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Diagnosis and treatment of the early stroke

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DIAGNOSIS AND TREATMENT OF THE EARLY STROKE

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INTRODUCTION

This paper has a twofold purpose: First, the clinical and pathological definitions of a stroke with related problems in diagnosis and management are considered. Second, recent and perhaps more sophisticated approaches to the diagnosis and management of the specific type of cerebrovascular accident are discussed.

DIAGNOSIS AND TREATMENT OF THE EARLY STROKE

PART I

Clinical definitions.[1,2]

Clinical definitions of the various stroke syndromes are somewhat meaningful by the uniformity of usage and by virtue of a certain conformity to the clinical picture developed. They are:

A. Impending stroke: periods of focal cerebral ischemia -5 to 30 minutes - with intermittant recovery.

B. Progressive stroke: increasing neurologic deficit dur-ing a short period of time - less than 24 hours.

C. Completed stroke: maximal damage has occurred.

D. Healed stroke: maximal recovery has occurred.

Pathologic definitions.[3]

The term cerebrovascular accident (CVA) is a pathologic phrase often used interchangebly with the more clinical term, stroke. The clinical picture in stroke is the result of loss of neurologic function of a portion of the brain. The term CVA implies that the neurologic deficit is the result of a vascular lesion. These lesions may be classified as follows:

A. Cerebral hemorrhage.

Etiology:

1. Arteriosclerosis - most common.

2. Hypertention - although intracerebral hemorrhage

can occur in normotensive patients, it is much more common in the hypertensive patient.

3. Angiomatous malformations.

4. Aneurysms.

5. Accompanying infections (hemorrhagic encephalitis), toxic agents (arsephenamides, sulfonamides), or systemic diseases (polyarteritis nodosa).

B. Cerebrovascular occlusion.

The amount of tissue damage resulting from cerebrovascular occlusion depends to a great extent on the availability of collateral circulation. If the collateral circulation is intact, a major vessel such as the internal carotid artery may be occluded without the development of symptoms. If the collateral circulation is severely impaired, there is softening of the brain tissue (encephalomalacia). If the collateral circulation is partially impaired, softening will occur or transient symptoms may develop (cerebral vascular insufficiency. The two types of cerebrovascular occlusion are:

1. Cerebral thrombus.

Etiology:

a. Arteriosclerosis - most common.

b. Hypertension - frequently associated.

c. Acute inflammatory reaction around blood vessels (meningitis or en-cephalitis).

d. Other rare causes -polycythemia, tumor (by impinging or by vascular spasm - hypothesis).

2. Cerebral embolus

Source:

a. Associated with heart disease - most common. (In elderly patients embolus formation is frequently secondary to atrial fibrillation or mural thrombus resulting from coronary occlusion. Rheumatic heart disease and bacterial endocarditis are the most common cause of emboli of cardiac origin in children).

b. Associated with thrombotic or suppurative process in any part of the body.

c. Arteriosclerotic plaque.

d. Air embolism associated with bends or injury to the lungs.

e. Fat embolism-usually associated with fractures of the long bones.

Note: the mode of passage of an embolus from the systemic circulation to the brain is not understood. However, it could be explained by such phenomena as a patent foramen ovale or the paravertebral plexus of veins.

Diagnosis of Stroke.[3,4]

Frequently the physician does not see the patient with cerebrovascular disease until there has been a sudden and dramatic onset of coma or of focal neurologic signs, commonly described as "shock" or "stroke". Although there may be transient symptoms indicating intermittant cerebral ischemia (hemiplegia, hemianesthesia, headaches, and periods of giddiness), in the vast majority of cases, the symptoms of stroke are of sudden onset. If the onset occurs while the patient is awake, he may be found in coma or more commonly may slump to the ground in a state of paralysis with no alteration of consciousness. If the vascular accident occurs during sleep, the patient may also pass into coma, or he may fall to the floor on attempting to get out of bed. When coma or stupor occurs, it may be of a few minutes or a few hours duration, or it may persist until death.

Generalized or focal convulsions occur at the onset in a small percentage of cases. They are more common early in hemorrhage, as are headache and vomiting.

