**
   - Hyperuricemia and hypothyroidism may be associated with increased risk for stroke.

**C. Asymptomatic Structural Lesions**

1. **Physical Examination**
   - Carotid bruit is an indication of increased risk of stroke, but not necessarily of ipsilateral cerebral infarction in the territory of the artery with the bruit. However, the risk of stroke is even greater in subjects with a bruit and hypertension and those with a bruit, coronary heart disease, and diabetes.

   Retinal emboli, blood pressure differences between the arms, and reduced pressure on oculoplethysmography are manifestations of vascular disease and are associated with an increased risk for stroke.

2. **Imaging**
   - CT and MRI of the brain have resulted in the recognition of vascular abnormalities as well as clinically silent or unrecognized cerebral infarction and hemorrhage. The visualization of clinically silent cerebral infarction and hemorrhage identifies individuals who are at high risk for further events and offers the opportunity to institute risk intervention.

   With AVM, aneurysm, or vascular hamartoma, an individual is at increased risk not only for intracranial hemorrhage but also for cerebral ischemia. Although it has not been determined whether these lesions in asymptomatic individuals should be removed, the continued decrease in surgical morbidity and mortality may soon establish a favorable risk–benefit ratio.
Some patients who have a TIA have a lesion on imaging (see IV. Clinical Assessment). The site of the lesion may be related to the site of the TIA, or it may be in another location. Detection of such a lesion simply indicates the need to search for its cause.

Technology is now available to evaluate the extracranial arteries supplying the brain with minimal or no risk. Atherosclerosis and stenosis of an artery supplying the brain indicates an increased risk for stroke. As stenosis becomes more severe, the stroke rate increases, but it is not conclusively established that there is an increased risk of infarction in the distribution of the artery with increasing disease. Nevertheless, methods of identifying disease in such arteries enable the clinician to follow closely those individuals with increased risk, to monitor changes, and to treat other risk factors.

Routine visualization of extracranial arteries supplying the brain may reveal incidental disease processes such as fibromuscular dysplasia or arterial dissection. Some researchers consider that fibromuscular dysplasia is rarely the cause of clinical vascular disease. Others believe that the association with stroke is causative. For this reason, the value of treatment is unknown. Even though arterial dissection of the cervicocerebral arteries can be associated with fibromuscular dysplasia, dissection often occurs spontaneously or is associated with only trivial trauma to an artery. A dissection may be associated with ischemic symptoms or even severe stroke, but the outcome is usually favorable. There is no consensus concerning therapy for symptomatic dissection, so the value of treatment in preventing stroke is also unknown.

D. Multiple Factors in Combination

Most studies have concentrated on individual risk factors. Some risk factors, however, seem to take on additional significance when associated with others. For this reason, combinations of risk factors that identify groups of people at highest risk can be identified and prevention efforts can be better focused. The Framingham profile,3 consisting of elevated systolic blood pressure, elevated serum cholesterol concentration, glucose intolerance, cigarette smoking, and left ventricular hypertrophy by ECG identifies 10% of the population that will have at least one third of the strokes. In Göteborg, Sweden,4 the combination of increased blood pressure, abdominal obesity, an increased plasma fibrinogen level, and maternal death from stroke were accurate predictors of stroke risk.

IV. Clinical Assessment

This section provides a framework for the description of the evaluation and current status of the patient. Since the patient's status may be changing, it should be stated in relation to the time of assessment and the time of onset of the symptoms. At various times during observation, therefore, the patient's status may differ.

A. History

Usually a history of the onset and possible precipitating events can be obtained from the patient or a relative who observed the beginning of the event. One must probe for answers relevant to different sections of this Classification of Cerebrovascular Diseases III. Features of the circumstances of onset, including activity, how the onset was noted, body position at onset, and the rapidity with which maximal deficit developed are helpful in determining whether a stroke has occurred as well as the type of the stroke. For example, features such as preceding TIAs increase the likelihood of infarction as opposed to hemorrhage as the stroke type. Changes in the level of consciousness at onset, the presence of severe headache or vomiting, and the course of signs after onset are important since they help determine the type of stroke, its location, and the prognosis.

The patient's history or the family history may contain relevant information regarding hypertension, diabetes, cardiac disease, or previous stroke.

B. Physical Examination

1. General

The general physical examination is directed at finding evidence of disease of the cardiovascular system. Attention should be given to heart rate, rhythm, sounds, and size; blood pressure in both arms (with the patient supine and erect); peripheral pulses; and findings suggestive of congestive heart failure. Skin lesions such as ecchymoses and petechiae may suggest processes that produce similar lesions in the brain.
2. Neurologic
The neurologic examination and its interpretation usually locate the site of the lesion. Certain combinations of neurologic signs may establish the location of the disease (for example, the lateral medullary syndrome). Differentiating aphasia from confusion can help establish that the disease is focal rather than diffuse.

3. Vascular
   a. Palpation
      Palpation of the cervical carotid arteries is rarely helpful. Minor differences in the pulse between sides are unlikely to be important, and it is difficult to separate the pulses of the internal and external carotid arteries. Manipulation of the carotid arteries or compression tests may dislodge emboli, temporarily decrease carotid blood flow, or produce a change in cardiac rhythm.

      Palpation of the superficial temporal arteries may be helpful in the diagnosis of cranial arteritis.

   b. Auscultation
      Auscultation of the cervical vessels often provides important evidence concerning the patterns of blood flow. The patient should be sitting or lying. A bell-type stethoscope is most easily applied in the supraclavicular fossa and over the eyes without using pressure, which may produce artifactual noise. First the cardiac sounds should be listened to over the aortic valve, and then the bell should be moved superiorly to distinguish transmitted cardiac sounds from sounds arising in the brachiocephalic (innominate), subclavian, common carotid, or internal carotid arteries. If respiratory sounds obscure auscultation, the patient should be requested to “stop” breathing for a few seconds. Bruits should be graded on a 1–6 scale for loudness (1, barely heard with stethoscope; 6, heard without stethoscope). High-pitched bruits are more likely to be associated with significant carotid stenosis than are low-pitched sounds. When the bruit is heard during both systole and diastole, it is likely to be due to high-grade stenosis of the internal carotid artery. Severe carotid stenosis is often present without a bruit. A soft continuous sound that varies with changes in neck position or is obliterated by jugular compression is most likely a venous hum and is of no significance. A soft, often continuous, cervical bruit is fairly common in normal children.

      Auscultation over the cranial vault and orbits can disclose a bruit, which may indicate arterial stenosis or an arteriovenous shunt. When there is a complaint of rhythmic head noise, particular attention should be directed to auscultation where the patient localizes the sound. Auscultation of the orbit is performed by instructing the patient to close the eyes, placing the bell of the stethoscope over one eye, and having the patient open the eyes to eliminate artifactual muscle sounds. A continuous, machinery-like murmur or bruit over the orbit is most commonly caused by a cavernous arteriovenous shunt.

