REPORT OF THE JOINT COMMITTEE
FOR STROKE FACILITIES

VII. Medical and Surgical Management of Stroke

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Sensory and motor systems

Differential Diagnosis

Irreversible Coma ("Brain-Death")

First Aid

Transportation

Immediate Management in the Hospital (airway, adequate lung ventilation)

Subsequent Management (monitoring, respiration, neurological status, blood pressure, EKG, hypothermia; fluids, electrolytes and nutrition; urethral catheterization, EEG, CBF, hypertensive encephalopathy [differential diagnosis—cerebral infarction, intracranial hemorrhage, sequelae of head injury, other conditions; initial treatment, Table 1—antihypertensive drugs for parenteral administration in hypertensive encephalopathy])

Complications

Arterial hypertension

Cerebral edema and intracranial hypertension (adrenocorticosteroids, hypertonic solutions)

Inappropriate ADH secretion

Diabetes insipidus

Hyperglycemia

TIA and Stenotic Lesions of the Neck Vessels

Medical Treatment

Surgical Treatment

Basic evaluation for operation (laboratory tests, other special procedures, arteriography)

Contraindications to operation (acute neurological deficit, occlusion of the internal carotid artery, occlusion of the vertebral artery, concomitant myocardial or pulmonary disease)

Anesthesia management

Cerebral monitoring and protection (monitoring—local anesthesia, EEG, cerebral venous O₂ saturation, internal carotid back pressure—protection—internal shunts, hypothermia, hypercapnia and hypocapnia, hypertension)

Surgical techniques

General principles (selection of optimal lesion for surgery, sequence of operations—tandem lesions, surgically accessible; bilateral carotid lesions, accessible and inaccessible lesions occurring in tandem, timing of multiple operations)

Specific operative procedures (aortic arch lesions, carotid bifurcation lesions, vertebral artery lesions, intracranial arteries)

Complications of carotid endarterectomy

Related to general anesthesia—cardiac problems, airway problems, hypotension

Related to cervical wound—infecction, hematoma, nerve paresis, parotitis, airway obstruction

Related to the carotid artery—postoperative disruption of the artery, false aneurysm at the endarterectomy site, infection in Dacron patch grafts

Intraoperative causes—embolism from necrotic plaque or aggregation of platelets, cerebral ischemia

Postoperative causes—intimal dissection leading to thrombosis of the carotid artery reconstruction, arterial thrombosis for other technical reasons, hypotension, intracerebral hemorrhage or edema, headache

Postoperative management

Medications (anticoagulants, antihypertensive drugs)

Acceptable surgical morbidity and mortality

Management of patients with recurrent symptoms of cerebrovascular insufficiency

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VII. Medical and Surgical Management of Stroke

BY CLINICAL MANAGEMENT STUDY GROUP

JAMES F. TOOLE, M.D., CHAIRMAN, 1 B. LIONEL TRUSCOTT, M.D., PH.D., VICE-CHAIRMAN, 2
WILLIAM W. ANDERSON, M.D., 3 PHILIP R. ARONSON, M.D., 4 F. WILLIAM BLAISDELL, M.D., 5
HARRIET F. DUSTAN, M.D., 6 MARK L. DYKEN, M.D., 7 ARCHIE R. FOLEY, M.D., 8 JAMES H. HALSEY, JR., M.D., 9
THOMAS W. LANGFITT, M.D., 10 MONTAGUE S. LAWRENCE, M.D., 11 AND JOHN (SEAN) F. MULLAN, M.D., 12

Abstract:

Because it contains material concerned with the many critical problems of diagnosis, differential diagnosis, and treatment which arise when a patient presents with a possible stroke, Medical and Surgical Management is one of the most important Sections to appear in this series of publications from the Joint Committee for Stroke Facilities. Practical aspects of the management of transient ischemic attacks (TIA), strokes-in-evolution, completed strokes, and the comatose patient are discussed in detail, and attempts made to distinguish between ischemic and hemorrhagic stroke. Recommendations of the study group are listed.

Foreword

The group studying management of the stroke patient was charged with responsibility for developing optimal, yet feasible, methods adaptable to community and referral hospitals. It soon became evident that there is an optimal goal for good care, and that artificial division into several levels (depending on hospital or community size) might render disservice to the patient. The study group and their consultant subgroups have labored to produce a report providing current information on the best diagnosis and treatment with full knowledge that new discoveries will change radically approaches to some of these problems.

One conclusion is certain—that the term "stroke" no longer can be considered as referring to a single all-inclusive diagnosis. The condition has many ramifications calling for great diagnostic and therapeutic precision and individualization. In addition, we wish to emphasize that much more can be done to treat patients today than was possible a decade ago.

Many initial disagreements had to be resolved; however, in some areas, no consensus can be reached because available data allow for different viewpoints. In such instances, a middle road was chosen, neither endorsing nor rejecting controversial modes of treatment. In future years the gaps in our knowledge may be closed, allowing definite statements to be made about these areas of uncertainty. By prior agreement we have excluded consideration of intracranial hemorrhage secondary to trauma, and spinal cord vascular disease. Furthermore, we have not discussed in detail the facilities and personnel necessary to carry out the regimens outlined in our report. Lastly, laboratory tests and special procedures, such as lumbar puncture, are

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covered elsewhere. This report concentrates on the clinical aspects of patient care.

We will outline briefly methods by which the stroke-suspect patient should be evaluated. Details of these examinations are available in modern texts of medicine and neurology. We propose to discuss the management of transient ischemic attacks (TIA), occlusion and hemorrhage in the major vessels of the brain (carotid and vertebral-basilar systems), and the comatose patient.

**Evaluation of the Stroke-Suspect Patient**

**INITIAL EVALUATION**

The patient with neurological symptoms and signs is evaluated most often at home, in the office, or in the emergency room by a physician who may see relatively few persons with neurological complaints. What follows is our concept of the initial history and examination which can be performed in the above settings to ascertain whether hospitalization and/or referral to a neurologist or neurosurgeon are indicated.

**Neurological History**

The most important aspect of diagnosing cerebrovascular disease is the physician's ability to obtain an accurate history of the present illness. In about 80% of patients with central nervous system lesions a complete neurological and family history and review of systems should permit a presumptive diagnosis of the nature and location of the lesion. Because many stroke victims have difficulty in giving such history and in remembering the sequence of events, all information obtained from the patient must be confirmed and supplemented by interviewing the family or friends. On the basis of these facts the physician should attempt to answer the following questions:

1. Are the patient's symptoms compatible with a neurological disease?
2. What area or areas of the central nervous system are involved (neuroanatomical diagnosis)?
3. What is the most likely etiology: inflammation, neoplasm, degenerative disease, or vascular disorder?
4. If vascular disease, is it thrombosis, hemorrhage, embolism, arteritis, or hypertensive encephalopathy?

**Chief Complaints.** The chief complaints should be listed, beginning with the symptom which caused the patient to consult his physician. The mode of onset, duration, frequency, precipitating factors, and course of each symptom should be determined, and the standard questions of how, what, when, where, and why should be answered in relation to each complaint. Except for chronic subdural hematoma, cerebrovascular disorders usually begin abruptly and progress rapidly.

**Family History.** A history of diabetes mellitus, hypertension, collagen disease, atherosclerosis involving other organs, or disorders of lipid metabolism having occurred in family members is particularly significant (see: Section on Clinical Prevention of Stroke. Stroke 3: 803–825 [Nov-Dec] 1972).

**Past Medical History.** Similar attacks in the past which cleared spontaneously suggest TIA, possibly from extracranial vascular occlusive disease. One should ask especially about the possibility of head injury, since subdural hematoma can mimic cerebrovascular disease.

**History of Present Illness.** The history of the present disturbance should begin with its first manifestations and move forward to the time of examination. Nature of onset and progression of symptoms are particularly important. One should ask about medications of all types. Since meningovascular syphilis may cause cerebral infarction, the possibility of syphilitic infection should be explored. Electrocardiographic (EKG) evidence of coronary artery disease increases the risk of stroke almost five times. Both left ventricular hypertrophy secondary to hypertension and cardiac decompensation increase the hazard three times. In hypertensive subjects with electrocardiographic changes, the possibility of cerebral infarction is nine times as great as in normotensive persons with normal electrocardiograms.

Prospective epidemiological studies show hypertension to be the single most reliable predictor for all different varieties of cerebrovascular disease. Even moderate blood pressure elevation found by a single determination, in either sex, at any age, increases the risk of subsequent stroke. When evidence of prolonged and sustained hypertension (e.g., left ventricular enlargement) is present, cerebral infarction or hemorrhage will occur five times as frequently as in comparable normotensive individuals. Consequently asymptomatic hypertensives must be recognized early and treated vigorously and effectively in an attempt to prevent end-stage cerebrovascular disorders.

**Physical Examination**

Careful general physical, neurological, and neurovascular examinations are essential in all cases. Details of the procedures and significance of the results can be found in many modern textbooks of medicine and neurology which, however, are not immediately accessible to many physicians. Moreover, the format of the neurological examination is not presented in a separate section of most books.
The following pages will attempt to fulfill this need.

**Vital Signs.** Fever may be associated with intracranial hemorrhage and bradycardia may indicate intracranial hypertension, especially when accompanied by increased systolic blood pressure, widened pulse pressure, and fluctuations in the level of consciousness. On the other hand, bradycardia in the presence of normal intracranial pressure suggests cardiac dysrhythmia (Adams-Stokes attacks) rather than cerebrovascular disease. Disorders of respiration and alterations in the state of consciousness are common accompaniments of the severe stroke syndrome.

**Skin and Extremities.** The presence of petechiae suggests multiple emboli due to endocarditis or a hemorrhagic disorder. Precordial or neck tenderness may indicate polycythemia; congenital vascular nevi (port-wine stains) may be surface manifestations of angiomatous malformations in the central nervous system. Cyanosis with clubbing of the digits suggests possible congenital heart disease with right-left shunt as the basis for cerebral embolization or abscess.

**Heart and Lungs.** Because cardiac and cerebrovascular diseases are frequently associated, particular attention should be given to the cardiopulmonary examination. A murmur may indicate congenital, coronary, or valvular heart disease. Bacterial endocarditis, a frequent source of cerebral emboli, is usually accompanied by a murmur. Mitral stenosis, with its characteristic murmur, is also a frequent embolic source, especially when atrial fibrillation is present. Atrial myxoma is rare, but also may result in embolization. Atelectasis, pneumonia, and pulmonary embolization are major complications of stroke.

**Neurovascular Examination**

Neurovascular examination, which is an integral part of the general physical examination, is concerned with the state of the circulation supplying the central nervous system.

**Head and Neck**

*Inspection.* Periorbital hemorrhage or ecchymoses on the head suggests trauma and a possible skull fracture. Purulent, hemorrhagic, or watery fluids in the nasal passages or ears suggest chronic infection or skull fracture. Abnormal communication between these sources and the subarachnoid space may be the cause of meningitis or brain abscess.

Inspection of the eyes is the most important part of the cephalic examination. The palpebral and bulbar conjunctiva should be observed for evidence of anemia or petechiae. The funduscopic examination often gives a clue to intracranial vascular lesions. Although the state of the retinal arterioles does not necessarily reflect the condition of the intracranial arteries, funduscopy may reveal fibrinoplatelet or cholesterol crystal microemboli indicating the probable pathogenesis, and possibly the location, of a cerebrovascular lesion. Funduscopic abnormalities may be found in many systemic disorders which are associated with cerebrovascular disease, including hypertension, diabetes mellitus, uremia, disseminated lupus erythematosus and hematological disturbances. Some estimate of the degree and duration of hypertension is usually provided by using the Keith-Wagener scale.

**Palpation**

Differences between the two sides of the face and forehead in sweating and temperature suggest functional disturbance of autonomic nerves or alterations in blood flow, possibly secondary to carotid artery disease. The two superficial temporal, the facial, and the occipital arteries should be palpated simultaneously so that differences in pulsation can be detected. In patients with external carotid artery disease, the pulse in the temporal artery is frequently diminished or absent on the affected side. Increased collateral circulation occasionally produces hyperpulsations in the temporal arteries of patients with internal carotid artery disease.

Regions of the carotid bifurcations behind the angles of the jaw should be palpated gently, one at a time, so as not to evoke a carotid sinus reflex. Subclavian arteries above and below the clavicles, and then radial arteries at the wrists should be felt simultaneously. This method enables one to detect a pulse delay in either subclavian or radial artery, as well as to identify diminished or absent pulsations in any of these vessels. Finally, the aortic bifurcation, the femoral arteries, and their branches also should be palpated; absence of these pulses, especially if hypertension is present, suggests aortic coarctation.

**Auscultation**

Auscultation of the head, neck, and orbits is probably the best single method for ascertaining on physical examination the presence of extracranial cerebrovascular disease, but its value is directly proportional to the skill and experience of the examiner. Some patients with stenosis or occlusion of the carotid or vertebral arteries in the neck have very soft and harmless-sounding bruits or none at all. Consequently, every bruit in the head, neck, or orbits of the adult should be considered suggestive of disturbed cerebrovascular hemodynamics. However, some sounds, for example venous hums and soft cephalic bruits in children and young adults, are nearly always physiological. Thus, not all bruits represent serious disease.

A systolic bruit heard at the carotid bifurcation near the angle of the jaw, or over one orbit, increases
the likelihood that ischemic vascular disease is present, although these sounds may be produced by other vascular lesions such as an arteriovenous malformation. Because bruits are not heard in all patients with significant carotid stenosis, especially if the vessel is completely occluded, their absence does not eliminate the possibility of significant cervical arterial disease. The presence of a bruit, however, may be a helpful confirmatory finding.

Blood Pressure
Brachial and Crural. The blood pressure should be measured several times in both arms, and in the supine, sitting, and standing positions. When indicated (hypertension, peripheral vascular disease), pressures in the legs should be measured as well. When both systolic and diastolic readings in the two arms differ by more than 20 mm Hg, a stenotic lesion should be suspected. Blood pressure determinations are essential, since hypertension is the major risk factor for cerebral infarction and hemorrhage. A series of blood pressure readings made under a variety of baseline and stress conditions is more valuable than a single determination.

Ophthalmodynamometry
Measurement of blood pressure in the ophthalmic artery (so-called retinal artery pressure) can be helpful in the diagnosis of carotid artery disease (see: Section on Special Procedures and Equipment in the Diagnosis and Management of Stroke. Stroke 4: 111-137 [Jan-Feb] 1973).

Neurological Examination
A screening neurological examination should encompass evaluation of the following areas of possible impairment:

State of Consciousness and Cognitive Function. Fluctuations in level of consciousness are important indicators of severe, active intracranial disease (see: The Comatose Patient, Inspection and General Evaluation). A detailed formal examination of mental status may be inexpedient or impossible. However, the patient’s cognitive function can usually be evaluated to some extent on the basis of his ability to give a concise and accurate history.

Speech. (See: Section on Stroke Rehabilitation. Stroke 3:373-407 [May-June] 1972.) Aphasia refers to a type of language disturbance, usually due to a lesion of the dominant cerebral hemisphere, and characterized by a combination of motor and sensory (expressive and receptive) components.

Among the signs suggesting aphasia are difficulty in naming objects, hesitancy, and stuttering (if not previously present). Ordinary conversation usually will reveal the degree of impairment in the patient’s ability to comprehend and to organize and express his thoughts verbally. To test further the patient’s understanding, simple commands such as, “Lift your left hand in the air,” can be given. The common error of asking questions with “yes” or “no” answers should be avoided. When testing for receptive aphasia, the examiner should not give clues by gesture, expression, or direction of gaze.

Dysarthria is a disorder of the motor component of articulation and may be caused by lesions in both cerebral hemispheres, the basal ganglia, cerebellum, brain stem, or cranial nerves.

Reading and Visual Recognition. A newspaper or magazine with illustrations may be used to estimate the patient’s visual acuity, comprehension, interpretation, and retention.

Cranial Nerves
Visual Acuity. Visual acuity should be recorded quantitatively by means of a standard eye chart. Any recent alteration in vision is indication for a complete ophthalmological evaluation.

Pupils
Assymetrical pupils, regardless of reactions to light, may indicate transtentorial herniation, but also occur in many other neurological and ophthalmological disorders. A unilateral dilated pupil, with or without other third nerve signs, suggests the possibility of increased intracranial pressure.

Vascular lesions may produce Horner’s syndrome (miosis and ptosis with or without decreased sweating on the same side of the face). Disorders responsible for Horner’s syndrome are, in decreasing order of frequency: (1) brain stem lesions of the reticulospinal tract, (2) hypothalamic lesions, and (3) thrombosis of the carotid artery on the same side as the small pupil.

Fundi
The fundi should be examined for vascular changes and for papilledema. The absence of venous pulsations may be the first sign of increased intracranial pressure, appearing before the disc margins become elevated or blurred.

Visual Fields
Visual field examination by confrontation should be done in all cases, comparing the patient’s fields of vision with those of the examiner. Homonymous field defects usually are due to a cerebral lesion, and in the drowsy or aphasic patient are demonstrated most easily by failure to blink when menacing gestures are made from one side. However, large defects can be missed.

Ocular Movements
The third, fourth, and sixth cranial nerves are tested by having the patient turn his eyes through the full range of movements both volitionally and by following an object in all directions of gaze. Vertical
nystagmus suggests a brain stem lesion; horizontal and rotatory nystagmus can be found in a variety of central nervous system and labyrinthine disorders.

**Facial Movements**

The seventh cranial nerve is evaluated by noting the presence or absence of asymmetries of facial expression. Facial weakness of central origin frequently accompanies hemiplegia and, unless the lesion is in the lower pons (less than 5% of all strokes), it is always on the same side as the arm and leg weakness. Disturbances above the pontine level usually cause weakness of the lower facial muscles with relatively little involvement of forehead movement. Lesions in either the pons or the peripheral facial nerves affect upper and lower parts of the face equally. In altered states of consciousness, the examiner should make the patient grimace by simultaneous application of noxious stimuli bilaterally over the styloid processes or supraorbital ridges.

**Corneal Sensitivity**

Corneal reflexes should be tested by touching the cornea with a wisp of cotton.

**Auditory Function**

Although deafness may indicate impairment of the eighth cranial nerve, hearing tests are usually of little value in assessing the patient with acute cerebrovascular disease, unless vertebral-basilar disease is present.

**Vestibular Function**

Vertigo, dizziness, and light-headedness are common complaints of patients with cerebrovascular disease. In the absence of other neurological signs or symptoms, there may be great difficulty in determining the significance of vertigo. When the presence of vertigo can be established, one must determine whether it is due to cerebrovascular disease or to nonvascular factors. Electronystagmography may be helpful in making these decisions. (see: Section on Special Procedures and Equipment in the Diagnosis and Management of Stroke. Stroke 4: 111-137 [Jan-Feb] 1973).

**Swallowing and Related Functions**

Difficulty in swallowing may occur with bilateral corticobulbar disease or lesions of the medulla. Dysphagia is a frequent complication of stroke and when of corticobulbar origin may be accompanied by slow and inadequate tongue movements, accumulation of oral secretions, frequent choking, emotional lability, and dysarthria; the snout reflex is hyperactive. When the disease is located in the medulla, one may encounter vertigo, singultus, nausea, vomiting, Horner's syndrome, increased salivaition, nasal regurgitation, palatal weakness, and also dysarthria. Deviation of the tongue to one side may be due to a lesion involving the twelfth cranial nerve, the brain stem, or corticobulbar fibers.

**Other Signs**

**Neck Flexion.** Nuchal rigidity (resistance to flexion) may result from injury to the vertebræ and soft tissues from arthritis; from extrapyramidal syndromes; from meningeal irritation due to infection, tumor or subarachnoid hemorrhage; or, rarely, from metabolic disorders.

**Locomotion and Motor Skills**

Examination of the motor system in the ambulatory patient begins with observation of his gait. Mild hemiparesis may be manifested on the affected side by circumduction of the leg and decreased arm swing. Minimal upper extremity weakness can be detected by observing the patient with his eyes closed and his arms extended; the affected arm may drift and the distal portions assume a position of flexion and/or pronation. Specific muscles should be examined individually to identify movements such as abduction and external rotation at the shoulder and extension of the elbow, which may be impaired in patients with mild dysfunction. Other motor defects may be ascertained by having the patient perform fine motor acts, as picking up small objects, buttoning his clothes, and tying his shoes.