Focal neurologic signs are dependent on the size and site of the lesion, and are variable depending on the location of the lesion. If the lesion is in a relatively unimportant area, there may be no focal neurologic signs. The degree of permanence of focal neurologic signs cannot be predicted within the first few days after onset; they may be due to actual destruction of nervous tissue, or to temporary interruption of function by edema or other factors.

The presence of signs and symptoms of disorders known to be etiologic factors of the stroke syndrome, e.g. cardiovascular disease -- arteriosclerosis, hypertension, rheumatic heart disease, are helpful. A patient with mild impairment of the cerebral blood supply is likely to become victim of a stroke in the presence of a decrease in cardiac output, a decrease in pulmonary function, or anemia; for example, coronary thrombosis, pneumonia, or at the time of surgery.

Although there are no definitive laboratory tests, the following abnormalities in laboratory values are frequently noted:[3]

A. Urine analysis.

Albumin and casts are found in the urine of the majority of patients with cerebrovascular lesions. This is, in part, due to concomitant kidney disease, but a transient albuminuria may occur in patients with relatively normal kidneys. Glycosuria may be present in the first 24 hours.

B. Blood

Transient hyperglycemia may be present. The NPN is usually normal without the presence of renal disease. Leukocytosis is mildly to moderately common with cerebral hemorrhage and uncommon with cerebral occlusion.

C. Cerebrospinal fluid

1. with cerebral hemorrhage, the C.S.F. is usually bloody and is under increased pressure. It may occasionally

be clear, but the pressure is still usually elevated.

2. with cerebral thrombus, the C.S.F. is usually normal, although there may be a slight increase in pressure, a slight pliocytosis and a slight increase in protein.
3. with cerebral embolus, the C.S.F. may be clear or xanthrochromic, and there may be moderate pliocytosis and increase in protein contents, especially if the embolus is septic.

Management of stroke

In light of the fact that the conservative methods of treatment are not specific for vascular disease, and that the newer methods have somewhat controversial results; there is no completely satisfactory medical treatment for the cerebral vascular lesion. The conservative treatment consists of the following general supportive measures:[1,3,5]

A. Maintain adequate airway. This may include: removal of secretions by suction, tracheostomy, and turning the patient every two hours to prevent hypostatic pneumonia
B. Prevent hypoxia by treating with oxygen and maintaining blood supply through efficient cardiac output
C. Maintain fluid balance and nutrition
D. Good nursing care is essential to the ultimate recovery of the patient. This should include, where necessary, catheterization, keeping bowels open, skin care, prevention of contracture, and so on.

E. Attention to the miscellaneous problems which may arise: These are pain, seizures, etc.

PART II

Diagnosis of the type and location of the lesion.

Because the newer methods of treating strokes are aimed at the treatment of a specific lesion, diagnosis of the type and location of a lesion now becomes important and will be considered here.

Of primary importance is the distinction between occlusive and hemorrhagic vascular lesion. The single most important factor in the differential is the presence of symptoms of signs indicating that blood has escaped into the subarachnoid space, suggesting hemorrhagic disease. These are: Severe headache, stiffness of the neck, and bloody cerebral spinal fluid on lumbar puncture.[3]

The absence or marked decrease of pulsation of the carotid arteries is indicative of occlusive vascular disease.[1] A bruit over the carotid artery, when not transmitted from the heart, indicates either narrowing of the artery or (when the opposite vessel is occluded) increased collateral circulation between the ophthalmic and external carotid arteries. Absence of pulsation in the temporal artery may indicate complete obstruction at or below the bifurcation of the common carotid artery.

Arterial compression, particularly in conjunction with electroencephalographic monitoring, can provide valuable information in patients with cerebrovascular insufficiency. Abnormality following compression of one carotid vessel indicates that the collateral circulation of the other carotid artery and the

vertebrobasilar circulation are inadequate to compensate. Bilateral carotid or vertebrobasilar insufficiency is usually present when signs and symptoms can be elicited through compression of either carotid artery. Hyperextension and rotation of the neck to the left or right may elicit signs and symptoms of vertebrobasilar insufficiency in the presence of pathological occlusive processes (such as spondylosis or disk protrusion). These procedures must be applied with caution, since they are accompanied by obvious risks.