4. Ophthalmoscopy
Ophthalmoscopy provides an opportunity to directly inspect small blood vessels. The central retinal artery itself is about 200 μm in diameter. This blood vessel is a direct continuation of the internal carotid arterial system. The retina should be inspected for arterial or venous occlusion, emboli (cholesterol, platelet fibrin, calcific, mixed, or foreign body), hemorrhages, cotton-wool patches, venous-statis retinopathy, microaneurysms, changes associated with arterial hypertension, papilledema, and ischemic retinopathy.

The most commonly observed emboli are composed of cholesterol ester crystals. These appear as shiny orange-yellow emboli often situated at the bifurcation of retinal arterioles. The embolus may appear to be wider than the arteriole; one sees the outer dimension of the erythrocyte column rather than the wall of the arteriole. The presence of one or more retinal cholesterol emboli indicates that there is or has been an ulcerated atheromatous plaque in a major proximal artery, usually the carotid artery.

Another important type of embolus in retinal vessels consists of gray-white material, thought to consist of platelets and fibrin. These emboli may be long and may be seen to move through an arteriole but are commonly stationary. Blood does not appear to flow past these emboli, and they may be associated infarction of the retina. Often these emboli arise from atheromatous lesions of the internal carotid artery.
V. Evaluation

A. Laboratory

1. Laboratory Studies Usually Ordered
   a. Urinalysis: Especially testing for sugar and protein contents, specific gravity, and sediment microscopy. These are crude but valuable screening tests for abnormal glucose metabolism and abnormal renal function.
   b. Blood tests
      1) Serologic test for syphilis: Especially to consider meningovascular syphilis.
      2) Complete blood count with differential, hemoglobin, hematocrit, and platelet count: These tests aid in the recognition of hemoconcentration, polycythemia, inflammatory disease of the vessels, and thrombocytosis or thrombocytopenia.
      3) Erythrocyte sedimentation rate: This is helpful in the detection of vasculitis, especially cranial arteritis, as well as other types of collagen-vascular disease.
      4) Fasting or random blood glucose concentration: This is important in the recognition and assessment of diabetes, hyperglycemia from another cause, and hypoglycemia.
      5) Serum creatinine and blood urea nitrogen concentrations: If the results of these tests as well as the results of urinalysis are normal, significant renal disease is generally not present.
      6) Serum cholesterol and triglyceride concentrations: Elevated serum cholesterol is a risk factor for stroke in persons under age 50 years and is also a prominent, treatable risk factor for coronary artery disease. If the total cholesterol concentration is elevated, further fractionation into high and low density lipoprotein cholesterol is necessary. Classification of the hyperlipoproteinemia (types I–V) by electrophoresis is often desirable. Apolipoprotein fractionation is assuming increasing clinical importance.
      7) Serum electrolytes (i.e., Na⁺, K⁺, Cl⁻, and CO₂) concentrations: Abnormal concentrations of serum electrolytes may be seen as the result of diuretic therapy or as the result of inappropriate secretion of antidiuretic hormone or atrial naturetic factor, which can be secondary to brain insult.
      8) Prothrombin time and activated partial thromboplastin time.

2. Laboratory Studies Ordered Selectively
   a. Urine: Occasionally required are 24-hour collections for metanephrines and vanillylmandelic acid concentrations (to rule out pheochromocytoma), porphobilinogen and lead concentrations, urine osmolality, and screens for drug abuse.
   b. Blood
      1) Serum protein concentration: Dysproteinemias, such as multiple myeloma and macroglobulinemia, can be detected by serum protein electrophoresis.
      2) Coagulation studies: In addition to the routine screening tests, other factors such as the lupus anticoagulant and decreased antithrombin III activity may be involved in excessive clotting. The following additional tests may be useful: platelet adhesion and aggregation; plasma fibrinogen concentration; factors VII, VIII, IX, and X concentrations; euglobulin lysis time; clotting time; and platelet factor 3 activity. Occasionally during investigative studies, thromboxane A₂ and prostaglandin I₂ levels will be determined, as well as numerous other coagulation tests that are available.
      3) Other blood studies: Some of the following blood tests may be necessary in special circumstances: blood gases (Po₂, Pco₂, pH, bicarbonate), liver function studies, thyroid function studies, Ca²⁺ and catecholamine concentrations, hemoglobin electrophoresis (looking especially for S and C hemoglobins), special serologic studies for collagen-vascular diseases, and tests for human immunodeficiency virus infection.
   c. CSF
      CT or MRI are usually done prior to lumbar puncture in patients with stroke. Cerebral infarction can usually be distinguished from hemorrhage with CT or MRI. An important indication for lumbar puncture is the suspicion of SAH when brain imaging fails to show subarachnoid blood. Lumbar puncture may be useful when there is a suspicion of meningitis, endocarditis, vasculitis, sickle cell anemia, a hematologic/oncologic problem, or a fever of unknown origin. Lumbar puncture should be done whenever the clinician has a problem in establishing the diagnosis. However, lumbar puncture is generally to be avoided if there are signs of increased intracranial pressure.
      1) Pressure: If CSF pressure is elevated, the smallest amount of fluid possible is withdrawn for required studies.
2) Color: Normally the CSF is crystal-clear and colorless. If there is gross blood present and if the intensity of the color decreases after a few drops of CSF appear, the bleeding is due to trauma to a blood vessel during the procedure. If the CSF 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. If the CSF appears to be clear, a trace of xanthochromia may be quickly detected by putting ≤1 ml of CSF in a small test tube and comparing it 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 a high protein content or icterus, but it is commonly due to bleeding into the CSF.

3) Cell count and differential: The CSF cell count is valuable in the differential diagnosis of bleeding, infection, and neoplastic process. Occasionally the cell count is modestly elevated in patients with cerebral infarction.

4) Protein: Modest elevation of CSF protein content is common in patients with various categories of cerebral vascular disease.

5) Glucose: Except with massive SAH, the CSF glucose content is seldom altered in patients with cerebrovascular disease. Central nervous system infection associated with a stroke may lower the glucose content, and under this circumstance it is important to have a simultaneous blood glucose determination for comparison. The concentration of glucose in the CSF is usually about two thirds that in the blood.

6) Test for syphilis: Occasionally central nervous system syphilis, usually meningovascular, will be detected.

7) Bacteriologic and serologic studies: Suspicion of infection is an indication for fungal, bacterial, and occasionally viral cultures, in addition to microscopic examination. Moreover, India ink preparation, cryptococcus antigen determination, and other special serologic tests may be indicated.