Patients with cerebellar disease, particularly if it affects midline structures, have a broad-based and ataxic gait. Hemiataxia superimposed on hemiparesis indicates concomitant involvement of the frontopontocerebellar pathways. A lesion in a cerebellar hemisphere may result in a homolateral intention tremor on finger-to-nose testing, or difficulty in performing rapid, skilled, and coordinated movements.

**Refluxes**

Abnormally increased deep-tendon reflexes generally indicate a central nervous system lesion. Confirmatory evidence usually is afforded by the Babinski sign and absence of abdominal reflexes.

**Sensation**

A pin and wisp of cotton are used to determine cutaneous sensation. Joint position should be identified in the toes and fingers, and vibratory sensation tested with a tuning fork. The interpretation of sensory alterations is one of the most difficult aspects of the neurological examination and isolated changes must be interpreted with caution. However, a unilateral sensory loss frequently accompanies hemiplegia. Parietal dysfunction is manifested by an impaired ability to discriminate the size, form, and shape of objects, and by failure to identify the affected side during double simultaneous stimulation. Sometimes the patient denies that he is ill and may ignore the affected side.
History and Findings Suggestive of Carotid and Vertebral-Basilar Artery Syndromes

Carotid Artery Syndrome

Hemisphere Signs
Lesions of a cerebral hemisphere usually produce contralateral weakness or paralysis, and sensory impairment is often present as well. When the dominant (usually left) hemisphere is affected, the patient often becomes aphasic. Extensive hemispheric lesions produce severe neurological deficits, hemianopia, and obtundation; if, however, hemiplegia, hemianesthesia, and hemianopia occur without altered consciousness, the lesion is often small and is likely to be located in the internal capsule.

Visual Symptoms
Transient blurring of vision or blindness in the ipsilateral eye (amaurosis fugax) sometimes accompanies carotid artery disease. Conjugate ocular deviation away from the hemiplegic or hemianopic side in the absence of focal seizures usually indicates a contralateral hemispheric lesion. If the eyes deviate toward the hemiplegic side, the lesion is probably in the brain stem.

Headaches
Unilateral headaches of migraine-like nature beginning after middle life should suggest the possibility of carotid artery disease. This uncommon symptom may be caused by atherosclerosis or cranial arteritis.

Seizures
Convulsions are unusual in acute occlusive strokes but are encountered in intracranial hemorrhage, or as a late sequel to any type of cerebrovascular lesion.

Other Clinical Findings
The following findings help to confirm a suspicion of carotid artery disease:
1. An arterial bruit over the carotid bifurcation or rarely over the orbit.
2. Rarely, diminished pulsation in the superficial temporal, internal carotid, or common carotid artery, and

Vertebral-Basilar Artery Syndrome

Usual Manifestations
The symptoms produced by vertebral-basilar disease depend upon the location of the lesion. Hemiplegia due to a disorder of this vascular system almost always presents in association with one or more of the following: disturbances of ocular movement; vertigo; ataxia; facial numbness; involvement of one or more cranial nerves (especially V and VII) contralateral to the limb paralysis; hoarseness, dysarthria, dysphagia, nausea, vomiting and hiccoughs; severe disturbance of vital signs (blood pressure, pulse, respiration, and temperature).

Dizziness is a common symptom of vertebral-basilar atherosclerosis. Occipital headaches are encountered occasionally. The precipitous onset of weakness or paralysis in all four extremities, which may or may not be accompanied by any of the preceding signs or symptoms, is highly suggestive of brain stem vascular disease. This condition may be manifest by sudden drop attacks (weakness of legs) with or without loss of consciousness. Homonymous hemianopia, cortical blindness, temporal lobe seizures, and short-term memory defects may result from ischemia of one or both posterior cerebral arteries.

Additional Clinical Findings
The following may indicate proximal arterial lesions which impair vertebral-basilar blood flow:
1. Bruits at the subclavian-vertebral junction and along the course of the vertebral artery to the mastoid process,
2. Unequal blood pressures in the two arms,
3. Blood pressure lower in the arms than in the ophthalmic arteries, and
4. The precipitation of vertebral-basilar artery insufficiency by exercising one or both arms (subclavian steal syndrome).

Transient Attacks of Neurological Dysfunction

The most important tools in the evaluation of transient attacks of neurological dysfunction are an adequate history and a careful neurovascular examination. In every case a triggering mechanism such as polycythemia, a decrease in the oxygen-carrying capacity of the blood, episodic hypoglycemia, hypotension, carotid sinus hypersensitivity, or cardiac arrhythmia should be sought. Thereafter consultation with an internist, ophthalmologist, or otolaryngologist may be indicated. Accessory studies, including lumbar puncture, skull x-rays, electroencephalogram and brain scan, all have their places. The differential diagnosis should be pressed as far as possible with these techniques before the patient is subjected to complicated, expensive, and potentially dangerous diagnostic procedures.

Angiography is indicated if there is reasonable suspicion of a cerebral mass lesion, or if a clinical diagnosis of TIA (see: TIA) has been made. The latter could be due to surgically accessible atherosclerotic stenosis of the extracranial cerebral arterial tree. Angiography should be performed only in
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hospitals where personnel with considerable experience in dealing with cerebrovascular disease are available (see: Section on Special Procedures and Equipment in the Diagnosis and Management of Stroke. Stroke 4: 111-137 [Jan-Feb] 1973).

TIA

Episodes of neurological dysfunction followed by complete recovery within 24 hours are consistent with a diagnosis of TIA. Persistence of minor neurological signs (for example, reflex changes) beyond that period indicates the presence of a permanent brain lesion, but does not necessarily alter management procedures.

Carotid System

Patients with a history of recurrent hemiparesis are most likely to have vascular insufficiency of the contralateral cerebral hemisphere. Transient aphasia is a sign that this disorder is affecting the dominant side. Transient monocular visual disturbances, although uncommon, increase the likelihood that the homolateral carotid ophthalmic artery circulation is impaired.

Vertebral-Basilar System

TIA involving the brain stem may be difficult to recognize, since symptoms frequently are vague and nonspecific (dizziness, blurring of vision, unsteadiness). Recurring brief episodes of weakness or numbness on alternate sides of the body, particularly when accompanied by diplopia, perioral numbness, and vertigo, strongly suggest episodic vascular insufficiency of the brain stem.

DIFFERENTIAL DIAGNOSIS

Disorders producing transient focal neurological dysfunction frequently are of vascular origin. However, there are many possible causes for episodic disturbances.

Epilepsy

Epilepsy from any cause may simulate the motor and/or sensory signs of cerebrovascular disorders. Focal convulsions sometimes mimic TIA so that these two conditions may be difficult to differentiate. The probability of seizures rather than TIA is increased if focal clonic movements have occurred. If there are grand mal convulsions, it is unlikely that the patient has TIA. If focal or generalized convulsions are followed by transient paresis (Todd’s paralysis), the etiologies are usually other than vascular. Whenever there is a focal component to epilepsy the physician should consider such possibilities as brain tumor, abscess, arteriovenous malformation, traumatic scar, and/or degenerative disease rather than TIA. Major phenomena indicative of a convulsive disorder include tongue-biting, incontinence, lip-smacking, upward rolling of the eyes, and postictal confusion.

General Medical Conditions

Cardiac Disorders. Many patients with cerebral atherosclerosis have coronary artery disease as well. Heart failure, causing insufficiency in a compromised cerebral vascular system, can produce transient neurological deficits. Disorders of cardiac rhythm (paroxysmal atrial or ventricular tachycardia, atrial fibrillation, heart block) and hypotension associated with, or following, myocardial infarction occasionally produce syndromes of cerebral insufficiency.

Other Diseases. Episodic and transient cerebral disturbances may be produced by intoxications (alcohol, drugs), metabolic disorders (hypoglycemia, adrenal insufficiency), postural hypotension (anhydrotic medication, prolonged bed rest, diabetic neuritis), anemia, respiratory disease, hypoxia, renal and hepatic failure, as well as fluid and electrolyte imbalance.

Migraine

Migraine is a common disorder which ordinarily does not produce focal neurological deficits. Occasionally patients with migraine will have recurrent hemianopia, aphasia, hemiparesis, or unilateral sensory loss. Rarely do these signs persist permanently.

Labyrinthine Disorders

Vertigo usually is caused by peripheral (end-organ) diseases, some of which probably are ischemic in origin. In exceptional cases brain stem vascular insufficiency, particularly in the vertebral-posterior inferior cerebellar artery distribution, is responsible. In contrast, dizziness (light-headedness) is such a nonspecific symptom that it lacks localizing value.

Intracranial Mass Lesions

Mass lesions, such as brain tumors, subdural hematomas, and abscesses, may cause transient motor and sensory disturbances, ocular symptoms, convulsions, and altered states of consciousness. These conditions may be mistaken for TIA, particularly in their initial phases (see: Epilepsy). Brain scan can be helpful in differentiating among these causes.

Ocular Disturbances

Glaucoma, optic neuritis, primary retinal vascular disease, and simple refractive errors are among the ocular disorders that may be confused with amaurosis fugax due to insufficient flow in the carotid or ophthalmic artery.

Emotional Conditions

Conversion reactions and anxiety states, including hyperventilation syndrome, may be mistaken for epilepsy, syncope, TIA, and other organic conditions.

Metabolic Disorders

Occasionally hypoglycemia and hypokalemia, for example, will produce clinical states which might be
mistaken for TIA. especially when there is associated atherosclerosis of cerebral or neck vessels.

**METHODS FOR REPRODUCING TRANSIENT NEUROLOGICAL DISTURBANCES**

If TIA are precipitated by a particular activity, such as limb exercise or a change of body position, the examining physician has a good clue to the possible etiology of the illness and also an opportunity to observe an attack under controlled conditions by having the patient perform the activity in question. Some mechanisms which may trigger episodes of cerebrovascular insufficiency include change of body position, arm exercise, and change of head position resulting in mechanical obstruction of the vertebral artery. Severe sinus bradycardia or asystole may be due to a sensitive carotid sinus; therefore, massage over this structure, when indicated, must be done under carefully controlled conditions.

**Management of the Patient**

**DECISION TO HOSPITALIZE**

Almost all clinicians admit stroke patients to the hospital during the acute phase of illness when further diagnostic or special therapeutic programs are contemplated. However, three interrelated aspects of the natural course of acute ischemic cerebral infarction should be reemphasized: (1) the probability of some recovery in most cases and of total recovery in a few, (2) the probability of progression of neurological disability during the first few days, sometimes after a minor episode, and (3) the probability that in many cases systemic complications (congestive heart failure, myocardial infarction, aspiration of gastric contents, pneumonia, or hyponatremia) will follow shortly after a major stroke. Failure to appreciate these considerations is implied in the traditional dictum that nothing can or should be done for a stroke, a cliché often implemented in classical emergency room management: “If the patient can swallow, send him home; if he cannot swallow, insert a nasogastric tube and send him home.” Such disposition might have been adequate in the past for patients with strokes for which a definite etiologic diagnosis had been established and which had been stable for several days. At the present time, reasoning of this nature is decidedly inadequate for all stroke patients, particularly for those with acute stroke of less than 48 hours’ duration, whether initially mild or severe, and regardless of diagnostic certainty.

**CONSULTATION AND DECISION TO TRANSFER**

Consultation

The following conditions might call for immediate consultation with a specialist in neurological or vascular disorders.

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**Semicoma or Coma.** If a cardiac, metabolic, or infectious cause is not evident, a neurological specialist should be consulted. Many patients in this state have mass lesions (subdural, extradural, or intracerebral hematoma, or brain tumor). Consultation is essential for diagnosis and treatment.

**Stroke-in-Evolution.** If the signs of stroke are progressing, active intervention by a specialist in vascular surgery may be indicated.

**Subarachnoid Hemorrhage.** This condition requires prompt diagnosis and effective management, and may necessitate surgical treatment of an aneurysm, intracranial clot, or arteriovenous malformation.

**TIA.** These disorders require careful evaluation, clinical diagnostic measures, and possibly angiography, and treatment by the vascular surgeon.

**Transfer**

Any of the following conditions constitutes a need for transfer of the patient to an institution with specialized personnel and facilities:

1. The comatose or semicomatose condition of the patient cannot be attributed to cardiac, metabolic, or infectious disease;
2. An exact diagnosis remains in doubt after appropriate laboratory evaluation and consultation locally with available specialists;
3. Abnormal and unanticipated laboratory findings cannot be explained;
4. The patient’s condition is deteriorating and proper management is uncertain;
5. Management requirements of the patient are beyond the capabilities of the local hospital;
6. Specialized evaluation is indicated; and
7. Adequate facilities for rehabilitation are not available locally.

In most cases, the following conditions should be handled in the local hospital:

1. Massive intracranial hemorrhage. If the patient would not survive transfer, then he should be given the best possible treatment available in the community hospital.
2. Cerebral embolism. If a cardiologist is available for consultation, the underlying cardiac or peripheral vascular disease can be treated in the community hospital, unless cardioversion and other special procedures beyond local capabilities are considered (see: Section on Clinical Prevention of Stroke. Stroke 3: 803–825 [Nov-Dec 1972]).
3. Completed stroke. Most cases in which the diagnosis is clearly established and emergency transfer is unnecessary require appropriate help with the rehabilitation program and with recommended therapy designed to prevent further episodes (see: Section on Stroke Rehabilitation. Stroke 3: 373–407 [May-June] 1972).

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*Stroke, Vol. 4, March-April 1973*
THE COMATOSE PATIENT

Coma is that state in which the patient is unresponsive to environmental stimuli. The condition may result from many causes which either are of generalized (systemic) origin secondarily affecting the brain or arise from intracranial lesions. Severely depressed consciousness seriously threatens life, because the protective reflexes regulating blood pressure, heart, and respiratory functions are compromised. Coma secondary to stroke may be due to massive intracranial hemorrhage, extensive cerebral hemispheric infarction, and lesions of the upper brain stem. Consciousness is not lost ordinarily with small infarctions in the hemispheres. Deterioration of alertness following a stroke may be due to a number of factors, including progressive intracranial hemorrhage, extension of infarction, cerebral edema with or without transtentorial herniation, hypoxia, infection, and metabolic derangements.

Initial Approach

The first step in managing the comatose patient is to institute emergency treatment, followed by an attempt to obtain a brief, accurate history, and then by a physical examination. Immediate measures to be instituted are:

1. Assure an adequate airway by whatever means necessary,
2. Determine blood pressure and other vital signs,
3. Provide access to the circulatory system by a large needle or intravenous catheter,
4. In most cases insert a nasogastric tube,
5. Insert an indwelling urinary catheter to obtain a sample for urinalysis and to record urine output during initial treatment,
6. Perform a funduscopic examination, and
7. Obtain a complete blood count (including hematocrit determination); measurements of blood glucose, and blood urea nitrogen or its equivalent; blood gases and electrolyte determinations; liver function studies; toxicological determinations, and others when indicated (see: Section on the Laboratory Evaluation of Neurovascular Disease [Stroke]. Stroke 3: 503–526 [July-Aug] 1972).

History

Because many comatose patients are brought to the emergency room unaccompanied by a reliable informant, an adequate history is often difficult to obtain. In the absence of other sources, the patient's personal belongings may afford relevant evidence; they should be searched for medical information cards, empty bottles, appointments to a doctor's office, prescriptions, and any other clues. An informant, if available, should be asked about the patient's use of drugs or alcohol and about his social and occupational history. Exposure to toxins is a possible cause of coma in industrial laborers, farmers, gardeners, and hobbyists. Details of any injury, particularly to the head, in both the recent and remote past should be obtained. A thorough systems review should be done with particular emphasis on heart disease, hypertension, kidney disease, diabetes mellitus, allergies, convulsions, syncope, and mental illness.

The above points in the history are important because they may give clues as to whether one of the three most common causes of sustained, deep alteration of consciousness is involved. These are:

1. Supratentorial or infratentorial lesions, which are usually of vascular, infectious, traumatic, or neoplastic origin,
2. Encephalopathy secondary to systemic disturbance (most commonly an exogenous toxin), and
3. Psychiatric disorders (catatonic and hysterical states).

Whether or not the historical assessment gives some indication as to the cause of the patient's problem, the next step is to establish the anatomical locus of any abnormality, and to determine the degree of interference with consciousness.

Inspection and General Evaluation

The first step is to undress the patient and carefully make an overall inspection with particular attention to scalp, skin, nail beds, mucous membranes, sclerae, body orifices, and abnormal postures. In addition, the following information should be recorded early: temperature, blood pressure, respiratory function, heart rate and rhythm.

The examiner's approach should be systematic, so that some of the most obvious physical signs will not divert him from making an all-inclusive inspection and complete examination of the patient. For example, there may be an ecchymosis in the mastoid region (a sign sometimes associated with basal skull fracture), but smelling the patient's breath may suggest an alternative diagnosis of diabetic ketoacidosis, uremia, hepatic failure, or alcohol intoxication. As another example, a blood pressure reading of 210/110 mm Hg does not necessarily mean that hypertensive encephalopathy is the cause of coma.

A thorough general physical examination is imperative. Important areas include the neck, heart, lungs, abdomen, lymph glands, and extremities.

Physical Signs

The standards usually applied by anesthesiologists in grading depth of anesthesia provide a helpful approach to the analysis of unresponsive patients.
Depth of anesthesia (or coma) increases from Stage 1 through Stage 3, and three planes of decreasing responsiveness are recognized in the latter stage. These categories are based upon the characteristics listed below:

1. Pattern of respiration
2. Ocular and vestibular systems
   a. Spontaneous phenomena
   b. Induced phenomena
3. Sensory and motor systems
   a. Voluntary (purposeful) responses
   b. Responses of reflex origin
4. Meningeal signs

Respiratory Pattern. Respiration varies with the degree and cause of alteration in consciousness, and bears some relationship to the location of the lesion. Although breathing is automatic when one is conscious, voluntary control can be superimposed. In the comatose state breathing is wholly automatic. The rate, depth, and rhythm may be a reflection of the anatomical level of nervous system dysfunction. A change from one pattern of respiration to another is an observation of extreme importance, because it denotes that the level of neural function has altered. A patient may pass from normal to Cheyne-Stokes respiration as cortical function is impaired. This may be followed by rhythmic hyperventilation as the central pontine area is released from higher controls (Stage 3, Plane 2), and by periodic breathing as medullary structures in the lower brain stem are no longer influenced by rostral mechanisms. Finally, Biot’s ataxic (irregular) respiration may occur when medullary drive persists exclusively (Stage 3, Plane 3). This entire pattern typifies the respiratory deterioration associated with any supratentorial lesion which compromises brainstem function in a progressive and more or less orderly fashion.

In contrast, lesions of the posterior fossa may compress the brainstem rapidly and change the respiratory pattern from normal to ataxic within a matter of minutes. Therefore, disorders of the posterior fossa are potentially more dangerous and often constitute greater emergencies than do supratentorial disturbances.

Ocular and Vestibular Systems. The necessity for funduscopic examination during emergency evaluation of the patient already has been noted. This should include a careful analysis of the retinal vascular pattern, a search for hemorrhages, exudates, microaneurysms and emboli, estimation of retinal venous pulsations, and notation of papilledema.

Eyelid Tone
Although many authorities state that resistance to passive eyelid opening indicates a psycho-
Oculocephalic reflex (doll’s-head eye phenomenon)
The reflex is tested by turning the patient’s head quickly from side to side and then tilting the chin up and down while observing eye position. In a conscious person, when the head is moved passively, the eyes tend to lag, particularly if there is a considerable degree of (voluntary) visual fixation. In the obtunded patient the eyes will lag behind the head movement only if pontine-mediated gaze reflexes are preserved. Therefore, if the response is absent in the obtunded patient an extensive mid-to-upper brain stem lesion should be considered, especially if there is other evidence of structural brain stem disease. Absence of the oculocephalic reflex, as indicated by lack of deviation when the head is rotated, will take place in the deeply comatose patient regardless of the cause of the unconscious state.

Vestibuloocular reflex (ice water caloric test)
This reflex tests the vestibular apparatus and its brain stem connections; its response varies directly with the state of consciousness. The test is performed by douching one external auditory canal with ice water. Unless the labyrinthine complex has been rendered inoperative by some preexisting disorder, the patient showing no response can be concluded to have extensive brain injury (Stage 3, Plane 3).