The measurement of vascular pressure can be of help in determining the presence and location of vascular occlusive disease. The following methods have been suggested:

A. Ophthalmodynamometry.[1]

The retinal vessels are observed with an ophthalmoscope while a known degree of pressure is progressively applied to the lateral aspect of the eyeball. The pressure is raised until arterial pulsation collapses (diastolic reading), and until pulsation ceases (systolic reading). A rapid screening method known as the ambliopic method, which utilizes the systolic pressure required to change the vision, has recently been introduced.[1] Both false-positive and false-negative readings may be obtained, but a decreased pressure may give evidence of an obstructive lesion on the same side. The most common complication is subconjunctival hemorrhage, although retinal

detachment and infarction also may occur. The most valuable application is in the evaluation of vascular surgery.

B. Temporal artery pressure.

The external carotid arterial pressure may be estimated by the amount of pressure required to compress its superficial temporal branch. A decrease in pressure may also give evidence of an obstructive lesion on the same side.

C. Direct measurement of the common carotid artery pressure.

Direct measurement of the common carotid artery pressure involves cannulization of the artery. This procedure may be used in conjunction with common carotid arteriography, but has some of the same disadvantages.

Arteriography [1,3,6,7,8,9] of the blood-vascular supply to the brain is practically a prerequisite to the newer forms of treatment available for stroke victims. This technique will frequently demonstrate the type and location of a vascular lesion. There is justifiable reluctance to perform cerebral angiography in elderly patients with arteriosclerosis of cerebral vessels. It should not be performed when the diagnosis is obvious.

Clinical examination often fails as a diagnostic tool, in the differentiation of: cerebral artery occlusion from small non-penetrating hemorrhage, cervical carotid stenosis or occlusion from that in a middle cerebral artery, or hemiparesis due to

atheromatous disease from similar weakness produced by slowly growing mass lesion. Senile dementia caused by carotid artery disease in the neck may be impossible to tell from dementia occurring in the absence of such lesions.

Methods of cerebral angiography have passed through a gradual evolution from injection of the exposed carotid artery to various transcerevical techniques in general use today. Percutaneous puncture of the carotid artery, by far the most common procedure, carries a three to four percent complication rate in most clinics. These complications include a variety of local and neurologic sequelae ranging from periarterial hemorrhage to death. In recent years, although cognizance of the important role played by disease of extracranial arteries in cerebral dysfunction has increased, enthusiasm for extensive angiographic survey has been tempered by recognition of the dangers sometimes accompanying puncture of the neck arteries. This situation has led to a search for methods that will introduce opaque media without damage to any part of the cerebrovascular tree.

In some clinics, the site of puncture has been changed to the subclavian artery or to the arch of the aorta, which lessens the risk of embolization and substitutes damage of the larger arteries nearer the heart. Pneumothorax and hemathorax represent a major disadvantage to these approaches. Regardless of the site and land marks used, insurmountable disadvantages of all blind techniques

have been: individual anatomical variability and unpredictable effects of local disease.

A number of indirect methods of cerebral angiography proposed during recent years. A peripheral vessel is catheterized percutaneously or through an incision, and opacifying media is then forced retrograde through the vessel until it flows into the cerebrovascular tree. For example, if a 10-12" catheter is introduced into the right brachial artery and up to 30cc of a suitable substance is suddenly forced into the catheter, the substance will flow retrograde into the subclavian artery and the innominate artery, opacifying the right vertebral artery and the right carotid arteries. Often the substance will also flow into the aorta - opacifying the left common carotid and the left vertebral arteries via the left subclavian artery. As an adjunct to catheterization of the right brachial artery, the left temporal artery is often catheterized, allowing visualization of the left carotid arterial tree. The left brachial artery can also be used to insure visualization of the left vertebral artery.

Kuhn and Kugler studied the cerebral circulation in more than 500 patients by retrograde injection of opaque media into the brachial artery. Despite the use of relatively large volumes of contrast substance, the only complication encountered with this technique was three generalized seizures and no neurologic sequelae.