B. Neurophysiologic

1. EEG

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

EEG is most likely to be helpful in evaluating cases in which there is concern that there may be an underlying epileptogenic process associated with a focal cerebral lesion. During carotid arterial or cardiac surgery, the EEG is of value in monitoring brain function. In certain instances, the EEG may be of aid in evaluating the stages of "irreversible coma." The various criteria for "brain death" have been outlined by the Ad Hoc Committee of the American Electroencephalographic Society.

C. Cardiovascular

1. Cardiovascular Studies Usually Ordered

a. Standard ECG: ECG should be performed to detect recent or old myocardial infarction, cardiac arrhythmia, or left ventricular hypertrophy.

b. Chest roentgenography: Standard radiographs of the chest give valuable information concerning heart size and configuration, evidence of congestive heart failure, and the presence of aortic or pulmonary pathology, including infection, neoplasia, and infarction.

2. Cardiovascular Studies Ordered Selectively

a. Long-term ECG monitoring: Long-term monitoring of heart rate and rhythm can detect intermittent arrhythmias.

b. Echo: Two-dimensional echo with or without M-mode is valuable in the diagnosis of mitral valve prolapse, atrial myxomas, infective endocarditis, nonbacterial thrombotic endocarditis, and mural thrombosis. Mural thrombi may be seen in patients with mitral stenosis, prior myocardial infarction, idiopathic myocardiopathy, cardiac transplants, and artificial hearts or prosthetic heart valves. Doppler echo is usually the initial technique used to evaluate valvular dysfunction. Contrast echo is valuable in the diagnosis of right-to-left cardiac shunts. Echo is the best way to diagnose left ventricular hypertrophy, asymmetric septal hypertrophy, diffuse cardiomyopathy, mitral valvular calcification,
ventricular aneurysm, and ventricular dyskinesis. A normal cardiac echo does not exclude a cardiac source of emboli.

Transesophageal two-dimensional echo is useful to detect thrombi in the atrium or left atrial appendage, to examine the atrial septum, to detect disease of the mitral valve, and to screen for aortic dissection.

c. Other
   1) Nuclear cardiology
      a) Myocardial infarct-avid scanning (using technetium-99m pyrophosphate)
      b) Myocardial perfusion imaging (using thallium-201)
      c) Radionuclide angiocardiography (using technetium-99m pertechnetate)
   2) Invasive investigations
      a) Cardiac catheterization and angiography
      b) Intracardiac electrophysiology
      c) Swan-Ganz catheterization
   3) Long-term blood pressure monitoring

D. Brain Imaging
   1. CT
      CT utilizes an x-ray beam, the absorption of a portion of which, when analyzed by a computer, enables construction of a map (image) of the relative densities of brain tissue. CT examination can be carried out before and/or after the administration of an intravenous radiographic iodinated contrast material. Gray matter, white matter, and CSF within the subarachnoid space or ventricles can be differentiated on a plain CT scan, while the lumen of blood vessels (arteries and veins) can be visualized after the injection of contrast material. Disturbances in the blood-brain barrier that permit leakage of contrast material into abnormal tissue is detected on the postcontrast CT scan as an increase in the density of those tissues (contrast enhancement). The decision whether to inject contrast material depends on the clinical indication and the likelihood that the enhanced CT scan will provide additional information without significant additional risk to the patient.

      At present, CT has scan times on the order of seconds per slice, so that patient motion is usually not a problem. A complete examination of the brain is possible within 10–20 minutes. High-spatial-resolution images of the brain, with a pixel size of 0.45×0.45 mm, are possible with a slice thickness of only 1.5 mm. Thin slices are used for special circumstances, such as in evaluating the brainstem, while in the routine situation slices of 5–10 mm are used. With thicker sections (5–10 mm), artifacts in the posterior fossa may obscure the brainstem and portions of the cerebellum, rendering CT suboptimal for evaluation of these areas. Thinner sections (1.5–3 mm) through the brainstem produce less prominent artifacts. Reconstruction algorithms enable the stacking of multiple thin axial sections so that coronal, sagittal, and paraxial images may be obtained. Usually, such reconstructed images are less sharp than the original images.

      Acute nonhemorrhagic infarcts within the first 24 hours following ictus are often not seen on CT, but they may be seen as an area of subtle hypodensity with or without mass effect. Hypodensity becomes more apparent on CT scans as edema increases over the first 24–72 hours. Hypodensity as a finding is usually apparent, if it is to become so, by 8 days after ictus. The mass effect increases over the first 24–72 hours, being most evident 3–5 days after ictus. The subsequent course is one of involution of the mass effect. Organization of necrotic tissue contributes to the hypodensity seen later, months to years after ictus. Small infarcts may not be identifiable on CT scans. Subacute cerebral infarction, from several days to several weeks after ictus, usually shows as areas of enhancement following contrast injection, due to either a leakage of the contrast material through the disrupted blood-brain barrier (at the site of new vessel proliferation) or a loss of autoregulation (luxury perfusion).

      Flowing blood is of slightly higher density than the brain parenchyma, and clotted blood (following serum extrusion) is of still higher density. Thus, the lumen of a large aneurysm may be seen as an area of slightly higher density. Aneurysms and AVM are identified after contrast injection as enhancing round or tubular structures. Blood clots from bleeding within the brain parenchyma can be seen immediately, and the areas increase in density over the
ensuing several hours. Clots can be seen for as long as several weeks after the hemorrhage. After resolution of a hematoma, the region is hypodense. Blood within the subarachnoid space is identifiable as an area of localized or diffuse increased density in the cisterns and sulci. Intraventricular hemorrhage can be seen as areas of increased density within the ventricles, sometimes associated with a CSF–blood fluid level; the blood (of higher gravitational density) is more dependent. The location of hematomas within the brain parenchyma may suggest their etiology. Hematomas within the basal ganglia, brainstem, or cerebellum are most commonly associated with hypertension. Hematomas adjacent to the circle of Willis most often result from rupture of a saccular aneurysm. Hematomas in the brain parenchyma suggest trauma, AVM, bleeding diathesis, hemorhagic infarction, congestrophic angiopathy, and neoplasm. The CT appearance of hemorrhagic infarcts is not typical. The most common appearance is that of an area of abnormal mixed density (low, normal, and slightly high) in a typical vascular territory. In a number of instances of hemorrhagic infarction, on CT the infarct appears hypodense, not hyperdense. This is because the degree of hemorrhage is not of sufficient density relative to the nonhemorrhagic (adjacent) brain tissue included in the same slice thickness to give a high-density image (volume averaging). The hemorrhagic infarct that is grossly hemorrhagic with clot formation may give a CT appearance not distinguishable from that of a hematoma.