Normally the introduction of ice water into the external auditory canal causes coarse nystagmus in all fields of gaze, with the fast phase directed away from the stimulus. In the alert patient, vomiting, ataxia, and past-pointing are induced. During drowsiness the eyes deviate toward the side of stimulation, and nystagmus is less coarse than in the alert state but continues to show a fast phase away from the stimulus. In slightly deeper stages of unconsciousness tonic deviation of the eyes to the side of the stimulation is retained, but there is no nystagmus. Absence of nystagmus usually indicates some abnormality in cerebral function.

If the central nervous system has no lateralized interfering abnormality, the ocular responses of the comatose patient to cold caloric stimulation are those associated with the typical waking state, even though the patient may neither respond to painful stimuli nor exhibit corneal reflexes. The associated responses of vomiting and past-pointing do not occur during coma.

Tonic deviation of the eyes to the right which does not vary when the head is moved to the right suggests a destructive lesion either in the left side of the brain stem or in the right cerebrum. If the eyes do not move to the left of the midline after ice water stimulation of the left external auditory canal, it can be concluded that a lesion exists in the left posterior fossa, provided the patient does not have a “dead labyrinth” or barbiturate intoxication.

Sensory and Motor Systems. Pinprick is not an adequate stimulus for sensory evaluation of the comatose patient. The patient should be stimulated by means of a painful maneuver (as forced flexion of the great toe or distal phalanx of the thumb) to determine whether he withdraws and reacts in the normal protective manner. Both sides of the body should be tested in order to distinguish asymmetrical responses. This statement applies also to the face, where bilateral supraorbital stimulation will indicate the symmetry of facial grimace.

Voluntary (purposeful) responses must be differentiated from those which are reflex. Turning the head toward a noxious stimulus applied to the face, for instance, is always reflex (similar to the “rooting” reflex of the infant). If the head is turned away from the stimulus, the response is probably voluntary. Withdrawal of a limb in response to pain almost certainly indicates a “high level” response; however, extension, adduction, and pronation, such as occur in decerebrate posturing, should always be considered reflex in origin.

Interpretation of changes in the deep tendon reflexes must be made with caution, since they are unreliable indicators of the depth of unconsciousness. Hyperactivity of deep tendon reflexes may have no significance unless it is unilateral, or accompanied by clonus. The absence or marked depression of deep tendon reflexes may indicate a preexisting peripheral neuropathy which might be associated with the cause of coma, e.g., diabetic acidosis or hepatic encephalopathy.

The presence or absence of the Babinski sign is unreliable in evaluating the comatose patient. Even in early stages of unconsciousness due to barbiturate intoxication, the Babinski sign may be present either unilaterally or bilaterally. If the patient has grasping or sucking reflexes, one can conclude that the corticospinal or corticobulbar tracts are affected. Changes in muscle tonus may help in lateralizing a neurological abnormality. If the limbs are spastic on one side and flaccid on the other, it is possible that both sides of the central nervous system are involved.

Decerebrate posture is an ominous sign and indicates either supratentorial disease or midbrain transection. Complete recovery can occur in such cases, however, particularly when the posturing follows a prolonged generalized convulsion.

Meningeal Signs. In subarachnoid hemorrhage, signs of meningeal irritation may not appear until several hours or days after the ictus. Furthermore, in the comatose patient, the meningeal signs due to either
subarachnoid hemorrhage or meningitis may be absent or difficult to elicit.

**Differential Diagnosis**

The cause of coma may be determined by the history, physical and neurological examinations, appropriate laboratory tests, and sometimes by a period of observation to ascertain whether there is progressive neurological improvement or deterioration.

Loss of awareness due to a single, unilateral cerebral lesion is infrequent unless it results from a generalized convulsion. In a patient with supratentorial disease, loss of awareness usually indicates superimposed brain stem complication.

The following may be helpful in differentiating the causes of coma:

1. In coma of metabolic origin, confusion and stupor usually precede the appearance of motor signs. Generally the presence of impaired awareness without motor signs, or with symmetrical motor signs and intact pupillary responses, should suggest toxic or metabolic encephalopathy due to depressant drugs, electrolyte imbalance, liver or kidney failure, endocrine disturbances, and many other causes. This type of coma is often associated with flapping tremor, myoclonus, and hyperventilation.

2. Intracranial lesions leading to coma may be vascular, infectious, neoplastic, or traumatic in origin. Supratentorial disturbances usually manifest themselves with appropriate focal signs and often, but not invariably, follow a specific pattern of neurological deterioration (see: The Comatose Patient, Physical Signs). Infratentorial lesions frequently produce localizing brain stem signs before the onset of coma.

**Irreversible Coma (“Brain Death”)**

With the development of artificial methods for maintaining circulation and respiration and the success of organ transplants, the subject of irreversible coma has become one of considerable importance and controversy, with legal, religious, and philosophical overtones.

The physician responsible for a comatose patient must preserve life if there is any hope of eventual recovery. He must decide the duration and extent of treatment in consultation with medical colleagues and with the family. Justification for prolonged artificial maintenance of vital functions is either to ascertain that recovery is impossible or to ensure viability of organs for transplantation. In the latter instance, the decision that death exists should be made by two or more physicians who are not immediately concerned with performing the operation.

The age-old criteria of death (cardiac and respiratory standstill) are being modified to include new concepts of irreversible loss of brain function (brain death). Various criteria for irreversible coma have been established and the physician is urged to examine the policy which pertains in his own hospital or region.

The diagnosis of brain death is made clinically, then substantiated by electroencephalography (EEG) and perhaps by cerebral angiography or by cerebral blood flow (CBF) studies. The history is important, because deep hypothermia (due to exposure, for example), as well as intoxication with barbiturates and other central nervous system depressants, can produce both physical and EEG findings suggesting an irreversible state.

As the brain stem ceases to function, profound changes occur in the vital signs. Body temperature usually falls, although in some cases hyperpyrexia results from respiratory complications or hypothalamic damage. The blood pressure gradually drops, and increasing quantities of vasopressor drugs are required.

Once spontaneous respirations cease, their absence must be demonstrated for five minutes after the mechanical respirator is removed before the patient is pronounced dead. Even this period of time is inadequate to be certain of permanent respiratory arrest unless the patient has been breathing room air while on the respirator and has an essentially normal PaCO2. The respiratory centers in the brain stem are driven by both CO2 and O2 concentrations, the latter operating through peripheral chemoreceptors. A patient with a high PaO2 and low PaCO2 will not breathe spontaneously until the respiratory center is adequately stimulated by hypoxia and/or hypocapnia.

The patient with brain death is “completely comatose.” Painful stimulation produces no detectable response, and all reflexes have disappeared. Doll’s-head eye movements and corneal and pharyngeal reflexes are absent, and in most cases the pupils are widely dilated and fixed to light; rarely the pupils are small and fixed. The eyes do not move in response to irrigation of the ear canals with ice water. The patient is flaccid, all deep tendon reflexes are usually absent, and there is no response to plantar stimulation.

Several EEG criteria for cessation of brain function are now generally accepted, which may be defined further with more experience (see: Section on Special Procedures and Equipment in the Diagnosis and Management of Stroke. Stroke 4: 111-137 [Jan-Feb] 1973). The patient should not be significantly hypothermic or under the influence of central nervous system depressants at the time the EEG is obtained. The EEG must be isopotential (no detectable activity), both at standard gains (10 mv/mm) for each channel and at twice the standard...
gains. Ten minutes is the minimal recording time. At some point maximum gain should be used, although this procedure often introduces an artifact which only an experienced electroencephalographer can distinguish from minimal electrical activity. The EEG should be repeated after 24 hours, and if both records are isopotential, then a diagnosis of brain death is strengthened.

Another criterion of brain death is absence of CBF, which can be demonstrated by failure of the intracranial vessels to fill with opaque material injected into the carotid and vertebral arteries, or failure of a radioactive isotope injected into the carotid artery to be cleared from the brain. These techniques are rarely necessary and not always helpful, since some patients demonstrate adequate CBF but have no cerebral function. In such cases the brain does not absorb oxygen from the blood passing through it, and the oxygen content of the jugular venous blood is identical to that of the arterial blood.

The judgment that brain death has occurred should be made by an informed physician employing the criteria listed above.

**First Aid**

Coma in a stroke patient constitutes an emergency requiring first aid and usually rapid transportation to a properly equipped hospital. The first person to reach the patient can assist in many ways, but professional help should be obtained as soon as possible. In scattered areas of the United States the telephone number 911 has been set aside to give toll-free access to an emergency communication center.

If a medical emergency system is not available, a physician or hospital emergency ward should be called at once.

The physician should determine whether to examine the patient at the scene or have him transported to the hospital at once. On certain occasions instructions may have to be transmitted by telephone describing the signs of airway obstruction, and giving instructions on extending the patient's neck, opening his mouth, pulling the tongue forward, and clearing the upper airway of dentures, secretions, or vomitus. If respirations are very shallow or if respiratory rate is less than eight per minute, mouth-to-mouth ventilation may be needed. If this is not required, the patient should be turned into the semiprone position with the head somewhat dependent, thereby permitting the tongue to fall away from the posterior pharynx, and fluid to flow out of the mouth rather than into the trachea. A comatose patient should not be placed on his back or have a pillow under his head. Flexion of the head and neck tends to close off the upper airway, and aspiration is more likely to occur in the supine position.

If the physician elects to attend the patient at the scene, his first step on arrival is to determine the respiratory status. When death from respiratory insufficiency appears to be imminent despite the measures described above, further action is required immediately. An oral airway, or preferably a double-ended device, such as the Resuscitube, which permits effective mouth-to-mouth resuscitation, should be available.

Generalized seizures sometimes occur in comatose stroke patients, or the coma may be the result of a seizure. At this stage, differentiation of stroke from epilepsy may be difficult. During convulsions the airway may become obstructed and many patients become cyanotic. Postictal confusion or coma follows and is characterized by deep, often stertorous, respirations.

If there is sufficient warning of an impending seizure, a properly padded object, such as several tongue depressors taped together, should be inserted between the jaws if possible. Once the teeth become clenched attempts to pry the jaws open with metallic objects may be dangerous. Retraction of the mandible and simultaneous extension of the head will usually improve airway patency. The patient may have to be restrained lightly to prevent injury. In order to end seizures quickly, appropriate anticonvulsants should be administered, usually diphenylhydantoin sodium (Dilantin) or primidone (Mysoline). Repeated generalized seizures (status epilepticus) require prompt medical therapy, usually with diazepam (Valium). Convulsions would be the major reason for administering sedative medication to such a patient. The use of narcotics in patients with depressed states of consciousness for any other reason should be discouraged.

After the physician is satisfied that airway patency is established, he determines the patient's vital signs (i.e., blood pressure, cardiac and respiratory rate and rhythm, and temperature). Peripheral shock (manifested by arterial hypotension and tachycardia) is uncommon in a patient with acute stroke, and when present may signify either terminal brain stem decompensation or a failing cardiovascular system secondary to myocardial damage. There is no treatment for terminal collapse of brain stem function, and treatment of cardiac failure is beyond the scope of this presentation.

Arterial hypertension in the comatose patient may reflect an antecedent disorder or may be secondary to the immediate intracranial event. Disturbances of cardiac rate and rhythm (including EKG changes) are found commonly in acute stroke, particularly in intracranial hemorrhage. The physician should determine whether these changes are the cause or result of the cerebrovascular lesion.

The physician should now critically evaluate the level of consciousness as an essential part of the
neurological examination. A brief description of the patient's reaction to command and to painful stimuli should be recorded. Reporting specific responses is preferable to use of terms such as "comatose," "semicomatose," or "stuporous." A note stating that the "patient moves one side but not the other in response to pinching the ear lobes" is valuable because it permits comparison with subsequent reactions to the same stimulus. A progressive decrease in level of consciousness is the most reliable sign of deterioration in a patient's condition.

The pupils should be examined for size and reaction to light. Unilateral pupillary dilatation may indicate transtentorial herniation as the result of localized pressure.

Integrity of motor pathways is assessed by noting limb movements in response to painful stimuli. The significance of these neurological signs is discussed in detail elsewhere (see: The Comatose Patient, Physical Signs). The examination may have to be repeated so that a clear picture of the extent and progression of the lesion is provided. A written account of the patient's course during this interval is of great value to the physician or to other personnel responsible for subsequent care.

Transportation
The comatose stroke patient should be taken directly and immediately to the nearest hospital if possible, and preferably to one that is staffed and equipped to manage effectively problems of this nature.

Privately owned cars are the most readily available but the least satisfactory for emergency use. Flat-bottom vehicles, such as station wagons and trucks, are better than standard automobiles for this purpose. Vehicles are often available through police units, fire departments, taxi companies, or funeral directors, although some are not equipped for medical emergencies.

No uniform standards are established for vehicular design, equipment, staff training, or licensure of organizations providing emergency transportation. Guidelines have been proposed by the Committee on Acute Medicine of the American Society of Anesthesiologists, the National Research Council, and the American College of Surgeons. In scattered localities throughout the country are hospital-based mobile vehicles equipped to serve as emergency rooms with doctors in attendance. Although expensive, this is the most satisfactory means for immediate management and transportation of an unconscious patient. Helicopters equipped as ambulances are becoming readily available in an increasing number of locations, but their use is still limited because of high initial and maintenance costs.

The main role of attendants during transportation of the patient is to support vital functions and to prevent complications. The principal concern is airway maintenance. In some areas ambulance attendants receive special in-hospital training which enables them to perform endotracheal intubation, or possibly even tracheostomy.

The patient should be transferred in the semiprone or the lateral position. The semiprone is better for drainage of secretions and vomitus but the lateral allows more rapid access to the airway for mouth-to-mouth resuscitation. Oxygen administration by mask or nasal tube is of little benefit and can lead to dangerous accumulation of CO₂ if the equipment is operated improperly. During transportation the patient's temperature should be kept as near to normal as possible. Hypothermia is preferred to hyperthermia, but either extreme is dangerous. The tendency to swaddle the patient in blankets should be discouraged.

Although the patient should be transported with minimal delay, the driver of the vehicle must observe local traffic regulations.

Immediate Management in the Hospital
When the comatose patient is admitted to the hospital emergency area, proper evaluation and management of respiration are again the first and most important steps. The physician should keep in mind two fundamental questions: Is the airway patent? Is lung ventilation adequate?

Airway. The airway extends from the nares to the alveoli. In the adult the volume of this airway is about 2 ml per kilogram of body weight, a third of which is located above the larynx. Respiratory rate and volume are regulated normally from the medullary respiratory center in response to the concentration of arterial carbon dioxide (PaCO₂) and arterial oxygen (PaO₂). Since CO₂ functions by rendering the blood acidic, i.e., increasing the hydrogen ion concentration, expressed by a pH less than 7.35. Blood CO₂, O₂, and pH are important laboratory values to be determined concurrently, as often as indicated, in the acutely ill stroke patient. The normal ranges of these values are as follows:

- \( \text{PaCO}_2 \) 35 to 45 mm Hg
- pH 7.35 to 7.42
- \( \text{PaO}_2 \) 70 to 90 mm Hg, breathing air
  450 to 500 mm Hg, breathing 100% O₂

Complete tracheal obstruction is recognized by ineffective attempts at inspiration, cyanosis, and circulatory failure, and is most frequently caused by the tongue obstructing the pharynx, and by the aspiration of vomitus, dentures, or foreign material. Severe tracheal obstruction is rapidly fatal.

Extensive obstruction of the bronchi by aspirated and secreted liquids leads to hypoxemia and hypercapnia. The resulting respiratory acidosis
is characterized by a subnormal arterial pH and an elevated $P_{aCO_2}$. Partial and segmental obstruction of the bronchi leads indirectly to hypoxemia. As the patient hyperventilates in an attempt to overcome the obstruction, excessive amounts of carbon dioxide are eliminated from the unobstructed units and, as a result, arterial pH is raised above normal and $P_{aCO_2}$ is reduced below normal—the syndrome of respiratory alkalosis. All of these problems may appear without identifiable cyanosis.

When the diagnosis of airway obstruction has been made, the following steps should be taken in this order:

1. Clear the mouth and pharynx of foreign materials manually and by suction.

2. Introduce a sterile suction catheter, either directly by laryngoscopy or transnasally, into the trachea, and suction until all possible aspirate has been removed. Then allow the catheter to touch the carina and to produce coughing in attempting to clear lower segments of the respiratory tract. If vomitus has been aspirated, lavage with from 3 to 5 ml of bicarbonate solution is recommended. Adrenocorticosteroids should be administered promptly. If a tracheal catheter is tolerated by the patient, it may be left in place for insufflation of humidified oxygen during resuscitation.

3. Extend the head and move the mandible forward manually in an attempt to further relieve respiratory obstruction. If these maneuvers succeed, insert a double-ended pharyngeal airway to be used for mouth-to-mouth breathing when no other means of ventilation is available without delay.

4. If the above measures are not immediately successful, orotracheal intubation should be carried out with a laryngoscope. Nasotracheal intubation is accomplished best by a specialist. The endotracheal tube should be equipped with a long, soft, inflatable cuff which is molded on the tube. The cuff is inflated and further suctioning and ventilation are carried out through the tube. Sterile handling is difficult, particularly in an emergency, but this precaution is essential in attempting to prevent respiratory infection.

The use of endotracheal tubes with inflatable cuffs carries with it certain hazards, and safety measures should be observed. It is desirable to use an endotracheal tube with a soft, low pressure cuff which is inflated to the minimal pressure necessary to achieve a good seal. The cuff should have a relatively large surface area and should be deflated five or more minutes of every hour (or ten or more minutes every two to three hours), and the tube should be changed after the first 24 hours. At that time the larynx and upper trachea should be inspected indirectly through a laryngoscope to determine cord movement and possible pressure damage to the cords and upper trachea, including ulceration, granulation, or necrosis of tracheal rings. If several attempts to insert the tube into the trachea fail, a tracheostomy should be performed. Similarly, if long-term respiratory assistance with a respirator is anticipated (more than four days), a tracheostomy should be performed. Use of a cuffed tracheostomy tube requires the same precautions outlined above for the endotracheal tube. Whether endotracheal or tracheostomy tubes are used, they should be removed every 24 hours if possible and replaced by a clean tube; at these times the larynx and trachea should be inspected. Care should be exercised not to insert too large an endotracheal tube because the vocal cords may be damaged, or the arytenoids may be dislocated with the result that permanent changes in the voice will occur. Most experts believe that tracheostomy is preferable to cricothyreotomy, because irreparable damage may result from the latter procedure, necessitating the use of a tracheostomy tube permanently.

Because it is time-consuming and may be accompanied by excessive bleeding or other surgical complications, tracheostomy should be performed as an emergency procedure only if the equipment or expertise for oral endotracheal intubation is unavailable. Tracheostomy should be an elective procedure when it is clear that prolonged intubation will be required.

**Adequate Lung Ventilation.** After the airway has been cleared, some form of mechanical ventilation usually is required to maintain normal blood levels of $O_2$ and $CO_2$ and also to keep the alveoli adequately inflated to prevent atelectasis. Pulmonary function cannot be assessed properly by simply observing the volume of air the patient is exchanging. Tidal volume can be measured with a Wright respirometer attached to an endotracheal tube or a face mask. Multiplication of tidal volume by respiratory rate provides a measurement of the volume of air exchanged per minute. This information is important, particularly for patients who appear to be hypoxic even when they are receiving an adequate supply of $O_2$. Such patients may be tachypneic because of inadequate tidal volume, and are thereby predisposed to microatelectasis.

The most accurate means of assessing blood oxygenation is by direct measurement of $P_{aO_2}$. The patient's acid-base status should be evaluated at the same time by measuring arterial pH and $P_{aCO_2}$.

Mouth-to-tube ventilation is the most rapid means of support and when utilized should be given at a rate of 10 to 12 expirations per minute with a volume twice the normal tidal volume. A physician can learn to approximate the needed tidal volume (1,000 ml) by practicing with the Wright respirometer. However, a self-inflating bag, such as the
AMBUL bag, is preferable to mouth-to-tube ventilation and is an essential piece of equipment in all emergency units. Since the bag holds approximately 1,000 ml of gas, it accurately delivers a known tidal volume, and can be connected to an oxygen source for increased O₂ concentration.