They believe that the freedom from complications is ascribable to the method, and that sequelae during and after percutaneous transcervical angiography are due to local and distant effects of the puncture.[9]

Thomas H. Newton has found a similar approach, utilizing the axillary artery, the most useful in surveying patients with suspected extracranial arterial disease;[10] the right axillary artery is usually chosen in these cases. Usually two injections of 40cc. of 76% Renografin are made, one with the patient in the antero-posterior position with the head turned to the left, and the other with the patient in the right posterior oblique position and the head turned to the right. The catheter may be withdrawn into the innominate or the subclavian artery so that the right carotid and vertebrobasilar systems are better seen; in this case only 15 - 20 cc. of contrast material is injected. The axillary approach may also be used for catheterizing the vertebral arteries directly. No serious local complications occurred in 160 cases of axillary artery aortography. Transient hemiparesis occurred in one patient on inadvertant injection of 40cc. of contrast material directly into the innominate artery. Partial extravascular injection occurred in four patients who suffered no sequelae.

In other studies in which the same or similar methods of arteriography were used, temporary signs and symptoms of impaired radial artery circulation (decreased radial pulse) were encountered. These did not last more than a week.

Newer Forms of Treatment

Anticoagulants [1,3,11,12,13,14] have been administered to patients with embolic or thrombotic lesions with the aim of preventing the development of new lesions. There is disagreement as to the value of this form of therapy. At the present time it is generally accepted that anticoagulant therapy is indicated in cerebral embolus (cardiac, peripheral, etc.), and in patients with intermittant carotid or basilar artery insufficiency.

The administration of these agents is contra-indicated for patients with cerebral hemorrhage or arterial hypertension. Other contra-indications to the use of anticoagulants and all the common precautions for their use should be observed. They are of no value in patients with stable infarcts. There are no established criteria for the length of the period of anticoagulant therapy. If there are no untoward effects, the therapy should be continued for six or more months in patients with the diagnosis of intermittant insufficiency, and for many years, or perhaps for life, in those with a source of cerebral embolic phenomena.

The results of many large series of studies [3,7,15,16], 189 patients from nine Veterans Administration hospitals, 380 patients from seven clinical centers, and 142 patients in Great Britain, indicate that the use of anticoagulant therapy should be even more restricted. In these studies the mortality rates were higher in

the groups receivin anticoagulant therapy than in the control groups. When the number of deaths ascribable to hemorrhagic complications were deducted from the anticoagulant group, the mortality rates were generally about equal. The number of occurrances of cerebral ischemia is usually reported to be significantly decreased.

Not all investigators agree: for instance, the mortality rate in 53 patients with cerebral embolism who received no anticoagulants was 25%, compared with 6% in 34 patients who did receive the medication.[17]

The investigation at the Mayo Clinic separated the patients with vertebrobasilar systemic thrombosis from those with thrombosis of the carotid system. In patients with vertebrobasilar thrombosis who received anticoagulant therapy, the mortality rate was 8% in contrast to 58% in those who did not receive anticoagulant therapy.[18]

Whether or not the patient is a candidate for anticoagulant therapy, depends on several factors in addition to pathologic conditions: the expense of the procedure, the time involved for the patient and physician, the reliability of the patient and the laboratory, and the accessability of patient, physician, and laboratory to each other. The inability to follow the patient or inability to rely on a patient, constitute unequivical contraindications to anticoagulant therapy. It is also imperative that the clinician be fully cognizant of the disaster of overdosage and, also of the increased danger of thrombosis following the sudden withdrawal of

these medications.

During the past few years, attempts to dissolve a clot within the cerebrovascular system have been made by injecting fibrinolysin or plasmin intravenously or directly into the common carotid. The results of studies do not agree. In one study, 7 of 11 patients treated died and the best result in the 4 remaining was a patient who was ambulating, but had persistant hemiparesis.[20] In another study,[19] anticoagulant and fibrinolytic therapy produced clot lysis, as judged by arterograms made before and after treatment, in 10 patients to whom treatment was administered within the first 6 days of symptom occurrence. Over 50% of patients so treated showed some clinical improvement, but comparison with similar untreated patients is still under investigation. Double-blind studies of fibrinolysin therapy in 40 cases of progressive strokes indicate that enhanced fibrinolytic activity may be achieved without danger to the patient.[21] Allergic reactions and fever of 1-6 degrees F. above normal are common. The procedure involves arteriography, which entails its own risks.