CT readily distinguishes many lesions that can clinically stimulate the presentation or course of cerebral infarction. Subdural hematomas, whether subacute or chronic, are recognized by their location, the density of their blood products, and their displacement of the brain parenchyma. Neoplasms within the brain parenchyma (gliomas and metastases) and those external to the brain (meningiomas) are shown by their mass effect, density, and enhancement.

2. MRI
Images of the brain can be generated based upon the hydrogen proton—its concentration and biologic state (relaxation properties). Differences between gray and white matter, CSF, and bone give rise to image contrast depending upon the software program (pulse sequence) used to stimulate the proton to a higher energy level and the way in which the energy loss is observed. Proton MRI systems operate in a range from low field strengths, (0.3 T, resistive, permanent, superconducting magnets), up through middle field strengths (0.5–0.6 T, superconducting), on up to high field strengths (1.5–2.0 T, superconducting). In resistive systems the magnetic field is created and maintained by the input of electrical energy, while in the superconducting systems helium and liquid nitrogen coolants maintain the magnetic field once it is established. The higher-field MRI systems are generally more expensive to operate and require more expensive installation in the form of shielding to contain the magnetic field but have as the benefit a higher signal-to-noise ratio, which translates into images of higher spatial resolution thinner sections, and the potential for in vivo spectroscopy of phosphorous metabolites and other nuclei.

In general, MRI requires longer data acquisition than does CT because each MRI sequence takes 2–15 minutes. Patient motion during a portion of the examination often results in enough image degradation to make the study uninterpretable. Medically unstable, uncooperative patients present management problems for MRI studies. Control of motion through sedation or general anesthesia is possible. Intubation and ventilation of patients within the imaging environment requires skill and the use of special nonferromagnetic equipment. Monitoring of patients in the imaging environment is also difficult because the magnetic field may distort or render useless routine monitoring devices and their output displays. Nonferrous ventilators, intubation devices, and monitoring equipment exist. Their employment requires skill, as the accidental introduction of ferromagnetic items ranging from pens to i.v. poles constitutes serious hazards as potential projectiles when pulled into the magnet. The force of such a projectile may injure the patient, technician, nurse, or physician.

At present, contraindications for MRI are severe claustrophobia, inability to control the patient’s movements, and the presence of ferromagnetic foreign bodies, such as those in the globe of the eye or ferromagnetic aneurysm clips or a caval umbrella. With widespread use of prosthetic devices, it should be determined whether the device is ferromagnetic before MRI is done. An additional important contraindication to MRI is the presence of a cardiac pacemaker, which can malfunction in the magnetic field.
Radiofrequency (RF) coils surrounding the patient’s head are used to stimulate protons to a higher energy level. Following a short RF pulse, the decay of the proton back to its resting state is observed as a signal detected by the surrounding receiver coils. The detected signals are used to reconstruct an image of the brain in the axial, sagittal, or coronal planes. Images in a paraxial oblique plane are also possible. Slices varying from 1 mm to 1 cm thick can be obtained with a spatial resolution comparable to that of CT.

A variety of RF pulse sequences are currently available and give somewhat different information. Images with a short time to repetition (TR) (600 msec) and a short time to echo (TE) (20 msec) give images with predominantly T1 relaxation information (T1-weighted image) (T1WI). Such images show morphology of brain tissue relative to CSF spaces and osseous structures. Gray and white matter appear as different shades of gray and the CSF appears black, as does bone. Bone is black because it contains few protons and they are nonmobile. Fat-containing bone marrow in the diploic space is of higher signal intensity and appears white. Blood vessels most often have a hypointense (dark) lumen because the energized protons flow out of the imaging plane before a signal can be returned (flow voids). For a variety of physical reasons, slow-flowing blood may appear as a hyperintense signal (light area) depending on the location of the slice and the RF pulse sequence. Under these circumstances, the interpreter of the image must be aware of the possibility of an increased signal intensity artifact within the lumen of the blood vessel so as not to misinterpret the hyperintense signal as thrombosis. Nonhemorrhagic infarcts appear as areas of slight hypointensity (dark) and mass effect on T1WI. Hours after bleeding, acute hematomas consist of intracellular deoxyhemoglobin and appear as light gray masses of slight hypo/intensity on T1WI. With oxidation of the clot to intracellular methemoglobin 3–5 days after the hemorrhage, high-signal-intensity (white) intracellular methemoglobin is seen on T1WI.

An RF pulse sequence using the spin-echo technique with a long TR (2,000–3,000 msec) and a short TE (20–30 msec) gives an image predominantly of proton density with some T1 and T2 information. A long TR (2,000–3,000 msec) and a long TE (70–120 msec) gives a T2 weighted image (T2WI). Proton density increases within brain tissue when the extracellular water content is increased at the site of nonhemorrhagic infarction, vasogenic edema, or demyelination or within a tumor. Such sites appear as areas of high signal intensity (bright or white) on a proton density image. This high signal intensity is greater than that of the CSF. On T2WI, the area of abnormal water content within the brain parenchyma is also of high signal intensity (appears light), as do the CSF in the ventricles and sulci. Thus, to recognize an area of infarction that is adjacent to the ventricle or within the cortex next to the subarachnoid space, the proton density image is more important because on T2WI the signal intensity of the infarct may not be separable from that of the CSF. Acute nonhemorrhagic infarction is shown on the proton density image as a mass effect and a (bright) area of hyperintensity. Acute intracerebral hematoma in the deoxyhemoglobin state is shown on T2WI as a mass of marked hypointensity (blackness). This is due to a heterogeneous susceptibility effect. The magnetic field within the erythrocytes of the hematoma differs from that of the fluid in the interstitial space in the adjacent brain parenchyma so that nearby protons are thrown out of phase by the different fields, producing an area of blackness. Intracellular methemoglobin continues to show susceptibility effects on T2WI. Once methemoglobin is extracellular, a solution of methemoglobin and interstitial fluid is formed, erasing the susceptibility effect. Extracellular methemoglobin shows a prolonged T2 relaxation, appearing as a (light) area of high signal intensity. Extracellular methemoglobin is seen approximately 1 week after onset and is noted first peripherally; with time extracellular methemoglobin is found more centrally within the clot as oxidation and cell membrane lysis occur. Ingestion of the blood clot leads to the presence of hemosiderin within macrophages. Hemosiderin within the lysosomes of macrophages produces a susceptibility effect, leading to a hypointense (black) rim around the margins of the resorbing hematoma on T2WI. Such hemosiderin staining of the brain tissue is commonly present for the rest of the life of the patient, thus identifying on MRI the site of a resorbed hematoma.