Progressive hypoxemia in the comatose patient is not uncommon, in spite of patent airway, normal ventilation and perfusion of the lungs, and adequate peripheral circulation. In such cases blood-gas measurements show normal or decreased pH and low PaO₂. Deoxygenated venous blood perfusing unventilated alveoli and producing a right-to-left shunting or venous admixture usually can be ascribed to an inadequate ventilatory volume. The degree of shunting due to focal atelectasis can be estimated by means of the nitrogen washout technique.

**Subsequent Management**

Following emergency treatment and preliminary evaluation, further studies are required to make a precise diagnosis and to develop a program for continued therapy. Desirable diagnostic procedures and criteria for surgery in the acutely ill stroke patient are discussed further in this and other reports (see: Section on Special Procedures and Equipment in the Diagnosis and Management of Stroke, Stroke 4: 111-137 [Jan-Feb] 1973; Surgical Management, Ischemic Brain Lesions, Hemorrhagic Brain Disease).

The patient usually is transferred to an intensive care unit (ICU) or stroke unit where special attention is directed to respiratory management, to monitoring and support of his vital functions, to continued evaluation of his neurological status, and to management of any complications that may develop.

**Monitoring.** The physical status of the comatose patient is labile. Life-threatening crises occur frequently and the response to therapy is often difficult to evaluate because of the patient's changing condition. Thus, management often requires continuous monitoring of numerous vital signs.

**Respiration.** Proper respiratory function is essential to life. If the patient remains unresponsive to stimuli for longer than 72 hours, an elective tracheostomy may facilitate respiratory care. If tracheostomy is performed, the tube should be fitted with an inflatable cuff so that ventilation with a respirator can be implemented. The tracheobronchial tree is suctioned whenever secretions accumulate, and always with meticulously sterile technique. The head of the bed should be elevated 20° to 30°, the patient's head turned to the side, and pillows used to raise the opposite side of the body 45° or more from the bed. Elevation of the head lowers cerebral venous pressure and may diminish cerebrovascular congestion and edema. Turning the head to one side permits saliva to drop from the mouth instead of entering the trachea around the tracheostomy tube when the cuff is not inflated.

Several types of respiratory transducers now make possible uninterrupted observation of respiratory rate and rhythm on the oscilloscope simultaneously with the EKG. Instruments for measuring end-respiratory P O₂ and P CO₂ are available, and the output can be led to a channel of the stripchart recorder for continuous display. Unless pulmonary dysfunction is serious, these values reflect the arterial blood-gas values.

Periodic measurements of PaO₂, PaCO₂ and pH are needed to ascertain respiratory and acid-base status. Repeated puncture of an artery to obtain samples for blood-gas determinations carries the risk of thrombosis, a complication which might be lessened by using an indwelling arterial catheter.

Normally the cerebral arterioles dilate in response to increase in PaCO₂ and constrict when PaCO₂ is reduced. In an ischemic brain, however, these vessels may no longer respond to vasodilators such as CO₂. Studies of regional CBF in stroke patients and in animals have demonstrated that blood flow actually may decrease in ischemic portions of the brain when vasodilators are used. This occurs because administered CO₂ may dilate blood vessels in unaffected areas of the brain adjacent to the ischemic region, with consequent redirection of flow; in addition, a secondary rise of intracranial pressure may take place resulting from increases of cerebral blood volume. Such paradoxical CBF reactions to vasodilators are uncommon, and generally occur only within the first few days after stroke onset; their clinical significance is unknown.

Hyperventilation to the point of hypocapnia has been reported to increase blood flow in ischemic brain, a paradoxical reaction which has not been demonstrated in animal studies. In the usual clinical situation, this approach has not protected against neurological deficit resulting from cerebral ischemia. However, hypocapnia following hyperventilation may cause enough constriction of cerebral arterial vessels to decrease intravascular cerebral blood volume and secondarily to decrease intracranial pressure. Although the effect of hyperventilation on intracranial pressure may diminish within a few hours, this short-term action may keep the patient alive long enough to allow an emergency operation, for example, removal of an intracerebral hematoma.

**Neurological Status.** The patient's neurological status can be recorded at intervals by the nursing
staff on a standard evaluation form as indicated by the physician; he lists in writing those situations that will require his immediate notification. Proper use of a “watch sheet” requires some experience, but it has the advantage of easy interpretation because the patient's responses are described specifically rather than in poorly defined terms that may not have the same meaning for all personnel.

**Blood Pressure.** Arterial blood pressure should be recorded at least hourly during the acute phase. Several semiautomatic pressure-cuff devices are available, but all produce some artifacts. The most reliable method currently available for continuous blood pressure recording is the intraarterial catheter, but the technique is invasive and can introduce complications.

Continuous central venous pressure measurement is valuable in the diagnosis of impending congestive heart failure and in the regulation of fluid balance. This pressure can be recorded on the same stripchart concurrently with arterial blood pressure.

**EKG.** Standard practice in ICU and stroke units is to continuously monitor the EKG on an oscilloscope. This technique is most important for observing changes in the condition of a patient who is seriously ill.

**Hypothermia.** By slowing cerebral metabolism, hypothermia reduces the quantity of oxygen and metabolites required by the brain, protects the brain from continued ischemia or repeated ischemic episodes, and combats cerebral edema. Hypothermia is produced by placing the patient between two thermal blankets. If more rapid reduction in temperature is desired, bags of ice also are used. Body temperature is recorded at frequent intervals by means of a rectal or esophageal thermistor.

Limited experience with hypothermia has shown that it reduces intracranial pressure in patients who are comatose following an episode of severe cerebral hypoxia or ischemia. Its usefulness in managing cerebrovascular disease has not been determined, and in addition serious complications such as cardiac arrhythmias and gastrointestinal bleeding may result.

**Fluids, Electrolytes, and Nutrition.** Fluids are administered intravenously to all comatose stroke patients for the first few days. Improper regulation of fluids can cause congestive heart failure due to excessive sodium intake, cerebral edema from water intoxication, or dehydration which increases blood viscosity and further impairs CBF. Renal disease, diabetes mellitus or insipidus, as well as the long-term administration of diuretics or corticosteroids complicate the determination of fluid and electrolyte requirements. The following measurements are important in regulating fluid and electrolyte therapy:

1. Serum electrolytes (sodium, potassium, and chloride) and CO₂ are determined on admission and at 24-hour intervals thereafter, if the patient's status changes significantly. More frequent measurements may be necessary if diabetes insipidus develops or if there is inappropriate secretion of antidiuretic hormone (ADH).
2. Urine output is recorded hourly in severely ill patients and specific gravity is determined at least once every four hours.
3. Measurements of serum and urine osmolality are not needed routinely but are important in making the diagnosis of inappropriate ADH secretion and in distinguishing it from diabetes insipidus, dehydration, and salt depletion.
4. An elevation in the central venous pressure is one of the earliest clues to congestive heart failure secondary to excessive fluid administration.

Routine therapy for the comatose stroke patient consists of the intravenous administration of about 2,000 ml of 0.5% saline in glucose each 24 hours, with 40 to 60 mEq of potassium added (less when there is renal impairment). The volume of fluid is reduced in the presence of cardiac or renal failure.

Although the sodium content of the solution just described is higher than that recommended for general medical and surgical patients, the administration of lesser amounts of sodium to stroke patients usually results in hyponatremia. Those in whom hyponatremia develops in this way are usually found to have high urine and low serum osmolality. The hyponatremia is corrected by fluid restriction.

A nasogastric tube is inserted in all comatose stroke patients on admission and aspiration is carried out to keep the stomach empty. After 72 hours, intravenous feeding may be terminated and tube feedings begun if the abdomen is not distended and good bowel sounds are present. During tube feeding regurgitation and aspiration of stomach contents must be avoided. This is prevented by elevating the head of the bed, administering only a small volume of liquid at each feeding, and coloring the solution so that it can be identified easily if reflux occurs. As an added precaution, the cuff on the endotracheal tube should be inflated for one hour after each feeding.

Depending on the tolerance and caloric requirements of the individual comatose patient (1,500 to 2,000 calories per 24 hours for the average patient), tube feeding formula can be given to maintain metabolic requirements. One liter of a formula containing 50 gm protein, 100 gm carbohydrate and 35 gm fat (usually with electrolytes and
Urethral Catheterization. Instillation of a neomycin-containing lubricant into the urethra of the comatose male, and vulval preparation of the female with organic iodides should precede catheterization. Although a metal stylette or guide is rarely necessary, when their use is indicated the procedure should be done by a urologist. The catheter is connected to a closed drainage system which is kept patent (not clamped routinely) and can remain in place from four to six weeks without danger of encrustation if measures to prevent infection are adequate (e.g., application of neomycin ointment once or twice a day around the urethral orifice). Condom drainage should be utilized in the male patient whenever possible. If catheterization is necessary for longer than three weeks, a suprapubic drainage tube may carry less risk of infection than other methods.

If more than 50 ml of residual urine remains when the voiding capacity of the patient is tested, the catheter should be reinserted. In certain cases, even when the initial test is satisfactory, periodic rechecks for residual urine (due to obstruction by the prostate or by fecal impaction) may be desirable. Troublesome strictures (not due to inflammation) can be corrected by meatotomy or, less frequently, by the use of a sound.

EEG. For a general discussion of EEG and its value in the management of stroke, see the Section on Special Procedures and Equipment in the Diagnosis and Management of Stroke (Stroke 4: 111-137 [Jan-Feb] 1973), and also Irreversible Coma in this report.

CBF. Autoregulation methods of measuring CBF are discussed elsewhere (see: Section on Special Procedures and Equipment in the Diagnosis and Management of Stroke. Stroke 4: 111-137 [Jan-Feb] 1973).

Hypertensive Encephalopathy. Hypertensive encephalopathy differs from other neurological complications of hypertension (hemorrhage and ischemic stroke) in that it is related to the height of arterial pressure. The condition may occur at any age and may complicate any type of hypertension, with the exception of that due to aortic coarctation and possibly to primary aldosteronism. Although encephalopathy may develop with blood pressure elevation of either recent onset or long duration, it appears most commonly under the former circumstance, as in acute glomerulonephritis or toxemia of pregnancy. In chronic hypertension—except for that associated with uremia and chronic renal parenchymal disease—high diastolic pressure (often 150 mm Hg or above) is necessary for the development of encephalopathy.

The following course of events is typical. After a recent onset or a sudden exacerbation of long-standing hypertension, the patient has a severe headache, visual impairment, nausea and vomiting, extreme restlessness, convulsions, then stupor, and finally coma. The evolution of symptoms may extend over a few hours or several days. Focal signs may be transient and usually are absent early in the attack. Funduscopic examination may show papilledema, hemorrhage, and exudates. The CSF pressure is usually elevated. The fluid contains few, if any, erythrocytes or leukocytes, and the protein concentration may be normal or increased.

Differential Diagnosis

The diagnosis of hypertensive encephalopathy should not be made unless the blood pressure is elevated considerably. Since some conditions that produce unconsciousness, e.g., cerebral space-occupying lesions, can cause hypertension also, it is helpful to know whether the elevated pressure antedated the acute illness. Confirmation of a presumptive diagnosis of hypertensive encephalopathy is made by a therapeutic test of antihypertensive agents. The diagnosis is established if improvement follows reduction in blood pressure.

Cerebral infarction

Cerebral infarction often occurs in hypertensive patients, can cause mild hypertension, and is sometimes associated with confusion or coma. However, strokes are rarely mistaken for hypertensive encephalopathy because in the former the neurological deficit is of longer duration, is characteristically focal, and the signs fail to clear with improved blood pressure control. Distinction between the two conditions must be made primarily on clinical grounds, since EEG and CSF findings are not helpful.

Subarachnoid hemorrhage

Subarachnoid hemorrhage should not be confused with hypertensive encephalopathy. In the former, headache is of sudden onset. Blood pressure elevations occur after subarachnoid hemorrhage but the levels generally are not as high as those encountered in hypertensive encephalopathy. Other differential points characterizing subarachnoid hemorrhage are the presence of nuchal rigidity and
<table>
<thead>
<tr>
<th>Drugs</th>
<th>Intramuscular (IM)</th>
<th>Intravenous (IV)</th>
<th>Side effects</th>
<th>Onset of action</th>
<th>Remarks</th>
</tr>
</thead>
<tbody>
<tr>
<td>Diazoxide (Hyperstat-Schering)</td>
<td></td>
<td>Single dose</td>
<td>Nausea, vomiting, flushing, hyperglycemia</td>
<td>3–5 min</td>
<td>Prompt response and standard dosage will make this drug of choice when commercially available.</td>
</tr>
<tr>
<td>Sodium nitroprusside</td>
<td></td>
<td>Continuous infusion</td>
<td></td>
<td></td>
<td>Not commercially available but can be prepared easily. Requires constant supervision. Thiocyanate blood levels must be assessed.</td>
</tr>
<tr>
<td>Pentolinium (Ansolsyen-Wyeth)</td>
<td>1–20 mg</td>
<td>$10 mg</td>
<td>Urinary retention, paralytic ileus</td>
<td>IM: 30 min, IV: 5–10 min</td>
<td>Usually effective but large doses often necessary to reduce supine blood pressure since the effect is primarily orthostatic. Repeat doses usually lead to atony of bowel and bladder.</td>
</tr>
<tr>
<td>Trimethaphan camphorsulfonate (Arfonad-Roche)</td>
<td></td>
<td>1000 mg/liter</td>
<td></td>
<td>5–10 min</td>
<td>Occasionally produces dilated fixed pupils for a short period of time.</td>
</tr>
<tr>
<td>Hydralazine (Apresoline-Ciba)</td>
<td>10–50 mg</td>
<td>$10 mg</td>
<td>Tachycardia, palpitations, flushing, headache, vomiting</td>
<td>IM: 30 min, IV: 10 min</td>
<td>More effective in acute glomerulonephritis and eclampsia than in essential hypertension. Contraindicated in heart failure or coronary insufficiency.</td>
</tr>
<tr>
<td>Reserpine (Serpasil-Ciba)</td>
<td>1–5 mg</td>
<td></td>
<td>Drowsiness, stupor, Parkinsonian-like rigidity</td>
<td>2–3 hr</td>
<td>Delayed effect and somnolence are major disadvantages.</td>
</tr>
<tr>
<td>Metyldopa (Aldomet ester-Merck)</td>
<td>$250–500 mg</td>
<td></td>
<td>Drowsiness</td>
<td>2–3 hr</td>
<td>Has some disadvantages as reserpine and is less likely to be effective.</td>
</tr>
</tbody>
</table>

*Start with the smallest dose shown. Subsequent doses and intervals of administration should be adjusted according to the response of the blood pressure.
†Start the infusion slowly and adjust the rate according to the response of the blood pressure. Constant surveillance is mandatory. The concentration of the solution can be adjusted according to the patient's fluid requirements.
§Dissolve 2 g sodium nitroprusside in sufficient freshly prepared distilled H₂O to make 200 ml using a sterile glass-stoppered 200 ml volumetric flask. Since the solution does not tolerate heat sterilization, bacterial filtration is through a Seitz filter previously sterilized with live steam in an autoclave at 250 F for 1 hour. The stock solution is transferred from the volumetric flask to an amber-colored rubber-stoppered bottle by means of a sterile funnel. The stock solution and dilutions thereof should be kept under refrigeration at an average temperature of 15 C.
§The total dose should be contained in a volume of at least 20 ml and the solution should be administered from a 20 or 50 ml syringe. The blood pressure should be monitored continuously while the injection is being made. The rate of injection should not exceed 0.5 ml/min, and in order to avoid hypotension, the injection should be stopped frequently when the blood pressure is falling.
||Administer in 100 ml of a 5% solution of dextrose in distilled water over a period of 30 to 60 minutes.

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bloody CSF, and the absence of hypertensive retinal hemorrhages. With intracerebral hematoma, coma appears soon after development of headache, is usually deep, and is associated with a severe progressive neurological deficit.

Sequela of head injury
The hypertension sometimes caused by head injuries usually occurs with such obvious and severe trauma and such deep coma that it does not resemble the clinical picture of hypertensive encephalopathy. Therefore, differential diagnosis is not difficult. However, in a hypertensive patient, clinical identification of subdural hematoma may present a problem because headache, confusion, coma, and further blood pressure elevation can occur. Helpful points in differentiating subdural hematoma are the infrequency of convulsions, absence of severe hypertensive retinopathy, occasional focal neurological deficits, and ordinarily slower progression.

Other conditions
Other conditions that may need to be distinguished from hypertensive encephalopathy are the postictal state of epilepsy, cerebral vasculitis, encephalitis, brain tumor, and metabolic encephalopathies such as uremia. Ischemic and hemorrhagic strokes, however, are far more likely to present a picture suggesting hypertensive encephalopathy.

Initial Treatment
If arterial pressure can be reduced substantially before coma supervenes, all symptoms are relieved. The comatose patient responds slowly to the reduction of pressure, and several hours may elapse before coma begins to lessen.

This condition constitutes a medical emergency. If the patient is seen in a doctor's office, he should be sent to a hospital for admission, preferably to an ICU. The need for reducing arterial pressure is so urgent, however, that treatment should be started as soon as a presumptive diagnosis is established even in the physician's office or the emergency room. Response to treatment is, in fact, an essential feature of the diagnosis. Even if therapy in the emergency room establishes a diagnosis by bringing about the desired response, hospital admission is required for continued control of arterial pressure in order to prevent recurrence.

The goal of treatment is to reduce the arterial pressure as promptly as possible. The degree to which success is achieved relates directly to the energy and vigor with which therapy is pursued. In hospitals where physicians are available to administer intravenous and antihypertensive agents, and the nursing staff is trained to monitor arterial pressure carefully, hypertensive encephalopathy can be treated with diazoxide, sodium nitroprusside, or methaphan given intravenously in amounts carefully titrated to the blood pressure response. Table 1 provides a list of antihypertensive drugs for parenteral administration in this condition. If facilities and personnel for these ideal forms of treatment are unavailable, hydralazine, pentolinium or reserpine may be used intramuscularly. Since drugs in the latter group are not likely to produce rapid reduction in arterial pressure, several hours may elapse before improvement occurs.

When hypertension has been brought under control and the patient is sufficiently alert, treatment with oral antihypertensive drugs can be started. Because this type of hypertension is often resistant to therapy, use of the more potent agents, guanethidine and methyldopa, is advisable. The daily dose of guanethidine should be 25 mg the first day, increased by 25 mg each day until the arterial pressure (measured with the patient supine) is controlled at levels of 160/100 or below, or until the maximal dose of 100 mg is reached. Methyldopa is given four times daily. The amount given the first day is 250 mg; the next day, 250 mg and 500 mg are given alternately; and on the third day, 500 mg. To counteract the fluid-retaining effect of both drugs, standard doses of a chlorothiazide diuretic should be given. During the first day of oral drug treatment, parenteral medication should be continued to prevent recurrence of hypertension.

Complications
Arterial Hypertension. Autoregulation of the cerebral circulation is lost frequently in both ischemic and hemorrhagic stroke. As a result, CBF responds passively to arterial pressure, so that further ischemia occurs at reduced levels of arterial pressure ordinarily tolerated by the normal person. When arterial pressure is elevated in the absence of autoregulation, the volume of CBF rises above normal, causing or aggravating cerebral edema with resultant increased intracranial pressure. If the rise in intracranial pressure is marked, blood flow, which depends upon the gradient between arterial and intracranial pressure, may actually fall. Furthermore, intracranial hypertension may create or increase a tentorial pressure cone and thus produce brain stem compression by transtentorial herniation. These factors make difficult the selection of a proper arterial pressure at which to maintain the patient with ischemic stroke. Extremes of pressure (excessively high or excessively low) should be avoided. The desirability of treating hypertension in cases of subarachnoid and intracerebral hemorrhage is less controversial than it is in ischemic stroke.

The best regimen for treating arterial hypertension is that with which the clinician is most familiar, and which produces the most predictable response for him. One useful method is the administration of
hydralazine, 10 mg intramuscularly at hourly intervals. In about 20% of patients this program must be supplemented with reserpine, 0.5 mg intramuscularly every four hours; the nurse is instructed to withhold medication when the systolic pressure falls below an arbitrary level, e.g., 160 mm Hg in ischemic disease. When the use of hydralazine is undesirable (as in patients with myocardial ischemia), methyldopa hydrochloride, 250 mg intravenously every six hours, is a satisfactory substitute. A disadvantage of both methyldopa and reserpine in patients who may ultimately come to operation is the possible occurrence of hypertension during general anesthesia. The anesthesiologist can anticipate this complication if he has been informed that these medications have been used.