A series of experiments[22] demonstrate the ineffectiveness of cervical sympathectomy as a means of producing, in both dogs and humans, an increase in cerebral blood flow, after it has been reduced below normal by obstruction of the internal carotid arteries.

The circulation of blood in the small cerebral vessels (microcirculation) appears to be under chemical rather than neurogenic control. Of numerous drugs used in attempts to increase cerebral

blood flow, the majority (such as aminophylline, nicotinic acid, alcohol, papaverine, histamine and tolayoline (priscoline)) have reduced cerebrovascular resistance, but also have reduced intra-arterial pressure.[20] The net result has been, no increase and, in some cases, a reduction in cerebral blood flow.

Intravenous administration of acetazolamide and inhalation of 5-10% CO_2 are the only methods that have been shown to cause an absolute increase in cerebral blood flow.[1] Clinically, these two techniques have not been generally rewarding, since cerebral blood flow has already increased maximally as a result of homeostatic mechanisms responding to CO_2 at the site of the infarction and to local changes in intra-arterial pressure. However, the findings of one investigation[22], support the careful trial inhalation of mixtures of 5% CO_2 in the treatment of patients with strokes caused by partial obstruction of the cerebral arterial blood supply, provided that the patient is observed closely for evidence of CO_2 intoxication.

Intracerebral or subarachnoid hemorrhages have been treated by inducing hypotension[1] through the use of medications such as reserpine, hydrolazine, and ganglionic blocking agents in conjunction with elevation of the head using a tilt table.[23] Hypotension has also been produced to some extent by gradual lowering of cerebrospinal fluid pressure via lumbar puncture. These techniques require close observation, frequent neurologic examinations, and EKG monitoring. It has been advocated, particularly

for patients who are not immediate surgical candidates. Among 15 patients so treated, 12 survived.[24] The primary difficulty is increasing cerebral ischemia. This may be counteracted by hypothermia.[1] Hypothermia, with or without hypotensive therapy, seems to hold promise in the treatment of some severely ill patients - particularly in those with subarachnoid hemorrhage.

A number of agents, including steroids, hypertonic glucose, urea, and magnesium sulfate, have been used with various degrees of sporadic success to reduce cerebral edema secondary to infarction and hemorrhage.[1]

Research to date on the use of hyperbaric oxygen has been limited but it seems established that damage following cerebrovascular accidents can be minimized by high pressure oxygen therapy.[25]

Surgical intervention is beneficial chiefly in the impending stroke and advancing stroke,[1] where it may cure, although its efficacy in the latter is questionable.[2] In the maximally completed stroke, surgery is usually a therapeutic misadventure, although, there are infrequent and interesting exceptions. In the following paragraphs, the surgical management of the stroke syndrome will be discussed in correlation with specific cerebral lesions.

A patient having symptoms of cerebral ischemia may be considered a candidate for surgical correction only if: (1) a surgically correctible lesion is found and (2) physiologic correlation between symptoms and lesion is demonstrated.[26]

At present, surgical therapy can be applied principally to those patients whose arterial lesion is extra-cranial and stenotic in type.[2] Procedures leading to bypass of the pathologic section of the artery by means of a tube of synthetic material, such as dacron, or thromboendarterectomy are generally used either singly or in combination. In nearly 50% of the patients, more than one artery is involved. In one study, blood flow in the distal arterial bed was restored and symptoms improved in 90% of the patients in whom the vessel had only been stenosed; if the vessel has been occluded, blood flow is restored in about 36% and there is a correspondingly poor functional return.[27] In another study[2] considering 35 patients, 66% of the treated returned to normal, compared to 25% of a contrast group; and 17% suffered cerebral infarcts compared to 40% of the contrast group. Although the surgical mortality is 5 to 18% depending on a variety of factors, including the procedure employed and the selection of patients, this operation provides a long term prophylaxis of value against cerebral infarcts.