Rapidly moving blood produces a signal void on T2WI. Thus, the lumen of aneurysms and dilated vessels within AVMs can be detected on MRI without the injection of intravascular contrast agents. The presence of blood products in association with such vascular lesions indicates the site of previous bleeding. Even in the acute situation, MRI may be useful (if
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patient motion can be controlled) by showing the etiology of a clot when an aneurysm or AVM is responsible. The recent development of an MR RF pulse sequence that utilizes a flip angle of <90° (fast scan) enables shorter scan times and can demonstrate, as bright areas of high signal intensity, the lumen of blood vessels (angiographic effect). Cryptic vascular malformations or occult vascular malformations include cavernous angiomas, telangiectasias, and venous malformations. Except for the latter, these lesions are often not identifiable by conventional arteriography; some are identifiable by CT. Many more are shown by MRI as either a hypointense flow void (such as occurs in a venous malformation) or as an area of slow blood flow, blood clot, and old bleeding with hemosiderin deposition.

SAH, which is accurately demonstrated by CT as an area of high density, is not reliably shown by MRI. High partial oxygen tensions within the CSF, movement of the CSF due to arterial pulsations, and factors as yet unknown may explain this failure to see SAH on MRI.

MRI is an effective way of demonstrating other lesions that may mimic the clinical presentation or course of cerebrovascular lesions. Extracerebral hematomas and intraparenchymal brain tumors are usually well shown and differentiated.

At present, MRI is the best imaging method to differentiate nonhemorrhagic from hemorrhagic infarcts. This is based on the ability of MRI to show deoxyhemoglobin as an area of hypointensity in acute hemorrhage on proton density and T2WI and methemoglobin as an area of high signal intensity in subacute hemorrhage on T1WI. Many infarcts indicated by CT to be nonhemorrhagic are found to have hemorrhagic components when examined by MRI. The significance of these findings in the clinical management of such patients is at present unknown.

MRI is also the most sensitive method for demonstrating venous sinus thrombosis, one cause of hemorrhagic infarction. By utilizing T1- and T2-weighted RF pulse sequences and by compensating for blood flow artifacts, clot can be identified within the dural venous sinuses or cortical veins without entailing the risks of angiography. MRI has been commonly successful in demonstrating clot in the wall of the carotid artery as a result of intramural dissection. Subacutely, methemoglobin can be shown as high-intensity clot in the wall, narrowing the lumen. With occlusion of the internal carotid artery in the neck, the flow void within the ipsilateral intracavernous carotid artery disappears on MRI compared with the contralateral (normal) side. Thus far, MRI has not been successful in the demonstration of atherosclerotic plaque narrowing of the internal and common carotid arteries. MR angiography has been most successful in patients with normal blood flow patterns. At present, this is an unresolved technical problem.

Gadolinium diethylenetriaminepentaacetic acid (DTPA) is a contrast agent developed for MRI. As such, it crosses the damaged blood-brain barrier and is transiently deposited in abnormal tissues. On T1WI, gadolinium, which has seven free electrons, shortens T1 by affecting the water protons in its immediate vicinity. This produces an area of high signal intensity (white) on T1WI. The MRI scanner is more sensitive to the detection of gadolinium enhancement than is the CT scanner to contrast medium enhancement; thus, gadolinium DTPA–enhanced MRI may prove valuable in the demonstration of small subacute areas of infarction. Other advantages of gadolinium DTPA relative to CT contrast material are less osmotic load, less fluid volume, and less risk of sensitivity reactions. These are important in patients with congestive heart failure, renal insufficiency, and sensitivity to x-ray contrast dyes.

3. Other

CT has replaced plain skull radiographs, echo, and isotope brain scanning for the evaluation of typical cerebrovascular disease. Plain skull radiographs give significant positive diagnostic information in relatively few patients with clinically typical cerebrovascular disease/cerebral infarction and are rarely used for this purpose. Calcification of the carotid artery in the region of the sellae is very common but does not give statistically significant evidence concerning the presence or absence of occlusive disease of the carotid artery where the calcification is noted. Occasionally, calcification may be found in the wall of a suprasellar aneurysm or as part of an AVM.
E. Vascular

1. Noninvasive

A variety of technologies and methods are now available to evaluate the disease status of the extracranial vascular system. These testing methods can be classified as indirect or direct on the basis of the principles used. In indirect testing, instrumentation has been developed that detects abnormalities of blood flow, pressure, or pulse wave arrival time in the periorbital and retinal circulations secondary to the presence of severe disease in the ipsilateral internal carotid artery. In direct testing, the instrumentation detects alterations in wall morphology or blood flow patterns in the region of the disease itself at the carotid bifurcation, in the intracranial vessels, or in the subclavian or vertebral arteries.

a. Indirect tests

1) Periorbital circulation

a) Directional continuous-wave Doppler sonography

Among the tests that assess changes in the periorbital circulation as a means of detecting disease in the cervical portion of the internal carotid artery, directional continuous-wave Doppler sonography is the test most frequently used clinically. In normal supraorbital and frontal arteries, blood flows from within the skull to the surface of the scalp. Direction-sensing continuous-wave Doppler sonography identifies both the amplitude of the pulsations and the direction of the flow. In the presence of severe disease of the ipsilateral internal carotid artery, the direction of blood flow is reversed. The sensitivity of the test can be increased by using a variety of compression maneuvers to determine whether there is a compensatory increase in blood flow from collateral vessels. The test is relatively simple to perform but requires considerable skill if good results are to be obtained. It is sensitive only to severe stenosis of the internal carotid artery and cannot differentiate severe stenosis from occlusion. In addition, the test is difficult to interpret in patients with bilateral severe disease who frequently have false-negative results.

b) Photoplethysmography

This technique utilizes an infrared photosensor placed over the area of skin supplied by the frontal and supraorbital arteries, which is used to detect the volume of pulsation in these vessels. In addition to evaluating changes at rest, compression maneuvers of the facial, temporal, and carotid arteries can be used to increase the sensitivity of the test. This technique is relatively simple, but its accuracy is not comparable with those of more sophisticated tests, and it is now rarely used.