The important principle in treating hypertension is to achieve the desired effect by deliberate and controlled administration of antihypertensive drugs in divided doses at frequent intervals. A single dose usually is insufficient to cause much change, but the cumulative effect of several increments in a few hours will be adequate in most cases. During antihypertensive therapy continuous EKG monitoring, when available, helps to detect changes in cardiac rate or rhythm.

A marked and rapid reduction in arterial pressure should be avoided because it may precipitate myocardial infarction or aggravate cerebral ischemia. In some patients increased cerebrovascular resistance causes the brain to become ischemic at normotensive values and therefore the arterial pressure must be kept above normal levels in order to maintain adequate cerebral perfusion.

Cerebral Edema and Intracranial Hypertension. Regardless of the etiology of cerebral edema and the level of intracranial pressure, treatment is essentially the same. The ideal method is one that would control cerebral edema effectively and uniformly over a prolonged period; unfortunately, no single method is available which satisfactorily attains this objective.

Assessing the results of management in cerebral edema is difficult. If the patient who responds well to therapy directed specifically toward reduction of cerebral edema becomes worse when treatment is stopped, one can assume a cause-and-effect relationship. Several explanations are possible, however, if the patient does not respond well to treatment: (1) the treatment is effective in reducing edema and intracranial pressure, but the coma persists because they have already produced irreversible brain damage; (2) the coma is due to some cause other than increased intracranial pressure, for example, infarction of the brain stem; and (3) the therapeutic agent is effective, but was discontinued before achieving clinical improvement (see: Therapy of Increased Intracranial Pressure).

Adrenocorticosteroids

Glucocorticoids have been used extensively to treat brain edema from many causes. The mechanism of action, although poorly understood, is presumably related to regulation of electrolyte and water balance in the extracellular and intracellular spaces of the brain. Glucocorticoids have been most effective in reducing the edema that develops adjacent to a brain tumor. Their actions in stroke patients are not well documented, despite wide use in both hemorrhagic and thrombotic strokes. Dexamethasone is used most frequently, but other glucocorticoids may be equally efficacious. The usual dose of dexamethasone for the adult is 16 mg daily (4 mg every six hours). It may be administered to the comatose patient intramuscularly, intravenously, or through a feeding tube. The intravenous route is preferred because rapid passage into the brain is thereby ensured.

The principal short-term complication of steroid therapy is gastrointestinal bleeding; although the incidence of this difficulty is low, the administration of antiulcer medication through an indwelling nasogastric tube is recommended for comatose patients receiving glucocorticoids. Steroid therapy should be used with caution in patients with a history of peptic ulcer because of possible gastrointestinal bleeding. If treatment has lasted for 72 hours or less, it can be stopped completely without fear of adrenocortical insufficiency; after longer periods of administration the daily dose should be reduced gradually over a period of seven days.

Hypertonic Solutions

Intravenous administration of hypertonic agents reduces the fluid content of the brain. Because of the blood-brain barrier, these substances do not enter brain cells but readily absorb fluid from perivascular and pericellular spaces, leading to selective dehydration. The preparation used most frequently in the past was urea, but this drug has been replaced largely by mannitol because of complications such as hemoglobinuria, extensive sloughing of the skin following accidental extravasation, and a significant rebound in intracranial pressure when the urea infusion is discontinued.

Mannitol in concentrations of 10% to 25% is administered intravenously in a dose of 1 gm per kilogram of body weight. This amount can be given within a space of ten minutes if necessary. When mannitol is effective, intracranial pressure begins to fall in a few minutes, and maximal reduction is achieved within 30 to 60 minutes. The disadvantage...
MEDICAL AND SURGICAL MANAGEMENT

TIA AND STENOTIC LESIONS OF THE NECK VESSELS

Medical Treatment

*"Episodes of TIA are recognized widely as precursors of ischemic stroke, since more than 35% of affected patients are likely to have a cerebral infarct within the next five years. This is more than ten times the rate of expected stroke incidence for persons of the same age and sex in a general population.

"Some physicians believe that long-term anticoagulant treatment with coumarin drugs in patients with TIA will prevent stroke in a high percentage of cases. However, others think that the possibility of serious hemorrhage from protracted use of oral anticoagulants outweighs the potential advantages, or that the anticoagulant drugs may be ineffective.

"Bleeding of any kind is the primary danger of oral anticoagulants, a risk which appears small if good laboratory facilities are available to keep the prothrombin time in a safe and therapeutic range, usually about twice the control value in seconds (see: Section on Laboratory Evaluation of Neurovascular Disease [Stroke]: One-Stage Prothrombin Time. Stroke 3: 510 [July-Aug] 1972). Recent reports suggest that drugs such as aspirin and clofibrate inhibit the aggregation of platelets and also may prevent TIA. Although these agents probably carry less risk than coumarin compounds or similar anticoagulant drugs, proof of their clinical efficacy awaits the results of current studies. While numerous other medical treatments, including presumed cerebral vasodilators, have been advocated for patients with TIA, there are no supporting data that any of these consistently prevent strokes.

"When TIA are associated with angiographic evidence of severe stenosis of the cervical portion of the appropriate internal carotid artery or with an ulcerated plaque in a similar location, carotid endarterectomy has been judged beneficial in preventing strokes. There is less published evidence that patients with bilateral internal carotid stenosis, or stenosis on one side and occlusion on the other, are aided by such procedures because of increased surgical risk. Carotid surgical procedures must be carried out in a facility where the operative team is experienced in surgery on small arteries, familiar with surgical hazards, and capable of managing any ensuing complications. Opinions vary as to whether surgery is indicated for patients with more than two stenoses or occlusions in the cervical region.

"In persons with TIA who have no physical findings, for example, a localized bruit in the neck

Frequently multiple lesions in the cerebrovascular system may alter the demand for flow in the remaining vessels. Thus, when one carotid artery is completely occluded, stenosis of only 50% in the opposite carotid may have clinical significance. Furthermore, a tendency to underestimate the degree of stenosis as visualized on arteriograms is denoted by the observation that the amount of narrowing seen at the time of endarterectomy usually exceeds that estimated from films.

Emboli originating from ulcerated plaques or in regions of stasis or turbulence distal to stenotic areas may produce symptoms of obstruction. Thus a nonobstructing ulcerated plaque may have considerable significance as a source of emboli. When irregular or ulcerated atheromatous plaques are present or the patient's symptoms suggest embolization, many consider the existence of an accessible lesion as indication for operation.

Basic Evaluation for Operation

Laboratory Tests

The routine laboratory tests of hematocrit, leukocyte and differential cell count, blood urea nitrogen, and creatinine are desirable for any patient about to undergo a major surgical procedure. In those with cerebrovascular disease the following additional studies are needed: two-hour postprandial blood sugar, cholesterol, prothrombin time, partial thromboplastin time, and serological tests for syphilis.

Other Special Procedures

Patients who are candidates for arteriography and/or surgery should have skull and cervical spine films, chest films, and an EKG.

Arteriography

The surgeon has little to gain and much to lose if he consents to operate upon a patient who has less than a complete arteriographic workup. Approximately 2% to 5% of patients whose symptoms strongly suggest ischemia are found instead to have tumors (metastatic or primary), aneurysms, or vascular malformations. Vascular disease is common in older patients and the finding of an obstruction does not necessarily implicate it as the cause of symptoms. However, lesions are often multiple, and are found in several arteries, in which case the one most likely to be responsible for the clinical symptoms and most favorably located for surgical approach is selected for endarterectomy.

For detailed information regarding angiographic study in the selection of patients for arterial reconstructive surgery, refer to the Section on Special Procedures and Equipment in the Diagnosis and Management of Stroke (Stroke 4: 111-137 [Jan-Feb 1973]).

Contraindications to Operation

Acute Neurological Deficit

Many surgeons have reported a high incidence of complications when vascular operations were undertaken in the first few days after the development of an acute deficit. Some surgeons now defer operation for a minimum of two weeks following an acute stroke, and some believe that endarterectomy should be postponed for five to six weeks. Many authorities feel that surgery has little to offer in a completed stroke.
Occlusion showed mortality in patients with an acute neurological deficit to be twice as great among those treated surgically as among those managed medically. When operation was postponed 14 or more days following the stroke, surgical mortality was reduced greatly. A recommendation emerged from the study that operation should be deferred for a minimum of two weeks following the development of acute neurological deficit.

Occlusion of the Internal Carotid Artery
The question of whether to operate on patients with occlusion of an internal carotid artery is controversial. When an acute carotid occlusion is associated with a corresponding neurological deficit, deferment of operation is recommended, since revascularization in a segment of softened brain may result in intracranial hemorrhage and death. Yet, the optimal period for successful mechanical removal of a clot from the internal carotid artery would seem to be within the first 24 hours following thrombosis. After this time the clot may become firmly adherent to the internal carotid artery up to the base of the brain and cannot be removed readily. Although special techniques may sometimes be successful in reopening a chronically occluded internal carotid, the anticipated risk of these heroic measures appears to exceed any likely benefit.

Occlusion of the Vertebral Artery
This is generally considered to be an inoperable lesion. Since no collateral channels exist proximal to the entrance of the vertebral artery into the lateral mass of the sixth cervical vertebra, a patent, accessible segment in that portion of the neck proximal to the vertebral canal is impossible to find.

The only widely accepted indication for vertebral endarterectomy occurs in patients with symptoms of vertebral-basilar ischemia who have bilateral vertebral artery involvement. In this circumstance operation on one side may restore adequate blood flow. Unilateral vertebral artery stenosis is tolerated well and, even when progressing to occlusion, does not lead to stroke.

Concomitant Myocardial or Pulmonary Disease
Operations on cervical vessels are rarely accompanied by serious complications other than neurological. However, the operative risk of thoracotomy for surgical management of aortic arch lesions is significantly high, especially in patients with myocardial or pulmonary disease. Most lesions at this level can be treated successfully by extrathoracic crossover grafts, such as carotid subclavian bypass, thereby avoiding the risk of thoracotomy.

Anesthesia Management. Anesthesia for angiography is discussed in another section (see: Section on Special Procedures and Equipment in the Diagnosis and Management of Stroke. Stroke 4: 111-137 [Jan-Feb 1973]. The advantages and disadvantages of local versus general anesthesia are discussed subsequently.

Cerebral Monitoring and Protection. The collateral blood supply to the brain is adequate in most patients to prevent cerebral injury during the arterial occlusive phase of a reconstructive operation. The problem of cerebral protection is limited largely to surgery carried out at the carotid bifurcation. Many surgeons use routine internal shunting to avoid ischemic complications; others believe that operations can be performed effectively without the encumbrance of a shunt if special techniques are utilized to improve collateral flow. This requires a means for monitoring either CBF or cerebral function.

Monitoring
Local anesthesia
Some surgeons advocate the use of local anesthesia so that they can ask the patient to respond verbally or to move an extremity as assurance that functional integrity of the cerebrum is maintained during test occlusion of carotid arteries and during the operative procedure. An internal shunt is used for those patients unable to tolerate trial carotid occlusion. This method has the practical disadvantage of patient discomfort; most surgeons prefer general anesthesia, a technique which has an added advantage of decreased cerebral metabolic requirements for blood flow.

EEG
Continuous EEG during trial carotid occlusion has been used extensively and opinions vary as to its utility. The equipment is delicate, is cumbersome in the operating room setting, and requires alert and informed neurological personnel.

Cerebral venous O₂ saturation
Maintenance of the O₂ content is an added safety feature. Blood gas analyzers available in most hospitals can provide this information when the analyzer is in the operating room and is used by an interested, proficient technician. Because of the anatomical vagaries of cerebral venous drainage, this technique usually monitors total CBF rather than flow from the critical cerebral hemisphere.

Internal carotid back pressure
Many surgeons rely upon measurement of the back pressure in the internal carotid artery during trial occlusion of the common and external carotid arteries. Essentially this is a measurement of collateral blood flow to the ipsilateral hemisphere and its use is based upon the assumption that adequate cerebral protection is present when the
carotid back pressure exceeds a critical level. Clinical experience suggests that when the mean pressure is higher than 50 mm Hg, cerebral protection is adequate for the 15 to 30 minutes usually required to complete the endarterectomy.

**Protection**

*Internal shunts*

The use of shunts during the occlusive phase of operation becomes necessary when clinical observations indicate inadequacy of collateral blood flow to the ipsilateral hemisphere.

**Hypothermia**

The rationale for using hypothermia is that it lowers cerebral metabolism and prolongs tolerance of cerebral ischemia. When this method is used, the patient should be cooled to a temperature of 31 to 32 C. The failure of hypothermia to afford absolute protection from cerebral anoxia, and the hazard of inducing cardiac instability (ventricular fibrillation), as well as other complications, have led most surgeons to abandon this technique.

**Hypercapnia and hypocapnia**

Total CBF is known to increase with elevation of arterial P CO2. This can be accomplished by modification of the anesthesia technique, and many surgeons employ it on the assumption that blood flow to the ipsilateral hemisphere is increased during carotid endarterectomy. Some believe, however, that vasodilatation in the contralateral hemisphere "steals" blood from the area where it is most needed and have proposed that greater protection is created by depression of P CO2 levels. Both methods have been used and data at this time do not permit a firm conclusion as to the efficacy of one method over the other.

**Hypertension**

Carefully controlled hypertension increases blood flow to the ipsilateral hemisphere during carotid endarterectomy. Many surgeons have found it to be the most effective method for improving cerebral protection. Some have suggested that its use is monitored best by means of an indwelling radial artery catheter with continuous oscilloscopic recording.

**Surgical Techniques.** Far more important than the method selected for a cerebrovascular operation is the skill of the surgeon who performs it and his familiarity with the proposed technique. Bypass procedures may carry low mortality risk in the hands of one surgeon who deals with lesions at the aortic arch, whereas endarterectomy may be the method preferred by another.

Endarterectomy takes advantage of the cleavage plane created by arteriosclerosis in the innermost layer of the arterial media. Most occlusive lesions at the bifurcation of the common carotid or at the origin of the vertebral arteries, and some localized lesions in the subclavian, innominate, and carotid vessels at the level of the aortic arch can be cleared successfully by endarterectomy. Removal of the thickened intima inferior to the cleavage plane leaves an artery with a smooth inner surface and a lumen slightly larger than normal.

When endarterectomy is considered hazardous or impossible (as in some arteriosclerotic occlusive lesions or when it is desirable to avoid major intrathoracic procedures), revascularization using bypass grafts may be necessary.

Synthetic grafts or autogenous vein grafts are used from the aorta to a distal patent arterial segment to bypass these occlusions. For patients with symptomatic lesions in the proximal brachiocephalic arteries, an alternate technique which has become increasingly popular because of less risk is the use of cervical bypass grafts (usually the saphenous vein), one end of which is anastomosed to an undiseased cervical artery and the other to the patent arterial segment distal to the level of occlusion.

**General Principles**

When arteriography discloses a single lesion which is surgically accessible and presumed to be the source of the patient’s symptoms, the choice of operation is relatively simple. Frequently, however, arteriography shows multiple lesions with varying degrees of arterial obstruction. Under these circumstances, selection of the appropriate operation is based upon principles to be defined below.

**Selection of optimal lesion for surgery**

In a patient with obstructing lesions in both the carotid and vertebral systems, whose complaints are attributable to vertebral-basilar insufficiency, restoration of normal carotid patency has first priority. Collateral channels in the circle of Willis may increase basilar artery blood flow sufficiently to relieve symptoms in some of these patients. Operation on the subclavian or vertebral artery lesions is indicated only in the rare patient with persistent symptoms.

When the patient with combined lesions has symptoms limited to the carotid territory, there is no indication for operation on the vertebral arteries after successful carotid endarterectomy. On the other hand, if symptoms are believed due to embolism from an ulcerated plaque, the lesion which appears to be the source of emboli (usually located at the carotid bifurcation) should be treated initially, if possible.

Most surgeons consider stenosis or ulceration at the carotid bifurcation to be technically the most favorable for removal; lesions at the level of the aortic arch are next. The least favorable are those of
the vertebral arteries, not only because of the technical problems involved, but also because the vessels are so small that even successful operations do not result in notably increased intracranial blood flow.

Sequence of operations
When multiple lesions exist and several procedures seem indicated, the following guidelines have been accepted by many surgeons.

Tandem lesions, surgically accessible
When two lesions exist sequentially in the same vascular system (e.g., stenosis of the left common carotid at the aortic arch, and of the left internal carotid), the more proximal of the two should be removed first, thereby ensuring a high rate of flow following the second operation and reducing the likelihood of postoperative thrombosis, which is more prone to occur in the smaller vessel.

Bilateral carotid lesions
Lesions often occur simultaneously at both carotid bifurcations. If symptoms are limited to the cerebral artery supplied by one of the two vessels, this side (the symptomatic side) should be operated upon first. When one arterial lumen is considerably smaller than the other, and symptoms are not specific for one vessel, the more significant stenosis should be removed first, since temporary occlusion of that vessel is less apt to decrease overall cerebral flow.

If the carotid lesions appear about equal, and symptoms are not specific for one artery, the vessel supplying the nondominant hemisphere should be operated upon first.

Accessible and inaccessible lesions occurring in tandem
Occasionally intracranial stenosis of one internal carotid occurs simultaneously with a lesion at the origin of the same vessel. When no collateral bed separates two obstructions within the same vessel and both stenoses exceed 70% occlusion, flow will be determined by the one which narrows the lumen to the greater degree. If this lesion is at the carotid bifurcation, successful removal may improve CBF. If the more significant stenosis is within the cranium, removal of the proximal carotid lesion is rarely, if ever, indicated.

Timing of multiple operations
Most surgeons have found that simultaneous procedures on two vessels are associated with an excessively high incidence of complications. For this reason, both carotid bifurcations are rarely operated upon at the same time. Most surgeons prefer to delay the second operation by at least a week.

MEDICAL AND SURGICAL MANAGEMENT

Specific Operative Procedures
Details of all standard operative techniques are available to the vascular surgeon. In the National Cooperative Study of Extracranial Vascular Disease, certain trends in the utilization of specific procedures have been noted.

Aortic arch lesions
For many years endarterectomy at the origins of the cerebral vessels or bypass grafts from the aorta to the unobstructed extrathoracic vessels, both of which involved thoracotomy, were the most widely used procedures. The operations had the disadvantages associated with thoracotomy. In recent years most vascular surgeons have preferred to carry out local bypass operations in the neck, using a vessel with no proximal obstruction as the donor for one or more proximally obstructed arteries. The bypass graft can be done with an autogenous saphenous vein or a Dacron prosthesis.

Carotid bifurcation lesions
The standard operation used now by nearly all vascular surgeons consists of endarterectomy with or without patch angioplasty. A few surgeons perform transverse arteriotomies for endarterectomy. The remainder use a generous longitudinal arteriotomy, which may be closed either primarily or with an autogenous vein graft patch (saphenous or external jugular for example), or a patch graft of prosthetic material such as Dacron. Some surgeons conclude the operation with angiography to assure an adequate technical result. An optimal outcome of the procedure is the rule and later complications are negligible if angiography discloses a completely patent system.

Vertebral artery lesions
Lesions which narrow the first portion of the vertebral artery are frequently due to the extension of plaques in the subclavian artery to encroach upon the vertebral orifice. Whether primary in the subclavian or in the vertebral artery, endarterectomy is the procedure of choice if the subclavian artery at the level of origin of the vertebral artery is accessible from the neck. Endarterectomy of the vertebral artery can be carried out through the subclavian artery or by vertebral arteriotomy. If the latter method is chosen, autogenous vein patch of the arteriotomy is often necessary. Because the vertebral arteries are small, in only rare cases is bypass grafting technically possible. The mediastinal origin, when the left vertebral artery has its origin deep in the mediastinum, has prompted some surgeons to detach this artery from its origin and anastomose it to the side of the adjacent subclavian or common carotid artery.
Intracranial arteries
Although direct operations have been carried out for bypass procedures or to remove occlusive lesions within the skull, these techniques should be considered experimental at the present time.

Complications of Carotid Endarterectomy
Related to General Anesthesia
Cardiac problems
Because cardiac arrhythmia or cardiac arrest occasionally occurs, patients must be monitored constantly during operation as to pulse, blood pressure (preferably by a radial artery catheter), and EKG. Appropriate provision must be made by the anesthesiologist or a cardiac consultant if necessary to handle any of these cardiac problems.