Surgical intervention in subarachnoid hemorrhage and intracranial aneurysm is the treatment of choice, unless specific contraindications exist. Some of the methods used to control aneurysms by intracranial approach are:[28] (1) placement of a clip at the neck of the aneurysmal sac, (2) isolation of the aneurysm between two clips, (3) excision of the sac and closure of the vessel, and (4) coagulation of the sac after either inverting the sac or in-

serting muscle into the aneurysm.

Signs and symptoms of blood in the subarachnoid space are of grave prognostic significance. In a series of 40 patients of whom some had blood in the spinal fluid, but little impairment of cerebral function, the mortality rate was 2.5%.[29] Use of intracranial approach for patients who are definitely drowsy or stuporous, or who show signs of mild hemiparesis, was accompanied by 22% mortality rate. Unless bleeding is progressive and uncontrolled, patients of this group should be managed conservatively in the hope that they will improve and become better surgical risks.

Surgery is contraindicated in patients who are comatose or deeply stuporous; in this group, the mortality rate is 86%. The following are characteristic of patients with intracerebral hemorrhage: signs and symptoms of increased intracranial pressure, focal neurologic signs or convulsions, shift of calcified pineal gland away from the side suspected of hemorrhage, and localization of a mass through arteriography or ventriculography. [1]

The usual course followed by patients suffering from an intracerebral hemorrhage is:[30] an initial apoplectic onset, after which they often become unresponsive for several hours and then their condition gradually stabilizes. As a rule, these patients then will begin to improve and continue on a slow rise to maximum recovery. However, when patients begin to attain a plateau at from

2 to 5 days from the onset of the cerebral vascular accident, or when, during this interval, they begin a secondary decline, it may be in association with the expansion of a localized clot rather than in association with primary cerebral damage, infarction, or cerebral edema. In this group, the aspiration of the hematoma may contribute materially to further improvement. Long term consideration of this group of patients is not yet known, and their selection is still a matter of some difficulty. The surgical mortality for intracerebral hemorrhage averages approximately 20%; it is higher in the hypertensive patient than it is in the non-hypertensive patient.[1] It has become evident that little can be achieved by aspirating an intracerebral hematoma in the early hours following the onset of symptoms.[30] The blood at this time is semisolid and is interspersed with white matter from destroyed centrum ovale, so that aspiration cannot be accomplished. If the hematoma is decompressed in the acute stage, the evacuation of the cavity has an inconclusive end point, and bleeding continues.

Summary

In part I the clinical and pathologic definitions of stroke along with etiologic factors were discussed briefly and in outline form. The conventional means of diagnosis and management commonly in use were reviewed in a similar manner.

The second part was concerned with more recent, and perhaps more sophisticated forms of diagnosis and treatment of a specific vascular lesion. The importance of the differentiation between hemorrhagic and occlusive vascular disease was emphasized. Several methods of estimation of arterial patency and arterial pressure were outlined. Arteriography was extensively discussed and some newer approaches to opacifying the cerebrovascular system were presented. The indications and the contraindications of anticoagulant therapy were reviewed and; the results of anticoagulant therapy as opposed to non-anticoagulant therapy were presented in several different series of patients. The efficacy of various vasodilators and numerous other therapeutic regimes was presented. Surgical intervention in cerebrovascular lesions of different types was reviewed in a non-technical manner designed to give the reader an understanding of many surgical possibilities.

Conclusion.

From the for-going presentation it must be concluded that, in respect to the management of the stroke syndrome, there are definitely conservative and non-conservative methods. In the latter, while the patient no doubt has a better chance for more complete recovery, he must be subjected to the added risk of the therapy itself and also, the added risk of the diagnostic procedures necessary to determine the type of therapy indicated.