2) Orbital circulation

a) Quantitative oculopneumoplethysmography

This technique is a modification of the suction ophthalmodynamometry technique and uses scleral suction cups to raise intraocular pressure and interrupt the circulation. During removal of the vacuum, return pulsations are detected by a sensor in the cup. Absolute ophthalmic systolic blood pressure is measured, as well as the amplitude of the pulsations. Severe stenosis is identified by detecting abnormal values for the ratio of the ophthalmic and brachial systolic blood pressures, a difference between the systolic blood pressures of the eyes, or a difference between the amplitudes of ocular pulsations. This is the most widely used of the oculoplethysmographic tests and is the most accurate. It does, however, suffer from deficiencies in distinguishing occlusion from severe stenosis and is incapable of identifying stenoses of <60% diameter reduction. Bilateral severe disease may also be difficult to identify with this technique. Intrinsic eye pathology may preclude the use of this test.

b) Pulse-delay oculoplethysmography

This technique measures arrival of a pulse in the ophthalmic vessels while simultaneously recording the same parameter in the earlobe. Severe internal carotid artery stenosis is detected by a delay in arrival of the pulse wave between the eye and ipsilateral ear or a delay in arrival of the pulse wave between eyes. External carotid artery stenosis is conversely documented by a delay in arrival of the ear pulse compared with the ocular pulse. The results of this test are displayed in analog format in fluid-filled instruments or in digital format in...
air-filled instruments. As with all indirect tests, it is difficult to distinguish severe stenosis from occlusion and to accurately detect severe bilateral disease.

c) Ophthalmodynamometry
This test was the forerunner of the more commonly used oculoplethysmography described above. Ophthalmodynamometry is designed to detect stenosis of the internal carotid artery by measuring blood pressure in the ophthalmic artery. Pressure is applied to the globe using either compression or a suction apparatus, and both diastolic and systolic blood pressures can be obtained. The suction type of ophthalmodynamometry is used more widely than the compression type. Although it has a high positive predictive value, its low negative predictive value precludes the use of ophthalmodynamometry as a routine screening test.

b. Direct tests
1) Cervical carotid artery
a) B-mode ultrasonography
High-frequency sound waves are transmitted through tissues, and returning echoes are processed to display an anatomic image of the area evaluated. The arterial wall produces brighter echoes than the surrounding soft tissue and enables identification of the vessel. The lumen of the vessel is relatively free of echoes, thus providing contrast between the vessel wall and the lumen. Relatively mild disease of the internal carotid artery is acoustically homogeneous and can be readily detected with this test. Relatively small surface lesions, such as ulcers, may also be detected. With more severe disease, the acoustic properties of the atherosclerotic plaque are more complex and result in difficulties in interpreting the image. Calcification of the vessel wall is a major problem due to the acoustic shadowing produced in the areas below it. In addition, differences in the acoustic properties of flowing blood and thrombus are minimal, resulting in difficulties in distinguishing patent from occluded arteries.

b) Duplex ultrasonography
This test combines B-mode imaging with Doppler technology to produce an image with the capability of detecting blood flow within a vessel. This decreases the difficulty of differentiating occluded from patent arteries and provides a method for grading the severity of disease based on changes in the blood flow pattern in the region of stenoses detected by the Doppler component. The types of Doppler sonography used are either continuous-wave systems, which give an average of the cross-sectional velocity in the area examined, or pulsed ones, which have a finite sample volume, enabling more discrete examination of the blood flow pattern in the area of disease. This technique relies on interpretation of the image to identify minimal disease and interpretation of the Doppler data to detect severe disease.

c) Color duplex sonography
Newer instruments use multiple-gated pulsed Doppler sonography and color coding systems to provide a real-time color image of blood flow in vessels, which is superimposed on the simultaneously generated B-mode image. The color display is coded so that areas of major blood flow disturbance are readily identified. The clinical role of these instruments awaits further study.

d) Sequential ultrasonic arteriography
This technique uses either pulsed or continuous-wave Doppler sonography to build an image of the vessels, with disease being detected by defects in the image itself and by spectral analysis (as in duplex ultrasonography). Sequential ultrasonic arteriography is accurate for the identification of severe disease, but it is not as sensitive for less severe disease. Instrumentation is less expensive than that for duplex ultrasonography instruments, but the technique is more difficult to learn and the examination time is long.

e) Continuous-wave Doppler sonography
This is one of the earlier techniques used in the diagnosis of carotid artery disease. A continuous-wave Doppler probe is traversed along the line of the common and internal carotid arteries, with a constant angle being maintained. A shift in the Doppler frequency indicates the presence of severe stenosis. Interpretation is by audible means and by means of an analog-display strip chart recorder or spectral analysis. In practiced hands, this technique is useful for the identification of severe disease. Deviations from the normal anatomy of the...
TABLE 2. Comparison of Noninvasive Vascular Testing Modalities

<table>
<thead>
<tr>
<th>Modality</th>
<th>Operator dependence</th>
<th>Utilization</th>
<th>Accuracy</th>
<th>Instrument cost</th>
<th>Test cost</th>
</tr>
</thead>
<tbody>
<tr>
<td>Periorbital Doppler sonography</td>
<td>+ + + +</td>
<td>+</td>
<td>+ +</td>
<td>+</td>
<td>+</td>
</tr>
<tr>
<td>Periorbital photoplethysmography</td>
<td>+ +</td>
<td>+</td>
<td>+ +</td>
<td>+</td>
<td>+</td>
</tr>
<tr>
<td>Quantitative oculopneumoplethysmography</td>
<td>+ + +</td>
<td>+</td>
<td>+ +</td>
<td>+</td>
<td>+</td>
</tr>
<tr>
<td>Pulse-delay oculopneumoplethysmography</td>
<td>+ +</td>
<td>+</td>
<td>+ +</td>
<td>+</td>
<td>+</td>
</tr>
<tr>
<td>B-mode ultrasonography</td>
<td>+ + + +</td>
<td>+</td>
<td>+ +</td>
<td>+</td>
<td>+ + +</td>
</tr>
<tr>
<td>Duplex ultrasonography</td>
<td>+ + +</td>
<td>+</td>
<td>+ +</td>
<td>+</td>
<td>+ + +</td>
</tr>
<tr>
<td>Color duplex ultrasonography</td>
<td>+ + +</td>
<td>+</td>
<td>+ +</td>
<td>+</td>
<td>+ + +</td>
</tr>
<tr>
<td>Sequential ultrasonic arteriography</td>
<td>+ + + +</td>
<td>+</td>
<td>+ +</td>
<td>+</td>
<td>+ + +</td>
</tr>
<tr>
<td>Continuous-wave Doppler sonography</td>
<td>+ + + +</td>
<td>+</td>
<td>+ +</td>
<td>+</td>
<td>+ + +</td>
</tr>
<tr>
<td>Quantitative phonangiography</td>
<td>+ + + +</td>
<td>+</td>
<td>+ +</td>
<td>+</td>
<td>+ + +</td>
</tr>
</tbody>
</table>

+++ high; +, low.

carotid bifurcation result in misinterpretation, and the test is now infrequently used as a primary diagnostic approach.

f) Quantitative phonangiography

In this technique, the frequency content of bruits is analyzed and related to the residual lumen diameter of the internal carotid artery. This technique is accurate for identifying residual lumen diameter in patients who have bruits, but it suffers from the fact that approximately 50% of patients with severe disease do not have a bruit. Quantitative phonangiography does not provide any information about the features of the atherosclerotic plaque itself.