Airway problems
When general anesthesia is used for the operation, intubation is routinely performed and the patient can be monitored closely. Secretions must be aspirated, and members of the operating team must take care not to obstruct the airway.

Hypotension
Hypotension, which occurs frequently during operation and in the early postoperative period, may be caused by overmedication, deep anesthesia, or manipulation of the carotid sinus during dissection. Postoperatively it occurs more commonly after carotid operations than after operations elsewhere in the body. Low blood pressure is particularly hazardous if there are residual lesions in other arteries supplying the brain. The following precautions should be observed:
1. Minimal or no preoperative sedation,
2. Careful control of the depth of anesthesia,
3. Intravenous infusion of dilute solutions of vasopressors such as phenylephrine hydrochloride (Neo-synephrine) or metaraminol bitartrate (Aramine) given during and after operation to keep blood pressure at the level appropriate for the individual patient,
4. Infiltration of the carotid sinus with xylocaine if this area is to be dissected.

Related to Cervical Wound
Infection
Infection may jeopardize the integrity of the arterial suture line and all customary precautions should be undertaken to prevent its occurrence. The value of prophylactic antibiotic therapy is controversial.

Hematoma
This complication may be the result of anticoagulant therapy, of inadequate hemostasis, or of leakage at the suture line postoperatively. If tracheal obstruction due to laryngeal edema results from a hematoma, early reoperation is required and tracheostomy may be necessary.

Nerve paresis
Damage to the hypoglossal, vagus, recurrent or superior laryngeal nerves, or the marginal branch of the facial nerve may lead to paresis and usually may be prevented only by careful operative dissection. Before operation on a second carotid artery is carried out, it is imperative that vocal cord function be assessed to rule out unsuspected tenth nerve injury from the first operation.

Parotitis
This complication may result from excessive manipulation or dissection of the inferior portion of the parotid gland.

Airway obstruction
The airway may become obstructed by excess secretions (most often from prolonged endotracheal intubation), tracheal pressure from hematomas of the neck, bilateral carotid endarterectomy in one stage, or damage to the vagus or recurrent laryngeal nerves. The frequency with which this complication occurs is decreased by the use of careful surgical technique and avoidance of excessive anticoagulant therapy. Tracheostomy, if indicated, must be performed promptly.

Related to the Carotid Artery
Postoperative disruption of the artery
Serious complications may be caused by improper suturing of the arteriotomy, or by excessively high blood pressure in patients with very thin-walled vessels. Careful endarterectomy and meticulous suturing of the arteriotomy will usually prevent this catastrophe.

False aneurysm at the endarterectomy site
Use of the improper plane of cleavage for endarterectomy may be followed by postoperative disruption of the artery. The commonest cause of false aneurysms after endarterectomy, however, is infection or the use of silk sutures to sew Dacron patch grafts in place. If Dacron patches must be used, they should be sewn on with Dacron sutures.

Infection in Dacron patch grafts
This serious complication results from contamination of the wound at the time of operation or upon removal of a hematoma. It must be treated with massive antibiotic therapy, both parenteral and local, and the infected Dacron patch graft must be removed and replaced with a vein graft. Frequently total replacement of the inflamed friable artery is required.
Intraoperative Causes

Embolism from necrotic plaque or aggregation of platelets

Manipulation or excessive palpation of the artery may dislodge debris which produces embolism. To reduce this possibility, gentle handling is essential.

Cerebral ischemia

Possible causes of this complication are: (1) hypotension, (2) inadequate cerebral protection during operation, (3) inadequate flushing of the artery, so that following restoration of flow embolic debris is carried into the brain or arterial thrombosis occurs in intracerebral vessels distal to the site of the endarterectomy, and (4) postoperative arterial thrombosis.

Steps that can be taken to prevent these complications are: (1) gentle manipulation of the artery, (2) use of xylocaine in the carotid sinus area to prevent bradycardia and reflex hypotension, (3) use of adequate fluids and vasopressors during operation, (4) appropriate measures to ensure that the patient's brain is receiving sufficient blood at all times, and (5) use of heparin in appropriate doses to prevent arterial thrombosis in clamped-off vessels.

Postoperative Causes

Intimal dissection leading to thrombosis of the carotid artery reconstruction

The hope for preventing this complication lies in operative technique. If a plaque remains in the internal carotid above the site of endarterectomy, either the intima should be attached securely with interrupted sutures before the arterial incision is closed, or, preferably, the endarterectomy should be extended to the distal extent of the diseased vessel.

Arterial thrombosis for other technical reasons

If the internal carotid is extremely small, inappropriate suturing may lead to arterial thrombosis. With small arteries, patch grafts may be necessary.

Hypotension

This may result postoperatively from inadequate fluids, from overmedication, or from congestive heart failure, tachycardia, bradycardia, acute myocardial infarction, other cardiac causes, or as a physiological response to operation at the carotid bulb. Hypotension must be avoided for the first day or two following operation. Some surgeons prefer continuous monitoring in an ICU by means of an arterial line during this time.

Intracerebral hemorrhage or edema

This condition may develop if (1) hypertension occurs in the immediate postoperative period, or (2) operation during the acute phase of stroke results in restoration of flow through an infarcted area, particularly if a totally occluded carotid artery is opened. That an ischemic infarct will become hemorrhagic under these conditions is a danger previously described.

Headache

Severe headache is common following endarterectomy of a stenosed artery and is probably due to vascular distension. It is treated by means of analgesics such as aspirin or codeine.

Postoperative Management

After operation the patient should remain in the recovery room until consciousness has returned and respirations are satisfactory. He should then be moved to an ICU. Careful, continuous monitoring of the central venous pressure, arterial blood pressure, EKG, arterial blood gases and pH, and correction of any abnormalities are important, particularly in patients with chronic obstructive lung disease.

Medications

Anticoagulants

(See: Use of Specific Medicinal Agents in the Treatment of Ischemic Cerebrovascular Episodes, Anticoagulants.) Most patients undergoing surgical procedures do not receive anticoagulant therapy in the immediate postoperative period. If anticoagulant therapy is indicated, it is begun usually after the patient's condition is stabilized and he has returned to his room.

Antihypertensive drugs

(See: Hypertensive Encephalopathy, Initial Treatment.) Experience with large numbers of patients indicates that approximately 40% of those undergoing surgical therapy for extracranial vascular disease have significant hypertension. In most instances, antihypertensive drugs are not required during the immediate postoperative period but are reserved for later use if needed. When very high levels of blood pressure are recorded, however, antihypertensive therapy is indicated.

Acceptable Surgical Morbidity and Mortality

The commonest complication in all reported cases has been a postoperative neurological deficit. Its frequency has been reduced materially by avoiding operation on patients with a recent cerebral infarct, improvement in surgical technique, employment of means to enhance CBF during operation, refinement of anesthetic techniques, and scrupulous monitoring of blood pressure during and immediately following operation. In experienced hands, the incidence of permanent neurological deficit postoperatively is now less than 2%.

Management of Patients with Recurrent Symptoms of Cerebrovascular Insufficiency

The recurrence of symptoms or signs indicating cerebrovascular insufficiency in a patient previously
operated upon calls for another complete evaluation, including four-vessel arteriography.

**Evolving Infarction and Stabilized Infarction**

The first principle in treatment of cerebral ischemic disease is to rule out disorders which mimic this condition (see: Differential Diagnosis). The initial evaluation of such patients is described previously (see: Initial Evaluation).

**Diagnostic Procedures**

The laboratory workup has been outlined in the Section on the Laboratory Evaluation of Neurovascular Disease (Stroke). Stroke 3:503-526 [July-Aug 1972]. Further studies that may be extremely valuable include radioisotope angiography, lumbar puncture, brain scan, ODM, EEG, echoencephalography, and long-term EKG monitoring (see: Section on Special Procedures and Equipment in the Diagnosis and Management of Stroke. Stroke 4: 111-137 [Jan-Feb 1973]).

**Initial Management**

In evolving infarction, anticoagulant therapy should be considered (see: Anticoagulants). The prothrombin and/or clotting times should be followed, and the patient watched carefully for evidence of bleeding in other parts of the body.

The use of vasodilators (such as CO₂ or spasmylocytic drugs) to produce increased blood flow, and adrenocorticosteroids to reduce edema are discussed subsequently (see: Specific Medicinal Agents in the Treatment of Ischemic Cerebrovascular Episodes).


Rehabilitative measures should be instituted as early as deemed advisable by the patient's physician and/or surgeon (see: Section on Stroke Rehabilitation. Stroke 3:375-407 [May-June] 1972).

**Identification and Control of Precipitating or Complicating Factors**


Proper management of the patient with cerebral infarction requires diligent search for and, if possible, correction or control of factors which may have precipitated the attack in order to avoid subsequent strokes so far as possible. Continued vigilance for the recognition of complications is essential.

**Heart Disease.** Heart disease is two to three times as frequent in patients who have had strokes as in the general population. In some, cerebral infarction is associated with myocardial infarction, heart failure, and/or cardiac arrhythmia, which produce decreased cardiac output and secondary cerebral ischemia, as well as emboli. Prolonged EKG monitoring may be required to diagnose the presence of intermittent abnormalities such as dysrhythmia or a conduction defect. When an abnormality is found, consultation should be sought. Treatment of conduction defect and dysrhythmia is beyond the scope of this report, but it is important to note that many episodic disorders attributed to cerebrovascular ischemia are in fact due to cardiac dysrhythmia, control of which will cause the cerebral episodes to cease (see: Section on Clinical Prevention of Stroke. Stroke 3:803-825 [Nov-Dec 1972]).

Serial measurements of SGOT and LDH are also useful in cases of probable or possible myocardial infarction and should be extended over a period of three to five days. If such infarction is diagnosed, therapy for this condition should take precedence over subsequent active physical therapy, although safe compromises usually can be achieved.

Not unusually, stroke patients have occult or overt heart failure early in the course of illness and appropriate therapy should be instituted at once. Care must be taken not to overburden the heart with excessive fluids. The treatment of embolization and of cardiac arrhythmia is discussed in the Section on Clinical Prevention of Stroke (Stroke 3:803-825 [Nov-Dec 1972]).

**Hyperlipidemias.** Some population studies show abnormally high serum lipid values in young patients with nonembolic infarction of the brain (see: Section on Epidemiology for Stroke Facilities Planning. Stroke 3:351-371 [May-June] 1972). The role of hyperlipidemia in the immediate production of cerebral infarction is not known, nor is effectiveness of dietary or drug manipulation certain in reducing the risk of stroke in persons with hyperlipidemia.

**Polycythemia and Erythrocytosis, Including Sickle Cell Anemia.** Polycythemia alters the rheological characteristics of the blood and is well recognized as a factor predisposing to cerebral infarction. The determination as to whether a polycythemia is primary or secondary to dehydration or chronic pulmonary disease is crucial to proper therapy.

Primary polycythemia can be treated by phlebotomy. The exact hematocrit level at which treatment should be initiated and the rapidity with which the hematocrit should be reduced are debatable. The production of hypotension must be avoided because of the associated risk of cerebral ischemia. Seemingly paradoxical hemorrhagic disturbances sometimes occur in patients with polycythemia.

Sickle cell anemia should be identified and precipitating factors should be avoided.
MEDICAL AND SURGICAL MANAGEMENT

Hypovolemia. Depletion of blood volume by dehydration or blood loss may occasionally precipitate stroke in patients predisposed to cerebral ischemia. If the patient can sit or stand, postural tachycardia or hypotension may provide important diagnostic clues. Blood may be lost from many sources; gastrointestinal bleeding can be detected by stool examination.

Diabetes Mellitus. Diabetes often is a serious complication in the management of stroke patients. Because of the well-recognized high incidence of associated diffuse atherosclerotic vascular disease, diabetes mellitus is an important clue to the diagnosis of nonembolic infarction. The usual therapy of diabetes mellitus probably does not contribute appreciably to the improvement of associated vascular disease; nevertheless, regulation of blood sugar level in the diabetic stroke patient is important for many other reasons, especially to diminish the frequency of infections. Hypoglycemic agents must be used with caution, however, since hypoglycemia has undesirable effects on the brain. Attempts to achieve a near normoglycemic state are desirable.

Hypertension. At least 50% of stroke patients have antecedent hypertension. Evidence is emerging that control of elevated diastolic pressure by drugs reduces the risk of eventual stroke. Control of systolic hypertension may prove to be equally important, but present evidence is inconclusive. Overly energetic antihypertensive therapy with profound reduction in cerebral perfusion can precipitate cerebral ischemia, but this complication is so unusual that its possibility should not deter the physician from instituting adequate treatment measures.

Decreased O₂ Transport. Anemia and hypoxia can produce cerebral infarction by decreasing the amount of O₂ available to the brain, and a combination of the two is particularly dangerous. Although any of the following complications may lower the oxygen content of blood and none is always completely preventable, early detection and treatment may decrease appreciably the associated morbidity and mortality.

Asphyxia

Unfortunately asphyxia is all too frequent following stroke, even after hospitalization. Stroke victims who are stuporous or unconscious or who have pharyngeal paralysis are subject to obstruction of the upper airway. If they are permitted to lie supine with the head elevated by one or more pillows, the tongue obstructs respiration and saliva can be aspirated. To allow drainage from the mouth the patient should be placed on his side with the neck extended and the head somewhat dependent. In some cases an oropharyngeal airway is required to prevent obstruction by the tongue. Tracheostomy may be necessary to maintain a patent airway, particularly in patients with depressed consciousness and/or brain stem dysfunction. Experience shows that asphyxia occurs frequently during transport of unconscious or paralyzed patients by ambulance or hospital cart.

Aspiration Pneumonia

Pneumonia may be a consequence of aspirating mouth and nasopharyngeal substances, food, or gastric contents into the respiratory tract. Vomitus will usually drain from the mouth if the unconscious patient has been positioned properly. Since vomiting may occur in the early stages of stroke, deliberate emptying of the stomach with a nasogastric tube should be considered. Oral feeding and gavage should be avoided until the risk of aspiration has passed.

Hypoventilation

Neural control of respiration often is disturbed in stroke patients. The possibility of CO₂ retention and increased PaCO₂ should be anticipated, and blood gases measured at frequent intervals in the unconscious or stuporous patient. Patients with chronic obstructive lung disease are particularly prone to respiratory insufficiency when a stroke affects consciousness. Hypoxia may be treated by the administration of low concentration of oxygen (1 to 2 liters per minute), given either by Venturi mask, by nasal cannula, or by catheter. If CO₂ retention is a problem, orotracheal intubation or tracheostomy with assisted or controlled ventilation may be indicated. Endotracheal tubes should not be left in place longer than three to five days. Cuffed endotracheal tubes must be deflated for at least ten minutes every hour. Instead of using arbitrary Prins levels to establish the need for ventilatory aid, the physician must view the total acid-base situation and intervene only if acidosis is developing. If CO₂ retention develops suddenly, it may produce respiratory acidosis of such severity that ventilatory aids are necessary. However, when it develops slowly, PₐCO₂ values as high as 60 to 70 mm Hg may be tolerated well with little or no acidosis and no need for mechanical ventilation. Measurements of blood pH are therefore necessary.

In the early phases of treatment the need for tracheostomy usually can be avoided by proper positioning and the use of an oral airway. Orotracheal or nasotracheal intubation can be used to give satisfactory access to the lower airway for periods as long as three to five days. Intubation, a skill which can be acquired readily with the help of an anesthesiologist, requires much less time than
Atelectasis With or Without Infection

Lack of physical activity, dehydration, infrequent turning, ineffective cough, and aspiration of pharyngeal material are common causes of atelectasis. Frequent turning, induced coughing, and deep breathing may be sufficient to prevent this complication. Atelectasis can usually be diagnosed on the basis of the physical and x-ray findings, and the diagnosis is further suggested by the detection of hypoxemia without CO\(_2\) retention. Therapy consists of intermittent positive pressure breathing to inflate the lung, bronchial aspiration, or bronchoscopy to remove plugs of inspissated secretions. Sputum examination for evaluation of the polymorphonuclear cellular reaction and gram stains to detect predominance of specific microorganisms are important in reaching a decision with respect to using antibiotics. Transtracheal aspiration is a safe, effective means of obtaining sputum samples from comatose patients. Frequent chest examinations and temperature recording are necessary in order to detect infection and to institute appropriate therapy.

There is no real evidence that the prophylactic use of antibiotics in the unconscious patient performs a function other than to eliminate the less-resistant organisms and provide a milieu for more refractory pathogens to take residence. Frequent gross and microscopic examination of the sputum should be performed as an aid to determining when to use an antibiotic and which one to employ.

Pulmonary Embolism

The exact incidence of pulmonary embolism is unknown, but the condition is not uncommon. All bedridden patients are at great risk of manifest or occult phlebitis and subsequent pulmonary embolism. Passive physical therapy is useful as a measure in preventing the venous thrombosis which may occur in the paralyzed or weak leg without the usual warning signs. Properly fitting elastic stockings help in preventing the venous stasis which encourages thrombosis. Venous thrombosis, as well as pulmonary embolism, requires anticoagulant therapy unless there is some antecedent bleeding problem or a risk of hemorrhage.

Specific Medicinal Agents in the Treatment of Ischemic Cerebrovascular Episodes

Three classes of drugs have been used extensively in the treatment of cerebral infarction: (1) vasodilators, (2) vasopressors and depressors, and (3) anticoagulants. A fourth group of miscellaneous agents including hyperbaric \(O_2\), low-molecular-weight dextran, and estrogens will be discussed briefly.

Vasodilators. The most potent of these agents is \(CO_2\). Although inhalation of a mixture of 5% \(CO_2\) with 95% \(O_2\) has been used extensively in the treatment of acute cerebral infarction, doubts have been cast on the effectiveness of this therapy. Five percent \(CO_2\) inhalation usually causes a brisk rise in cardiac work, cardiac output, and systemic and pulmonary arterial pressure, and therefore may be contraindicated in patients with coronary artery heart disease, congestive heart failure, and significant systemic and pulmonary arterial hypertension. Cardiac arrhythmias may occur occasionally during or following \(CO_2\) inhalation.

Papaverine may increase CBF in normal people, but its effectiveness in patients with atherosclerotic arteries is debatable. Any decrease in CBF produced by the drug in patients with atherosclerosis can be explained by the predominance of its effect on splanchnic and peripheral vessels. Many other pharmacological agents have been recommended for therapy of acute cerebral infarction and for prevention of symptoms in long-standing ischemic cerebrovascular disease, but no adequately controlled studies have been published attesting to their clinical value.

In view of the meagerness of evidence that treatment with any known vasodilator is beneficial to patients with acute ischemic stroke, the routine use of these agents in the treatment of evolving cerebral infarction cannot be recommended at present. Furthermore, some evidence indicates that the use of cerebral vasodilators may bring about changes in blood flow which steal blood away from the hyperemic zone surrounding the lesion.

Vasopressors and Depressors. Although ischemic stroke is a complication of long-standing hypertension, the onset of an ischemic cerebrovascular lesion is not necessarily related to the blood pressure either at that moment or when the patient is examined. Sustained severe hypertension, however, may increase morbidity from the stroke, and for this reason some authorities advise the use of vasodepressor agents if systolic or mean blood pressure is extremely high. Trimethaphan or sodium nitroprusside is best for this purpose, because these drugs allow precise control of pressure at desired levels. The blood pressure should be monitored carefully to prevent hypotension. In severely hypertensive patients, the systolic pressure should not be allowed to fall below 160 mm Hg. Methyldopa hydrochloride can be given intravenously in doses of 250 to 500
mg at six-hour intervals as required. In less urgent situations, if intravenous medications are too potent, or if depressant effects are of no concern, reserpine can be given intermittently until the desired reduction in arterial pressure is achieved. Some physicians give hydralazine intramuscularly, 10 mg per hour, to reduce arterial pressure. If the patient can take medications orally, treatment with alpha methyldopa (250 mg q.i.d.) and a chlorothiazide preparation can be started immediately.