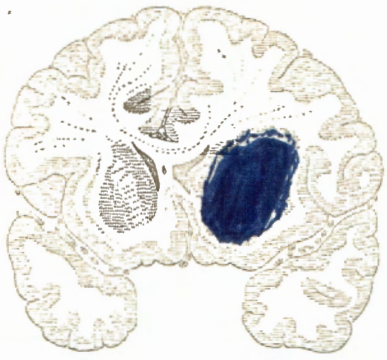
The decision as to whether a particular patient who is afflicted with the stroke syndrome is to be subjected to the non-conservative methods of management is a difficult one. This decision must be made jointly by the patient's family, the physician, and on occasion the patient himself. In many instances, this decision must be made to a large extent by the physician in charge.

Guidelines of value in making this decision are: The elderly patient is more likely to accept the possible neurologic deficits; he is more likely to lead the remainder of his life in a happy and productive manner, despite such a handicap. This patient is also less likely to survive the added insult of extensive diagnostic and therapeutic procedures. On the other hand, the younger patient is more likely to have a family less able to accept the patient as a complete or partial dependent. Thus the added risk is more warrented in the younger patient.

The objective evaluation of the clinical response of the

different methods of treating strokes in a statistical manner is hampered by the lack of the knowledge of the natural history of a large series of patients with different cerebrovascular lesions, which would serve as a control. Such a study has been instigated started? to correlate the clinical course of patient with non-hemorrhagic stroke with the various clinical catagories demonstrated by arteriography.[31]

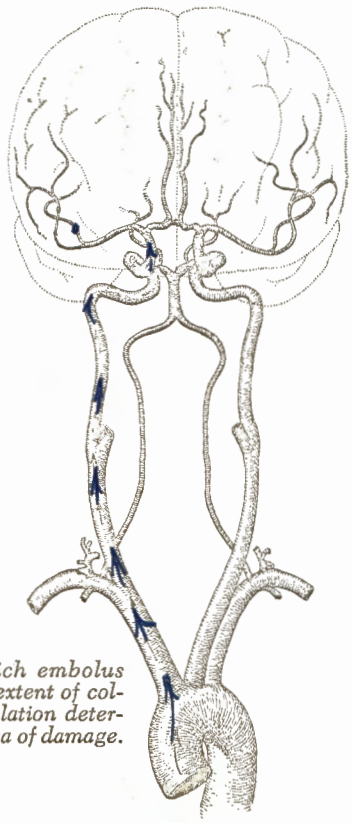
Another important study has been initiated by the International Federation of Neurology to correlate the variations in the severity of degenerative changes in the cerebral blood vessels found at autopsy in relation to sex, age, race, and geographical factors throughout the world.[32] Such a study is of particular importance since it has been well recognized that the degree of cerebrovascular disease does not always correlate with the degenerative changes in vessels elsewhere in the body. With the increasing age of the world population, there is a rapidly increasing number of cases of cerebrovascular insufficiency and it is imperative, therefore, that studies be instituted which might shed light on the causative factors in the degenerative process and the manner in which these changes differ from those occurring in the general systemic vessels.



INTRACEREBRAL HEMORRHAGE

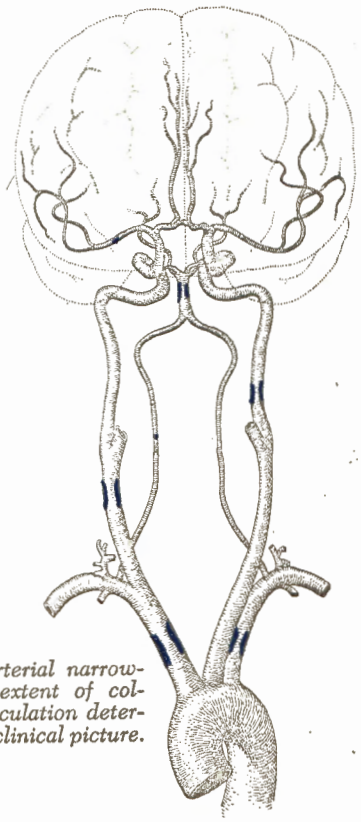
Site of arterial closure and extent of collateral circulation determine the area of damage.

CEREBRAL THROMBOSIS



Site at which embolus lodges and extent of collateral circulation determine the area of damage.

CEREBRAL EMBOLISM



Site of arterial narrowing and extent of collateral circulation determine the clinical picture.