2) Intracranial vessels

a) Transcranial Doppler sonography

The principles used in continuous-wave Doppler sonography of the carotid bifurcation have recently been extended to enable examination of the basal cerebral arteries. A low-frequency pulsed Doppler sound wave is used to insonate these vessels through the temporal bone, the orbit, and the foramen magnum. With this approach, blood flow in all vessels of the circle of Willis may be identified and the direction and character of blood flow determined. The major application of this technique is the detection of cerebral vasospasm and the evaluation of the intracranial collateral pathways, although other useful applications are likely to be determined with further research.

c. Utilization characteristics

Noninvasive testing, particularly those employing ultrasound, have become an indispensable part of the diagnostic approach to cerebrovascular disease. Appropriate utilization of these tests requires that those responsible for performing and interpreting the tests be well versed in the capabilities and limitations of the instrumentation used. The accuracy of testing techniques should be frequently monitored and maintained at acceptable standards. Technicians, ultrasonographers, and physicians should participate in ongoing continuing education programs. Table 2 compares the various testing modalities.

2. Invasive—Angiography

a. Intra-arterial

1) Conventional

The intravascular injection of iodinated contrast material for imaging of the carotid arteries, vertebral arteries, aortic arch, and intracranial vasculature is most often done by selective catheterization. Such studies are most often done using the Seldinger technique, which involves puncture of the common femoral artery below the inguinal ligament by a needle, insertion of a guide wire through the needle, removal of the needle, and insertion of a plastic catheter over the guide wire. Using fluoroscopic guidance, the catheter is subsequently placed selectively at the origin of the vessels to be studied so the contrast material can be injected and visualized. The recording method may utilize films in which x-rays strike intensifying screens that emit light onto the film, exposing it, following which the film is developed. This gives a permanent hard copy of the findings in a timed sequence from the arterial through the capillary to the venous phases. Resolution is sufficient to allow subdivisions of the branches of the middle cerebral artery to be identified and their occlusions seen.
TABLE 3. Comparison of Angiographic Techniques

<table>
<thead>
<tr>
<th></th>
<th>Conventional angiography</th>
<th>Intra-arterial</th>
<th>Intravenous</th>
</tr>
</thead>
<tbody>
<tr>
<td>Hospitalization</td>
<td>Yes</td>
<td>No</td>
<td>No</td>
</tr>
<tr>
<td>Time</td>
<td>Long</td>
<td>Intermediate</td>
<td>Short</td>
</tr>
<tr>
<td>Discomfort</td>
<td>Great</td>
<td>Little</td>
<td>Little</td>
</tr>
<tr>
<td>Risk</td>
<td>Thrombosis, embolization</td>
<td>Thrombosis, embolization</td>
<td>Renal failure, congestive heart failure</td>
</tr>
<tr>
<td>Contrast amount</td>
<td>Moderate</td>
<td>Low</td>
<td>High</td>
</tr>
<tr>
<td>Expense</td>
<td>High</td>
<td>Moderate</td>
<td>Moderate</td>
</tr>
<tr>
<td>Small-vessel detail</td>
<td>Excellent</td>
<td>Good</td>
<td>Poor</td>
</tr>
<tr>
<td>Large-vessel detail</td>
<td>Excellent</td>
<td>Good</td>
<td>Fair</td>
</tr>
</tbody>
</table>

2) Digital subtraction

A recent development has been the use of digital subtraction for angiographic studies. The roentgenographic information is taken from the fluoroscopic screen by photomultiplier tubes and transformed into a digital format. By obtaining information from the fluoroscopic screen immediately before passage of the contrast material through the blood vessels, the background information can be subtracted from that obtained following the injection of the dye, so that only the opacified blood vessels are visualized. The sensitivity of digital subtraction systems permits a decrease in the amount of dye injected to a fraction of that required for conventional film-screen angiography. Such savings in the amount of contrast material used per study can be important in patients with borderline congestive heart failure or renal failure. The combination of smaller catheters and digital subtraction equipment has enabled some patients to be examined as outpatients. Recently, the development of nonionic contrast media for intravascular injection has enabled angiography to be done with less patient discomfort but at greater cost (nonionic contrast agents are more expensive).

b. Intravenous

Intravenous digital subtraction angiography has been largely discontinued. A large volume of contrast material is injected rapidly so the dye travels as a bolus through the pulmonary system into the left heart and out the aorta to the brachiocephalic vessels. Such studies are successful only if the cardiac output is good so that the dye arrives as a relatively dense bolus. Since it requires the injection of large volumes of dye, congestive heart failure may be precipitated or renal failure induced. The procedure can be done on an outpatient basis but often provides studies that are not visually ideal. The intracranial circulation can be only grossly assessed because of the superimposition of major blood vessels. The indications for intravenous digital subtraction angiography are vascular disease of the brachiocephalic vessels and occlusion of the intracranial dural venous sinuses. The technique is being used less and less, and intra-arterial digital subtraction arteriography has virtually supplanted it.

c. Utilization characteristics

Table 3 compares the various angiographic techniques.

F. Cerebral Blood Flow and Metabolism

Cerebral blood flow can be measured in patients with cerebrovascular diseases by a number of methods including both tomographic (e.g., positron emission tomography [PET], xenon-enhanced x-ray CT, and single-photon emission tomography [SPECT]) and regional nontomographic methods employing the diffusible radio tracer xenon.

The only method that provides regional measurements of both cerebral blood flow and metabolism is PET. Studies in patients with stroke have provided information on regional cerebral blood flow, the cerebral metabolic rate for oxygen (CMRO₂), the oxygen extraction fraction (OEF), the cerebral blood volume (CBV), and the cerebral metabolic rate for glucose.

Extant PET scan data reveal that a complex sequence of events occurs during cerebral ischemia, ranging from an initial disproportionate reduction in cerebral blood flow relative to CMRO₂ with an accompanying elevation of OEF and apparent anaerobic glycolysis, to a period of reactive hyperemia (i.e., "luxury perfusion"), and finally to a stable period of reduced cerebral blood flow...
and metabolism. During evolving cerebral ischemia, there is no reliable way to distinguish viable from nonviable tissue.

Measurement of cerebral hemodynamics (i.e., cerebral blood flow, CBV) and metabolism has been used to supplement currently available clinical and radiographic data in the evaluation of the effect of severe carotid artery stenosis on the hemodynamic status of the cerebral circulation. Severe carotid stenosis may be associated with normal or abnormal cerebral circulation and metabolism. There is no known prognostic or therapeutic methods to distinguish these two circumstances. At present, measurements of cerebral blood flow and metabolism remain primarily research tools.

VI. Status of Patient Following Stroke

Classification of an individual's capabilities and limitations following stroke is essential for a variety of clinical, epidemiologic, and economic reasons ranging from choice of treatment to planning for discharge and evaluation of rehabilitation efforts. Unfortunately, classifications of this sort require describing a broad range of subtle variations with a limited number of descriptors, often forcing a choice between overwhelming (and difficult to collect) detail and superficial generality.