Anticoagulants. In terms of subsequent thromboembolic episodes in the brain or elsewhere, wide differences of opinion exist concerning the value of anticoagulant therapy after ischemic infarction. Several controlled studies have been carried out in an attempt to elucidate different aspects of the problem, but results have been inconclusive. Use of anticoagulants has been shown to increase the frequency of hemorrhagic complications, particularly in hypertensive patients, and there is uncertainty as to whether these agents are effective in diminishing the risk of subsequent cerebral infarction. If anticoagulants are employed, the prothrombin and/or clotting times should be followed regularly, and the patient should be watched carefully for evidence of bleeding in any portion of the body. Intracranial bleeding due to anticoagulant therapy is manifested by deterioration of neurological status.

Strokes-in-Evolution
In gradually progressive cerebral infarction, some opinion favors immediate institution of anticoagulant therapy. In this situation it is essential to rule out, insofar as possible, intracerebral and subdural hematoma and other space-occupying lesions. Consequently, lumbar puncture should be performed to make sure that the spinal fluid contains no blood. Angiography in such patients usually is not indicated.

Precautions in the Use of Anticoagulants
If heparin is to be used, the dose must be controlled carefully to obtain the maximal protective effect while minimizing hemorrhagic complications. This objective probably can be achieved by maintaining the clotting time above 15 minutes, but below one-half hour. Clotting times longer than one hour may be associated with hemorrhagic complications. The optimal heparin dose is from 20,000 to 60,000 units per day. The clotting time should be determined every four hours, immediately prior to the next dose, which can then be adjusted accordingly. For dependable control, heparin should be given only intravenously.

The use of heparin as the sole anticoagulant over a prolonged period has disadvantages: the necessity of using needle and syringe and the possibility of developing osteoporosis. For these reasons, many physicians use a coumarin compound (warfarin sodium or bis-hydroxycoumarin orally) for long-term management. Others advocate the use of acetylsalicylic acid or dipyridamole.

Lumbar puncture should be done before beginning anticoagulant therapy in an attempt to exclude the presence of hemorrhagic infarction or intracerebral hemorrhage and should be repeated if progression of clinical disability occurs during anticoagulant treatment. Because of the risks involved, consultation with a specialist may be advisable before initiating anticoagulant therapy. In summary:

1. Plan to initiate anticoagulant treatment in the acute and progressive stage, while the stroke is incomplete. When the lesion is far advanced, the chances for reversing the process are less favorable.
2. Perform lumbar puncture to exclude the possibility of intracranial hemorrhage as far as possible.
3. Initially, determine the clotting time (by Lee-White method) and obtain a platelet count. Many authorities prefer to use the partial thromboplastin time (PTT) instead of the Lee-White clotting time because the former value can be obtained more quickly and is considered more reliable.
4. Administer heparin intravenously as soon as these values are reported, adjusting the dose as necessary. Some physicians prefer to employ a continuous intravenous drip providing approximately 1,000 units per hour. Others administer intravenous heparin (approximately 70 units per kilogram body weight) every four hours.
5. The laboratory determinations listed in item 3, omitting the platelet count, should be repeated at the end of the first four-hour period of the administration by continuous drip; if the dose-at-intervals method is selected, the determination should be made in advance of the second dose, due to be given four hours after the first. If values at the end of four hours are satisfactory, the tests can be made thereafter every 24 hours.
6. Check the hematocrit determination every other day.
7. Repeat the lumbar puncture, if clinical deterioration occurs.

CEREBRAL EMBOLISM
That cerebral infarction can result from embolism is well known. From the therapeutic point of view the issues worthy of attention are: (1) whether the pathophysiology of cerebral infarction due to embolism differs significantly from that due to primary arterial thrombosis, and if so, (2) whether that difference influences treatment. If optimal therapy for cerebral embolism varies from that for cerebral thrombosis, ability to make a clinical distinction between the two at the bedside is vitally important.
Pathophysiology

Unlike a primary arterial thrombus, an embolus arriving at and occluding a cerebral vessel may subsequently move distally, thus altering the size of the ischemic region and changing the pattern of collateral flow. An embolus that occludes the distal internal carotid artery might cause some degree of ischemia of the entire hemisphere, but it will permit collateral flow from the basilar and contralateral carotid vessels if the circle of Willis is competent. The adequacy of this flow determines whether any permanent neuronal dysfunction occurs. If the embolus then moves distally into the middle cerebral artery, ischemic infarction is likely to develop in regions served by this vessel. At the same time movement of the embolus from its original location permits flow through the other internal carotid branches. If obstruction to flow has been sufficiently prolonged for infarction to occur, restoration of flow may then make the infarct in those areas hemorrhagic.

The same events will occur in the middle cerebral territory if the embolus undergoes lysis after causing ischemic infarction there. On the other hand, if a thrombus forms in the stagnant blood column distal to the embolic plug, the evolution of the infarct will differ in no way from that caused by primary thrombosis.

Clinical Diagnosis

Much of what is taught and written about the clinical features of cerebral embolism is based on experience with the neurological complications of mitral stenosis, especially when it is associated with atrial fibrillation. These complications are characterized by sudden onset of neurological deficits during the waking hours, followed by rapid recovery. The patients in this group usually have significantly less cerebral atherosclerosis than do those with cerebral emboli from other causes: myocardial infarction, ventricular aneurysm, and atherosclerotic heart disease associated with atrial fibrillation. Hence it does not follow that the clinical events will be the same as those produced by cerebral embolism due to rheumatic heart disease, or that cerebral infarction in such patients can be ascribed confidently to emboli unless simultaneous vascular occlusion elsewhere is evident. As a rule, a firm diagnosis of embolism as the cause of cerebral infarction can be made only in cases of mitral stenosis with auricular fibrillation, or in instances where there is strong clinical evidence of multiple arterial occlusions occurring either simultaneously or in rapid succession. Fortunately, management of the acute event is virtually the same for embolism as for thrombosis.

Sources of Emboli and Special Therapeutic Problems

Stenotic Carotid Bifurcation and Other Proximal Arterial Sources. Embolism from these sources is considered to be a common mechanism of cerebral infarction and TIA; the incidence of symptoms is reduced greatly after internal carotid endarterectomy. Ulcerated carotid artery plaques have an intimal defect on which platelet-fibrin aggregates can form. Because current criteria for the radiological diagnosis of ulcerated plaques have definite limitations, we do not know their exact role in producing cerebral infarcts. For this reason, some authors prefer the term cerebral thromboembolism to the traditional cerebral thrombosis for all cerebral infarction not obviously due to emboli.

Our inability to recognize the precise origin of many emboli of noncardiac origin seriously complicates any effort to formulate a comprehensive description of the epidemiology, pathophysiology, diagnosis, and treatment of cerebral ischemia and infarction. Probably the most serious deficit in our investigative armamentarium is the lack of an adequate noninvasive method for visualizing the extracranial and intracranial circulation.

Rheumatic Heart Disease. Both mitral stenosis and atrial fibrillation are associated with embolism, but emboli occur more frequently when these conditions are present in combination than when either exists alone. Many authorities regard a history of embolism in patients with rheumatic heart disease as an additional indication for operation to correct the valvular lesion. Most cardiac surgeons believe that a history of embolism favors correction of the valvular defect by open-heart technique, since mitral valvulotomy, for example, is much more likely to be complicated by further emboli if done by a closed technique.

Cerebral embolism following a satisfactory mitral valvulotomy is infrequent, and many believe that long-term use of anticoagulants following this procedure is unjustified. Although the incidence of embolism in these patients is slightly higher if auricular fibrillation continues, it is still not great enough to produce universal agreement regarding the need for long-term treatment with anticoagulants. Embolism in association with bacterial endocarditis requires appropriate specific management.

Cardiac Valve Replacement. Although valve replacement with a homograft is seldom followed by emboli, replacement with an artificial ball or disc valve prosthesis is attended by a significant incidence of late emboli. In these cases chronic anticoagulant treatment has some protective effect. When adequate doses of anticoagulants are given, cerebral emboli (if they occur) appear to follow the classical pattern of sudden onset of neurological disability, which usually resolves rapidly and is not complicated by progression, early recurrence, or hemorrhage. Whether this relatively benign course is an effect of
the prior anticoagulant treatment is uncertain. According to one study, the incidence of emboli from prosthetic valves has been significantly reduced by administering a combination of anticoagulants and dipyridamole.

**Atrial Fibrillation Without Valvular Disease.** The most frequent cause of atrial fibrillation in patients without valvular lesions is arteriosclerotic heart disease. Other cardiac arrhythmias do not predispose significantly to cerebral embolism. Many cardiologists believe that focal cerebral infarction in elderly patients who have fibrillation, but no evidence of other arterial occlusions, is due more often to primary cerebral thrombosis than to cerebral embolism. In younger patients (below 45 or 50 years of age) the combination of atrial fibrillation and cerebral infarction is probably a valid criterion for the diagnosis of embolism.

**Cardioversion in Atrial Fibrillation.** Most cardiologists hold that cardioversion for auricular fibrillation is indicated in patients who have had recent embolism, with the possible exceptions of elderly persons with slow fibrillation and patients with longstanding fibrillation or who are candidates for early surgical correction of the underlying valvular lesion. Direct current, synchronized electroshock has largely replaced pharmacological cardioversion, but the arrhythmia usually will recur unless the patient is maintained on quinidine or other antiarrhythmic drugs. Many advocate prior anticoagulant treatment in an attempt to minimize the possibility of embolism at the time of conversion.

Atrial fibrillation in patients without a recent history of embolism and without mitral stenosis is considered by some to be an inadequate indication for cardioversion. If it is to be done in these persons, a prior period of anticoagulant treatment is probably unnecessary.

**Myocardial Infarction.** Embolism from a mural thrombus formed subsequent to myocardial infarction is a relatively uncommon occurrence. The consensus is that cerebral infarction complicating myocardial infarction is probably due to primary cerebral thrombosis. Firm data bearing on this subject are comparatively few, however. In a relatively rare case of ventricular aneurysm, mural thrombosis may cause recurrent embolus, and would be an indication for surgical correction.

**Bacterial Endocarditis.** This may be a cause of peripheral and cerebral embolism. Opinion is virtually unanimous that anticoagulants are not indicated in this disorder, in part because of the coincident hazard of subarachnoid hemorrhage from a mycotic aneurysm. In some carefully selected cases, valve replacement may be performed as a means of eradicating the infected focus, although experience in this area is limited. Operation may be indicated also in selected cases of ruptured chordae tendineae or valve perforation.

**Anticoagulants in Cerebral Embolism.** The diagnosis of cerebral infarction due to embolism is reasonably secure in mitral stenosis with auricular fibrillation. Evidence from several controlled studies indicates that continuous anticoagulant treatment with warfarin reduces the incidence of recurrent embolism for the duration of therapy. Appropriate management consists of maintaining the prothrombin time at about twice the control as measured in seconds.

Some physicians hold that anticoagulants should not be given during the first few days after a completed stroke, for fear of aggravating a hemorrhagic lesion. There is evidence, however, that in selected cases of cerebral embolism, the use of heparin during the acute phase helps to prevent progression of the clinical disability; an example is the prevention of hemiplegia by inhibiting the propagation of a thrombus distal to the occluding embolus, or by deterring recurrence of emboli to the same or other areas of the brain and body. The patients who qualify for early anticoagulant treatment are those whose lesions are clinically incomplete with less than total hemiplegia; theoretically, the lesions in these cases are small and result mainly in ischemic neuronal damage. A large controlled study revealed decreased mortality and morbidity when anticoagulants were begun early in patients with clinically incomplete lesions, while the reverse was true with those whose lesions were clinically complete, i.e., those who had complete hemiplegia on initiation of anticoagulant treatment.

**INTRACRANIAL HEMORRHAGE**

Intracerebral hemorrhage is related to hypertension in at least 60% to 70% of cases and to aneurysm and arteriovenous malformation in another 20%; in the remainder, bleeding occurs as a result of anticoagulant therapy, blood dyscrasias, tumors, and miscellaneous causes. In at least three-fourths of primary subarachnoid hemorrhages, an aneurysm or an arteriovenous malformation is responsible. Many of these patients and from 20% to 30% of the rest also have evidence of atherosclerotic or hypertensive disease. In a smaller percentage, nontraumatic subarachnoid hemorrhage is caused by blood dyscrasia, cerebral tumor, the use of anticoagulants or, in rare instances, infection such as herpes encephalitis. In some no demonstrable cause can be found.

The patient who is unconscious should be taken to the nearest emergency room for respiratory care,
diagnosis and, if necessary, initial management of his elevated intracranial pressure. From there he may be transferred to an ICU or stroke facility on the neurological or neurosurgical service. In the event that such facilities and personnel are not accessible locally, the patient probably should be moved to a hospital where they are available, provided he is judged able to travel, and means of transportation are adequate. Coma in itself does not contraindicate transporting the patient over a reasonable distance. If moving him is considered unsafe, the patient should remain at the initial hospital until his physician judges transfer to be a wise course (see: Coma, Transportation).

**Diagnosis**

**Parenchymatous Hemorrhage.** Extravasation of blood into the substance of the brain usually is a grave complication of long-standing hypertension. Sites of bleeding are mainly in the basal ganglia and capsular region, less often in the brain stem and cerebellum. The diagnosis is suspected by the acute onset of headache and impairment of consciousness, followed rapidly by focal or generalized neurological deficits, and culminating in coma and instability of other vital signs. Systemic blood pressure usually is elevated markedly in the acute stage. Blood will be found in the CSF in approximately 80% of these cases. Since the diagnosis of encapsulated parenchymal hemorrhage is difficult to make on clinical grounds, and many such cases are diagnosed as cerebral infarction, their exact incidence is unknown. The mortality from intraparenchymal hemorrhage is extremely high.

**Subarachnoid Hemorrhage.** A diagnosis of subarachnoid hemorrhage is usually based upon the history of sudden onset of headache, followed by impairment of consciousness, and the rapid development of a stiff neck in the next few hours. Focal signs are much less common than in parenchymatous hemorrhage or cerebral infarction. If the history is unavailable, alternate diagnoses to be considered are: (1) trauma to the head and neck, (2) meningitis, (3) increased intracranial pressure. The diagnosis is confirmed by finding large amounts of blood uniformly distributed in the spinal fluid. The diagnosis usually is established by angiography.

**Contrast Studies**

About 60% of patients with hemorrhagic stroke have radiologically demonstrable lesions. If the diagnosis of hypertensive parenchymatous hemorrhage is in doubt, contrast studies may be indicated. Although one would expect the risks of an angiographic procedure to be greatest in the early stages of subarachnoid hemorrhage, data from the Cooperative Aneurysm Study reveal that the morbidity and mortality are lowest when studies are done during the first few days after the event.

Cerebral angiography should be performed promptly if a hematoma which might require evacuation is suspected. This usually should be done, however, only in a hospital where personnel and facilities for definitive surgical treatment are available. Usually the surgeon who will operate can judge when a study is adequate. The decision concerning technique to be employed should be made by the surgeon with the help of the radiologist and the physician in charge of the patient (see: Section on Special Procedures and Equipment in the Diagnosis and Management of Stroke. Stroke 4: 111-137 [Jan-Feb] 1973).

**Medical Management**

Medical management should be conducted in the ICU, or in the neurological or neurosurgical unit. When the diagnosis is not yet established, the patient is admitted (either directly or through the emergency room) and the diagnostic workup is begun immediately.

If a hemorrhage is massive or is in the brain stem, death usually occurs within a few hours or days. In patients who have a less fulminant clinical course, death may come later as a result of severe arterial spasm, reduced cerebral blood flow, elevated intracranial pressure, cerebral edema, or hypoxia. The usual picture of parenchymatous cerebral hemorrhage is one of profoundly disturbed vital signs, as indicated by severe impairment of state of consciousness, marked fluctuations in blood pressure, respiratory and cardiac irregularities, and alterations in body temperature (see: The Comatose Patient). Cerebral vasospasm may not be apparent by cerebral angiography within the first 48 to 72 hours and may not be firmly established for the next several days. It may persist for several weeks. As yet there are no specific means of reversing arterial spasm.

Other causes of death are: (1) complications specific to the comatose state, (2) a progressive increase in intracranial pressure, and (3) recurrent hemorrhage. These three factors warrant careful attention.


**Fluids and Nutrition**

In the early stages after subarachnoid hemorrhage, fluid intake should be adjusted to no more than 1,500 ml daily. Gastric feeding generally is unsatisfactory because of possible vomiting and aspiration (unless the tracheal tube is cuffed), and fluid must be given intravenously. When coma
persists longer than five days, adequate caloric intake becomes a consideration. A nasogastric tube may be indicated when the danger of vomiting and aspiration has passed. Intravenous caloric preparations are available for use in those patients who continue to vomit. Tube feeding may be required early in patients with inaccessible veins. Approximately 1,000 calories (depending upon the patient’s weight) are required daily (see: The Comatose Patient, Fluids, Electrolytes and Nutrition). Since acute gastric ulceration is a well-known hazard of many cerebral disorders, the use of milk or an antacid preparation every two hours is advocated by many, particularly when corticosteroids are employed.

Bladder and Bowel
Some method of collecting the urine must be employed to keep the bed dry, to prevent decubitis, and to record fluid output. Condom drainage is best for men unless there is urinary retention. In men with urinary tract obstruction, and in most women, an indwelling catheter will be necessary. Urinalysis and culture should be done every few days.

Periodic rectal examinations are indicated to identify and remove fecal impactions. The patient is not likely to have frequent bowel movements until solid food is ingested. Bowel care is then handled by using stool softeners, suppositories, and enemas.

Sedation and Relief of Pain
Chlorpromazine in appropriate doses will control restlessness and also may reduce blood pressure slightly, a desirable effect in most cases. As much as 200 to 300 mg daily may be required, but the tranquilizing effect of this drug must be taken into account when judging alterations in the level of consciousness. Physical restraints (except for the use of side rails on the bed) are generally unnecessary.

Headache is often difficult to manage. Acetylsalicylic acid and codeine are the drugs chiefly used. Meperidine may be employed sparingly for short periods, but the use of narcotic analgesics should be discouraged because they invalidate evaluation of the state of consciousness resulting from the initial disorder. Some authorities employ lumbar puncture after other measures fail to control the headache.

Control of Fever
Mild to moderate elevations of body temperature are usual after intracranial hemorrhage. Although hyperthermia is no longer used widely, agreement is general that hyperthermia (above 39C) should be prevented by appropriate means. A cooling blanket may be necessary if all other measures fail (see: The Comatose Patient, Cerebral Edema and Intracranial Hypertension, Hyperthermia). A careful search must be made for infections which may be producing or contributing to the fever. The usual sources are the respiratory and urinary tracts.

Increased Intracranial Pressure. Increased intracranial pressure is usually caused by the combined effects of the subarachnoid hemorrhage, the mass lesion, and brain swelling. Although the results of surgical removal of an intracerebral hematoma during the acute stage are poor, drainage may offer some chance of improving the condition and should be considered, particularly if the clot is located subdurally or within the cerebellum. When surgery is not contemplated, the patient can be treated with fluid restriction (1,000 to 1,200 ml daily), plus slow mannitol infusion (30 ml of 18% mannitol in water per hour for ten hours) in some cases. In less severely involved patients, fluid restriction can be utilized, together with dexamethasone, given in divided doses of 12 to 16 mg daily intramuscularly or intravenously. This agent increases the danger of gastric bleeding to which the patient already is predisposed. The administration of gastric antacids is advisable in most instances, especially during dexamethasone therapy. Sometimes the intracranial pressure remains elevated. In selected cases a shunt from the lateral ventricle to the right atrium or the peritoneum can be considered when the patient’s condition has stabilized (see also: The Comatose Patient, Complications).

Arterial Spasm. Arterial spasm demonstrated by angiography is a serious problem which usually is inapparent within the first day or two after bleeding, but often becomes firmly established in four to seven days. It may be associated with progressive deterioration of consciousness and, if severe, is a contributing factor in the patient’s death, sometimes from cerebral infarction. There is no specific treatment for this condition.

Antihypertensive Therapy. (See: Hypertensive Encephalopathy.) Evidence currently available indicates that careful reduction of arterial pressure in hypertensive patients with hemorrhagic strokes may improve chances of survival. In the acutely ill, however, the relationships among cerebral perfusion pressure, CBF, and intracranial pressure are critical, and there is danger that reducing systemic arterial pressure will disturb this delicate balance. Systolic pressures above 200 mm Hg should be lowered sufficiently to reduce the risk of recurrent bleeding, but not enough to impair CBF. A 20% to 30% reduction in systolic pressure is advisable. Since in many instances of hemorrhagic stroke arterial pressure is controlled with difficulty using oral or intramuscular medications, the intravenous route may be necessary. However, if the intramuscular...
method is selected, reserpine or hydralazine can be employed. Reserpine can be given intramuscularly in divided doses of 0.5 mg up to a total of 3.0 mg per day. Some clinicians prefer to use hydralazine hydrochloride intramuscularly, 10 mg every hour until the systolic arterial pressure reaches the desired level. Then, hourly doses are discontinued and the drug is given in an average amount of 20 to 40 mg every four to six hours.