FOCAL CEREBRAL ISCHEMIA

TABLE 1

GOALS AND METHODS OF TREATMENT IN ACUTE PHASE OF ILLNESS

ORGAN SYSTEM	GOALS	METHODS OF TREATMENT
SKIN	Prevention of pressure ulcers	Changing position every two hours Keeping all pressure off reddened or blanched skin Keeping skin dry and clean
RESPIRATORY TRACT	Maintenance of a clear airway	Suctioning; tracheostomy if necessary Drainage of secretions by postural positioning Encouragement of periodic deep breathing and coughing; use of ancillary chemical and mechanical agents as necessary Liquefaction of secretions by adequate hydration and use of aerosols, and by instillation of saline drops if a tracheostomy tube is in place Administration of antibiotics if clinical or x-ray evidence of pulmonary infection is present
CARDIOVASCULAR SYSTEM	Maintenance of adequate blood pressure for perfusion of vital organs	Judicious use of vasopressors and blood transfusions as necessary Preservation of vasomotor tone by early mobilization, including sitting and use of the standing table
	Reduction of peripheral thrombophlebitis	Wrapping of lower extremities with elastic bandages Early mobilization
GASTROINTESTINAL SYSTEM	Maintenance of hydration, electrolyte balance, and anabolism	Administration of adequate fluids, minerals, calories, and vitamins, orally, through gastric tube, or parenterally
	Promotion of normal bowel function	Proper bowel-training program
GENITOURINARY SYSTEM	Assurance of complete urinary elimination	Catheter drainage if necessary
	Reduction of infection, protection and preservation of the upper urinary tract	Adequate hydration and removal of catheter as soon as possible Antibacterial medications
CENTRAL NERVOUS SYSTEM	Avoidance of further depression of CNS	Restriction of sedatives Avoidance of narcotics
	Control of convulsive seizures	Diphenylhydantoin as needed
	Counteracting usual depression	Proper attitude and counseling; use of centrally acting antidepressant drugs
MUSCULOSKELETAL SYSTEM	Maintenance of normal range of joint-motion	Daily exercise Proper positioning with footboards, pillows, and removable splints Avoidance of excessive passive stretching against spastic muscles
	Maintenance of tone of uninvolved as well as affected muscles	Early activity Supervised resistive exercises Use of the standing table

TABLE 3

SPECIFIC GOALS IN PREVENTION AND TREATMENT OF CEREBROVASCULAR ACCIDENTS

TYPE OF STROKE	GOALS		METHODS USED TO ACHIEVE GOALS
ARTERIAL OCCLUSION	Removal of occlusion		Thrombectomy or embolectomy Fibrinolysin
	Reduction of thrombus propagation		Anticoagulant therapy Endarterectomy
	Prevention of formation of new thrombi		Anticoagulant therapy Endarterectomy
	Prevention of formation of new emboli		Conversion of atrial fibrillation to sinus mechanism Antibiotic treatment of bacterial endocarditis Mitral commissurotomy, ablation of left atrial appendage in mitral stenosis Endarterectomy Anticoagulant therapy
	Improvement of blood flow to cerebrum	Enlargement toward normal of arterial channels arising from aortic arch and carotid vessels	Endarterectomy Arterial bypass Vasodilatation
		Correction of pathologic conditions decreasing cardiac output and oxygen-carrying capacity of the blood	Treatment of congestive heart failure and serious arrhythmias Blood transfusions for severe anemia
	Reduction of cerebral edema		Corticosteroids Hypertonic agents intravenously (e.g., urea, hypertonic glucose)
INTRACRANIAL HEMORRHAGE	Establishment of normal blood coagulability		Correction of congenital or acquired coagulation defects Avoidance of anticoagulants if hemorrhage may occur or has occurred
	Reduction of cerebral intra-arterial pressure		Hypotensive drugs Postural position (tilt table) Ligation of, or Salibi clamp on, common carotid artery Ligation of intracranial artery
	Control of ruptured aneurysm		Surgical reinforcement or isolation (depending on anatomic location and accessibility)
	Elimination of hematoma		Surgical evacuation and control of bleeding
	Reduction of oxygen need of brain		Hypothermia

TABLE 2

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