Ideally, assessment is done with scales that are objective, reproducible, and capable of serial administration throughout the poststroke period. In the cases of cognitive status and communicative capabilities, these goals have been relatively well met. However, since functional abilities depends on a complex web of neurologic status, preexisting comorbidity (such as cardiovascular disease and osteoarthritis), educational background, family support, and even the accessibility of housing and the workplace, it is not surprising that no single measure has covered this latter area well.

Over the years a number of scales have been adapted or developed to assess cognitive status, communicative capabilities, and functional abilities following stroke. Some of the more common scales are discussed briefly in the following sections.

A. Cognitive Status

Cognitive status following a stroke may range from no impairment to coma. During the early stages of recovery, clinical monitoring is often sufficient. However, once the acute stage has passed, severe cognitive limitations, as well as less obvious deficits in higher-order function (e.g., judgment, planning, reasoning) and visual perception may require evaluation with detailed neuropsychological testing. Similarly, while indications of a patient's impulsivity and safety can often be obtained from observations made by a nurse, a physician, or the family, a more detailed evaluation by an occupational therapist and/or with psychological testing may be necessary. Assessment batteries, such as the Wechsler Adult Intelligence Scale-Revised, the Wechsler Memory Scale-Revised, and the Halstead-Reitan neuropsychological tests, provide useful information in these areas and are widely accepted.

It should be remembered that depression, whether due to organic changes in the brain or to reactive factors, may occur following a stroke. While the issue of prophylactic treatment of depression is controversial, the possibility of a depression that delays functional improvement following stroke should be considered, even in a patient who does not appear dysphoric.

B. Communicative Capabilities

Stroke patients with communication problems are traditionally categorized as having aphasia, dysarthria, or apraxia. Nevertheless, careful assessment usually reveals impairment in multiple areas.

Most standardized examinations for aphasia are based on the concept that language involves input (auditory, visual, tactile), processing, and output (speech, writing, gesture). Testing attempts to assess globally these components to help plan a language program. However, additional testing of specific areas, such as auditory comprehension, may be needed to isolate an individual patient's limitations. (Tests frequently used to examine aphasic language disturbances include the Porch Index of Communicative Ability, the Boston Diagnostic Aphasia Examination, the Minnesota Test for Differential Diagnosis of Aphasia, the Western Aphasia Battery, the Token Test, and the Reading Comprehension Battery for Aphasia.)
TABLE 4. Rankin Disability Scale*  

<table>
<thead>
<tr>
<th>Grade</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>No significant disability: able to carry out all usual duties of daily living</td>
</tr>
<tr>
<td>2</td>
<td>Slight disability: unable to carry out some previous activities, but able to look after own affairs without assistance</td>
</tr>
<tr>
<td>3</td>
<td>Moderate disability: requiring some help, but able to walk without assistance</td>
</tr>
<tr>
<td>4</td>
<td>Moderately severe disability: unable to walk without assistance and unable to attend to own bodily needs without assistance</td>
</tr>
<tr>
<td>5</td>
<td>Severe disability: bedridden, incontinent, and requiring constant nursing care and attention</td>
</tr>
</tbody>
</table>

TABLE 5. Barthel Index  

<table>
<thead>
<tr>
<th>Self-care Index</th>
<th>Grade</th>
</tr>
</thead>
<tbody>
<tr>
<td>Drinking from a cup</td>
<td>1</td>
</tr>
<tr>
<td>Eating</td>
<td>2</td>
</tr>
<tr>
<td>Dressing upper body</td>
<td>3</td>
</tr>
<tr>
<td>Dressing lower body</td>
<td>4</td>
</tr>
<tr>
<td>Putting on brace or artificial limb</td>
<td>5</td>
</tr>
<tr>
<td>Grooming</td>
<td>6</td>
</tr>
<tr>
<td>Washing or bathing</td>
<td>7</td>
</tr>
<tr>
<td>Urinary incontinence</td>
<td>8</td>
</tr>
<tr>
<td>Bowel incontinence</td>
<td>9</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Mobility Index</th>
<th>Grade</th>
</tr>
</thead>
<tbody>
<tr>
<td>Getting into and out of chair</td>
<td>10</td>
</tr>
<tr>
<td>Getting onto and off toilet</td>
<td>11</td>
</tr>
<tr>
<td>Getting into and out of bathtub or shower</td>
<td>12</td>
</tr>
<tr>
<td>Walking 50 yards on level surface</td>
<td>13</td>
</tr>
<tr>
<td>Walking up/down one flight of stairs</td>
<td>14</td>
</tr>
<tr>
<td>Propelling or pushing wheelchair (if not able to walk)</td>
<td>15</td>
</tr>
</tbody>
</table>

In contrast to aphasia, apraxia and/or dysarthria impairs speech intelligibility as the result of disrupted motor control. A current and frequently used method to determine speech intelligibility is the Assessment of Intelligibility of Dysarthric Speech, which measures the number of intelligible and unintelligible words spoken and the rate of speech as a patient reads sentences and single words. Measurements such as this are useful in determining a baseline of performance before beginning a speech rehabilitation program as well as in establishing goals and measuring progress.

C. Functional Abilities  
A useful classification of physical abilities and the performance of self-care should be objective, reproducible, and applicable to the wide variety of stroke patients. Unfortunately, the many functional and activities of daily living scales in use testify to the difficulty of meeting these criteria. Nevertheless, standardization of continuing observations is needed if treatment approaches and outcomes are to be assessed and compared objectively.

1. Retrospective Evaluation  
The choice of a functional classification system is arbitrary. However, two scales that are in relatively widespread use (the Rankin Scale and the Barthel Index) may often be appropriate. The first of these, the Rankin Scale (Table 4), places a subject in one of five broad categories ranging from Grade 1 (no significant disability) to Grade 5 (severe disability requiring constant care). The Rankin Scale is broad but can be used reliably for both prospective and retrospective assessments.

2. Prospective Evaluation  
The broadness that allows the Rankin Scale to be used retrospectively obscures small functional changes that may be important. Thus, in prospective studies in which more detailed functional classification is possible, more specific and detailed scales may be appropriate. An example of such a scale that is widely known and has had some evaluation is the Barthel Index (Table 5). Each of the 15 items on the Barthel Index is graded on four levels (Level 1, independent; Level 2, independent with assistive device [such as a cane or an adapted eating utensil]; Level 3, requires assistance from another person; Level 4, completely dependent on another person). A widely adopted modification of the Barthel Index is that reported by Granger et al.
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2. Hunt, Hess. 1968
3. Framingham profile. 1983

KEY WORDS  • cerebrovascular disorders  • classification
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Classification of cerebrovascular diseases III.

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