When management is difficult, methylpapa can be administered intravenously in the amount of 200 mg every four hours, gradually increasing to 400 mg every four to six hours until the desired effect is attained. In unusual circumstances, trimetaphan or sodium nitroprusside can be given intravenously to allow maintenance of blood pressure at a predetermined level.

In the less critically ill patient, arterial pressure above 160 mm Hg systolic or 100 diastolic is indication for the use of antihypertensive medication to reduce the risk of another hemorrhage. The commonly used antihypertensive agents usually are adequate for this purpose and the oral route is generally utilized. Some authorities feel that in the presence of a ruptured saccular aneurysm, a reduction of systolic pressure from the estimated normal by 10% to 20% is advisable until the patient is ready for operation.

**Antifibrinolytic Treatment.** Hemorrhage due to a ruptured aneurysm presumably is sealed by a blood clot which is subject to lysis by fibrinolytic activators after a period of one to two weeks. This interval coincides with the peak incidence of rebleeding, which is thought to be caused by lysis of a clot formed at the point of rupture. If lysis could be delayed for a week or two, fibrous tissue repair should help to prevent recurrent hemorrhage. Furthermore, a longer interval could be allowed before instituting elective surgery.

Antifibrinolytic therapy, using epsilon amino-caproic acid (EACA), is now being employed during the first two weeks after an episode of bleeding from a ruptured aneurysm. EACA is given orally or intravenously in divided doses of 24 to 36 gm in a 24-hour period. Early treatment (as soon as the diagnosis is made) is important; the intravenous route seems to be more effective than oral administration during the first several days of treatment. The efficacy of EACA after the two-week period is being evaluated.

**Surgical Management**

**Reduction of Intracranial Pressure.** When a hematoma is responsible for increasing intracranial pressure, its removal sometimes produces dramatic improvement.

**Removal of Hematoma**

Most of the patients who have a hemorrhagic stroke, but who do not have an aneurysm or arteriovenous malformation, fall into one of two categories: (1) old persons who have a high incidence of coma, hypertension, atherosclerotic disease, and intracerebral hematomas; in this group the mortality is very high; or (2) young patients with a low incidence of all the complications listed above; in this group the immediate mortality is low, and late prognosis is better. In the older group the hemorrhage usually destroys all traces of etiology; however, some pathologists believe that most of these cases are due to rupture of microaneurysms (0.5 to 2.0 mm in diameter) on the perforating vessels traversing the base of the brain. In some instances where arteriography fails to reveal the source of bleeding, the cause may be a saccular aneurysm which has become thrombosed, or a small (cryptic) arteriovenous malformation.

When a hematoma is demonstrated by angiography, the decision as to whether it is life-threatening depends upon the site and location rather than association with an aneurysm, arteriovenous malformation, or other cause. If the patient's clinical condition has deteriorated out of proportion to the size of the lesion, factors such as cerebral edema, venous or arterial thrombosis, or systemic causes should be sought. In these cases (small hematomas) operative removal is contraindicated. Unfortunately, most parenchymal hematomas involve the basal ganglion-capular regions and are so destructive that operative attempts are futile. On the other hand, if the hematoma is in the cerebellum or subdural region, surgical evacuation is frequently life-saving. In general, only a few hemispheral hematomas are suitable for removal and, even in this selected group, mortality is high, particularly if operation is attempted during the acute stage. Best results can be expected from evacuation of large, encapsulated hematomas confined to the frontal, occipital, or anterior temporal lobe. If the source of the hematoma is an aneurysm, the surgeon may find that the vascular lesion has been separated from brain tissue, and he may elect to remove it at the time the hematoma is evacuated.

**Burr Hole and Ventricular Drainage**

When monitoring intracranial pressure as a supplement to medical management is necessary, a burr hole through the anterior horn of the lateral ventricle will allow both measurement of pressure and drainage of the ventricle.

**Shunting of Ventricular Fluid**

Ventricular drainage affords relief for some patients but should be terminated before the tenth day after
insertion of the drainage tube to reduce the possibility of infection. A shunt may be necessary also in patients who fail to regain consciousness and whose lumbar pressure remains high because CSF is not resorbed. If hematomas have been excluded as a cause of intracranial hypertension and the angiographic pattern suggests ventricular distension, a shunt will help some patients to survive. Since the decision to operate can seldom be made until a few days, or even a few weeks, after the initial hemorrhage, serious neurological damage has already occurred in most cases and the results of operation generally are poor.

**Prevention of Recurrent Subarachnoid Hemorrhage.** In spite of inherent risks, surgical preventive measures on accessible lesions represent some of the most hopeful approaches to the treatment of saccular aneurysms. Many times a patient with verified aneurysm can be spared recurrent hemorrhage and the associated neurological devastation and mortality. If the aneurysm is not treated surgically, most authorities will continue antihypertensive measures in an effort to prevent transient elevations in systolic blood pressures.

**Carotid Ligation in the Neck.** This procedure is designed to lower intraaneurysmal pressure, but can be performed only if collateral circulation in the circle of Willis is adequate as determined by the Matas test. The greatest drawback to carotid ligation is the danger of sudden hemiplegia, especially during the first 48 hours after operation. This complication, which is probably caused by emboli arising at the site of ligation, is seldom fatal but can cause severe neurological deficit. Release of the clamp following the development of hemiplegia may or may not restore function. Complications are less apt to follow common carotid than internal carotid occlusion. Producing an occlusion gradually over a period of several days by means of a special clamp with a shaft protruding outside the skin of the neck is safer than occluding the vessel abruptly.

Although carotid ligation in the neck reduces pressure in the internal carotid and in the aneurysm by 20% to 30% or more, late follow-up shows that the pressure often returns to former values through collateral channels. In the meantime, the aneurysm may disappear or become smaller, or it may increase in size and even bleed again at a later date. Thus, despite low early mortality, this procedure is not definitive in many cases.

**Intracranial Occlusion and Ligature.**

This is the classical method of treating saccular aneurysms. If craniotomy is carried out in the first few days after a severe hemorrhage, the associated arterial spasm and cerebral edema lead to a prohibitively high mortality rate. The brain is usually edematous in patients who have had a recent hemorrhage. If the surgery can be postponed for two weeks or longer, operative mortality is reduced, but in the meantime, some will have rebled and died. For the patient whose aneurysm has not bled or whose hemorrhage has been only minimal, operation may be performed as soon as possible on accessible lesions.

Peak mortality from recurrent subarachnoid hemorrhage occurs at the end of the first and the beginning of the second week after the initial episode. In recent years, surgeons have reduced mortality rates significantly (some as low as 5%) by postponing operation, using the operating microscope, and keeping the patient hypotensive during surgery. Since The Cooperative Study of Intracranial Aneurysms and Subarachnoid Hemorrhage has listed in detail the mortality for each posthemorrhage day, every surgeon has statistics available by which to measure the possible success of each operating method.

Despite the wide variety of clips, their use is severely limited by the necessity for dissecting the aneurysmal neck. In some large aneurysms the neck is inadequate, and in others it is obscured by arterial branches. In all cases there is risk that dissection will cause rupture or spasm which may result in focal neurological deficits immediately or within a 24-hour period. Newer clip-grafts require less dissection than clips available formerly.

**Reinforcement of Aneurysms with Plastic.**

Aneurysms without a neck that can be occluded may be reinforced by plastic encapsulation. A follow-up study over 12 years indicates that this method is free of late complications. The procedure is now used routinely in some neurosurgical clinics. To prevent rupture, the patient should be kept hypotensive during the necessarily extensive dissection required by the operation. Arterial spasm, which dissection probably accentuates, remains a problem.

**Initiation of Intraaneurysmal Thrombosis.**

The techniques of initiating thrombosis within the aneurysm by open or stereotactic methods are still in the process of development. Those that have been described are: (1) electric thrombosis by the implantation of a copper needle or wire, (2) magnetic thrombosis by the intraaneurysmal injection of iron particles, and (3) the injection of animal hair, a procedure which does not appear to afford permanent obliteration in all cases.

**Excision of Arteriovenous Malformations.**

In about one-third of these cases epilepsy is the presenting symptom. In others, slowly developing paralysis or migraine-like headache may occur.
When cerebral hemorrhage is the initial manifestation, the risk of early recurrence is slight and, except in the presence of a life-threatening hematoma, ample time is available to allow the patient's recovery from the effects of hemorrhage before advising surgical treatment. Because repeated hemorrhages eventually do occur in about 30% of the cases, elimination of the malformation is mandatory if it is technically possible.

When the lesion is situated in a frontal, occipital, or temporal pole, or in the cerebellum, excision is feasible. When it is situated centrally or deep within the brain (especially if of massive size) simple excision is impossible. Ligation of arteries supplying the lesion is seldom of lasting benefit because collateral blood supplies develop. Radiation therapy has generally been proved inadequate, although it is still used in some clinics. Embolization of the malformation, by means of plastic beads introduced into the carotid artery, is now being studied; the procedure depends upon the fact that the beads follow the course of the larger arterial branches leading into the malformation.

**PSYCHIATRIC MANAGEMENT OF THE STROKE PATIENT**


The disability caused by stroke provokes a major identity crisis. Many patients view the attack as the first step in a decline that eventually ends in death. The change in level of functioning affects patterns of living, attainable life goals, and economic and social status. Along with alterations in living patterns, the patient with a damaged nervous system often has impairment of perception, control mechanisms, and communication, so that his abilities to cope and adapt are compromised. Alterations in cognitive and behavioral abilities lead to feelings of dependency. Moreover, the patient must come to grips with a new, and many times alien, body image that may include paralysis, aphasia, and sexual impotence.

Assessment of the degree of physical and intellectual loss is of paramount importance, since this often determines the stroke patient's psychic response to himself, his family, and his course of treatment. The psychological response to stroke is related to the time in the patient's life it occurs and to his prior level of functioning. To the dependent, senile individual it represents less change than to his younger more vigorous counterpart who is a working member of society and an important family figure.

The patient with poor interpersonal relationships who responds to anxiety with hostility and denial will experience a different emotional course from the one who had a more comfortable life pattern. Neurotic and psychotic tendencies can recur or be intensified.

A patient's mental ability following stroke can vary from remaining relatively intact to becoming grossly impaired; emotional responses may be totally appropriate at one extreme, or at the other may be completely colored by organic changes.

The patient's initial response is one of shock as he tries to understand what has happened to him. Fear of death and perhaps fear of insanity are major preoccupations. To help remove some confusion and to provide background for stabilizing thought, the patient should be oriented as soon as possible to place, day and time, diagnosis, and present condition. Thought processes and reactions to his disability should be elicited from him as far as possible. The physician can approach this situation by asking the patient to: (1) describe himself, (2) draw a picture of himself, (3) view himself in a mirror and describe what he sees.

Most patients seem apathetic, but for those who exhibit agitation, great anxiety, or psychotic thinking, appropriate dosages of psychotropic drugs such as phenothiazines or imipramine hydrochloride might be indicated. Great caution must be used, however, to avoid the cerebral, respiratory, and other complications related to the use of these drugs. For those who are only mildly agitated, chlordiazepoxide or diazepam may be chosen. Anxiety in an apathetic patient can be inferred from sweating, tremor, finger tapping, and from an inability to sleep at night or to relax during the day.

After the initial response, the patient often becomes mildly or severely depressed, as manifested by thoughts of low esteem, helplessness, irritability, low frustration tolerance, and anger at those around him. Often in the absence of obvious symptoms of depression, the patient is reported as functioning consistently at a level lower than that to be expected. Since depression can affect adversely recovery of function, antidepressants can be useful adjuncts to therapy.

The depressed patient should be watched for suicidal trends. The patient who wishes he were dead but has no other thoughts about death is different from one who wants to die, has tried to commit suicide, has a family history of suicide, or has a plan for suicide which he is physically capable of implementing. If a patient is thought to be a definite suicide risk, his transfer to a psychiatric unit should be considered.

Denial is one of the protective mechanisms most difficult to manage. A patient may deny any loss or disability at all, or he may admit the disability but deny its consequences. The family also, as a group and as individuals, have suffered a loss and experienced identity crisis and depression, and
their level of denial may rival or even exceed that of the patient.

The rehabilitation program is seriously affected by the response of the family toward the patient. For example, a wife may be so overprotective that she does not allow her husband to do things of which he is capable, or because of guilt feelings she may not be able to express appropriate anger. The attending physician might arrange periodic conferences, if appropriate, during which family members and the patient are helped to view the situation realistically in planning for the future. These sessions should continue until major problems are solved.

General progress and information pertaining to treatment and rehabilitation should be discussed with both the patient and his family. They must be adequately prepared for necessary changes in location, staff, or program because these, if unexpected, can reawaken a variety of emotional reactions. If the patient has significant organic impairment, abrupt changes in a familiar environment can result in disorientation, confusion, and bizarre behavior. Despite adequate precautions, environmental disruption may give rise to some psychopathological manifestations even in an apparently stabilized patient.

Group therapy has been suggested for stroke patients as an aid in helping dispel feelings of loneliness, and as an opportunity to draw strength from similarly affected persons. Such groups should include stroke patients who have recovered verbal or communicative ability.

The role of the psychiatrist in treatment of the stroke patient is sixfold:
1. To identify and manage the suicidal patient,
2. To help the staff deal with specific psychiatric problems in certain patients,
3. To help the staff increase its level of expertise in caring for stroke patients,
4. To elucidate the special needs of patients with perceptual losses,
5. To assist the staff in organizing in-house psychological training programs, and
6. To remain accessible in offering psychiatric follow-up support both to the patient and to his family throughout all phases of rehabilitation.

Recommendations of the Study Group

ASYMPTOMATIC PATIENTS WITH TREATABLE PREDISPOSING FACTORS

1. Further research should be performed in construction, testing, and validation of questionnaires designed to identify individuals in the general population at increased risk, so that appropriate preventive measures can be instituted.

2. Programs of education should be designed to heighten public awareness of the known risk factors, warning symptoms and signs associated with cerebrovascular disease.

3. Investigation is needed further to identify new risk factors for stroke.

4. Increased attention should be focused on early precursors of atherosclerosis and hypertension, particularly in children.

5. Blood lipid patterns and the blood pressures of asymptomatic persons who may be at high risk because of genetic predisposition or acquired disease must be determined and prophylactic measures instituted as early as possible.

6. Individuals with sustained hypertension must be identified early and treated vigorously through a program of national scope directed toward educating health professionals and the public.

7. Further research is needed on the possible effects of transitory elevations or reductions in arterial blood pressure as precipitants for stroke.

8. Epidemiological investigation of variations in incidence and prevalence of stroke among races, sexes, and in geographic regions, and the causes thereof is necessary.

9. Studies of the factors that predispose some arterial systems to atherosclerosis must be expanded.

10. A controlled study should be undertaken to determine the predictive value and the proper management of aorticocranial bruits in asymptomatic individuals.

11. Studies to determine whether there are detectable anatomical predictors for stroke should be expanded by systematic postmortem radiographic and anatomical analyses of the entire aorticocranial arterial network in asymptomatic and symptomatic populations.

12. Studies of the relationship between various cardiac disorders and stroke should be expanded.

13. Contraceptive techniques which do not increase risk of stroke in women should be developed.

SYMPTOMATIC PATIENTS

14. Public education concerning the urgency of recognizing and treating significant symptoms of stroke must be instituted.

15. Federal, regional, and local health authorities should identify and evaluate the adequacy of strategically placed stroke care facilities and their readiness to accept and treat promptly the stroke victim.

16. Every telephone system in the United States should have a single well-publicized number for emergency use.

17. Undergraduate and postgraduate medical education in every aspect of stroke should be intensified for health personnel.

18. Auscultation of the head and neck for aorticocranial atherosclerosis should become a routine
part of the screening examination at all levels of the health care delivery system.

19. Noninvasive, risk-free, rapid, and inexpensive methods for detecting extracranial and intracranial vascular disorders, and for measuring CBF and metabolism are needed urgently.

20. The community hospital should provide the best possible care for all patients with the following cerebrovascular disorders:
   a. The completed stroke (thrombosis)
   b. Cerebral embolism
   c. Massive intracerebral hemorrhage
   d. The transient ischemic attack in which surgery is not indicated.

   The community hospital should be prepared to obtain immediate specialized consultation and to arrange patient transfer if necessary in the following cases:
   a. The semicomatose or comatose patient
   b. Stroke-in-evolution
   c. Vertebral basilar thrombosis
   d. Subarachnoid hemorrhage
   e. Developing intracerebral hematoma
   f. Transient ischemic attack in patients for whom surgery is contemplated.

21. The community hospital should evaluate its capabilities in the following areas:
   a. Use of consultative services
   b. Training of a "local stroke consultant"
   c. Training and efficient use of allied health personnel (including physician's assistants)
   d. Concentration of stroke patients in the same nursing unit.

22. Leadership for improving the competence and confidence of a hospital staff in handling stroke problems should come from its own physicians, with outside consultants and teachers as required.

23. Each hospital should have at least one physician who functions as stroke coordinator. He must have a position in the hospital administrative echelon which gives him authority to serve in a surveillance capacity within his institution.

24. Every means available should be used by federal, state, and local governments and agencies to develop and support training programs for more neurologists, family physicians, occupational, speech, and rehabilitation therapists who will be active and interested in cerebrovascular disease.

25. Support should be continued for the development, constant revision, and dissemination of standardized diagnostic and therapeutic guidelines for cerebrovascular disease.

26. The patient and family should receive early instruction in rehabilitation procedures.

27. All pertinent deliverers of health care should be coordinated in discharge planning and follow-up of the patient.

28. Methods should be sought for effectively utilizing the stroke team concept.

29. The concept of "Stroke Care Centers" or "Cerebrovascular Disease Centers" should be explored further and the need for increasing their availability assessed.

30. Continued support of research into the pathogenesis, prevention, and management of stroke is imperative. Repeatedly during the preparation of this report, gaps in our knowledge became apparent and only through ongoing research coupled with the delivery of care can new and better forms of therapy be found. Particularly there is need for studies of antithrombotic agents, risk factors, screening techniques, noninvasive techniques for recognizing cerebrovascular disease, and the role of medical and surgical management. Such investigations will be required to establish the indications, limitations, and benefits of new approaches to the management of stroke.

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- Arthur L. Benton, Ph.D.
  Professor of Neurology and Psychology
  University of Iowa
  Iowa City, Iowa 52240

- A. B. Boutros, M.D.
  Professor, Department of Anesthesia
  University Hospitals
  University of Iowa
  Iowa City, Iowa 52240

- William F. Bouzarth, M.D.
  Clinical Associate Professor of Neurological Surgery
  Temple University,
  Deputy Director
  Department of Neurological Surgery
  Episcopal Hospital
  Front Street and Lehigh Avenue
  Philadelphia, Pennsylvania 19125

- Charles E. Brackett, Jr., M.D.
  Professor of Surgery, Neurosurgery
  Kansas University Medical Center
  Kansas City, Kansas 66103

- Roy H. Clauss, M.D.
  Professor of Surgery
  New York Medical College
  Flower and Fifth Avenue Hospitals
  Fifth Avenue at 106th Street
  New York, New York 10029

- Harold Collings, M.D.
  Professor of Neurology
  Medical College of Georgia
  Augusta, Georgia 30902

- Edward S. Cooper, M.D.
  Associate Professor of Medicine
  University of Pennsylvania School of Medicine,
  Co-Director, Stroke Research Center
  Philadelphia General Hospital
  Philadelphia, Pennsylvania 19104
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TRANSIENT ATTACKS OF NEUROLOGICAL DYSFUNCTION


THE COMATOSE PATIENT


Differential Diagnosis of Acute Neurological Deficits

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Differential Diagnosis of Acute Neurological Deficits


TIA AND STENOTIC LESIONS OF THE NECK VESSELS


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JOINT COMMITTEE FOR STROKE


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