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The Committee hopes to review and update its guidelines periodically as new methods of diagnosis and treatment are developed. Comments, criticisms, and corrections are invited. They should be sent to:

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The following subjects will appear in serial publications in STROKE, although not necessarily in the order listed:
- Epidemiology for Stroke Facilities Planning
- Clinical Prevention of Stroke
- Medical and Surgical Management of Stroke
- Strokes in Children
- Nursing Care of Stroke Patients
- Stroke Rehabilitation
- Laboratory Evaluation of Stroke
- Special Procedures and Equipment in the Diagnosis and Management of Stroke
- Community Health Services for Stroke
- Training, Education, Manpower, and Research

Cross-references will be indicated from time to time to material developed in other sections. Pages will be designated whenever possible, but the sequence of publications will not permit this in many instances. However, the Table of Contents included with each Section should aid in directing the reader to the appropriate pages.
REPORT OF THE JOINT COMMITTEE
FOR STROKE FACILITIES

V. Clinical Prevention of Stroke

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Stroke, Vol. 3, November-December 1972
V. Clinical Prevention of Stroke

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Abstract: V. Clinical Prevention of Stroke

Many risk factors are reviewed in relation to the occurrence of the different types of stroke. The considerations regarded as most important are the identification and successful treatment of hypertension, transient ischemic attacks (TIA), and cardiac disorders. The role of age is well known. The factors of family history, obesity, hypercholesteremia, hyperlipidemia, hyperglycemia, inactivity, and cigarette smoking are not as well documented. The role of oral contraceptives in the production of cerebrovascular thrombosis has not been established definitively. The management of TIA remains controversial at a number of points. Methods are described for identification and management of the stroke-prone individual at several levels of health care delivery, i.e., the teaching medical center, community hospital, physician’s office or clinic, and screening facility. Public education for prevention of stroke is discussed in the context of communication media and various settings.

Introduction

Many factors have been considered as possibly related to the occurrence of strokes, often with minimal evidence, so that to put a specific degree of priority on each is hardly feasible at our present stage of knowledge. The difficulty in discussing risk factors and stroke prevention is compounded when one considers that cerebral infarction due to intravascular occlusion has an entirely different pathogenesis from that of intracranial hemorrhage of any type. In addition, cerebral infarction may be due either to atherosclerotic occlusion of an intracerebral artery or to an embolus originating from an ulcerated atheromatous plaque in an internal carotid artery or from a diseased heart.

Nevertheless, it is clear that the most important considerations, outweighing all others with regard to stroke prevention, are to identify and treat successfully persons with the following disorders: (1) hypertension, (2) transient cerebral ischemic attacks, and (3) certain heart disorders that may be associated with stroke.

Requests for reprints should be directed to: Joint Committee for Stroke Facilities, 1776 K Street, N.W., Suite 1010, Washington, D.C., 20006.

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In the following discussion on stroke prevention, all known risk factors are mentioned. Identification and management of various problems are considered from the perspective of the level within the health care delivery system in which the patient is being evaluated. Initial attention is given to the teaching medical center where all types of expertise for consultation and skills for performance of diagnostic procedures should be available. Discussions of care at other levels emphasize possible differences in approach from that of the teaching medical center.

The sections on screening and public education are aimed at defining both the potential and the limitation of methods to detect persons who may have a greater than average risk of suffering a stroke.

**Management of Risk Factors for Stroke at the Teaching Medical Center**

**Nonembolic Cerebral Infarction**

**Historical**

*Age.* Advancing age is the factor most consistently associated with strokes of all types, a risk rising sharply in later years of life. There is no way of altering this effect, which presumably results from progressive degenerative changes in blood vessels of all types throughout the body.

*Family History.* Information concerning genetic predisposition as a risk factor for stroke is incomplete. Several studies indicate a higher mortality from vascular disease among the parents of patients with strokes as compared with controls. Studies among twins show a significantly (<0.05) higher concordance rate for "cerebral apoplexy" in monozygotic than in dizygotic twins. However, information is still insufficient to judge the extent to which inheritance may influence the risk for various categories of stroke.

*Transient Ischemic Attacks (TIA).* Episodes of transient focal neurological deficit lasting no longer than 24 hours, and probably due to cerebral ischemia, are known as TIA or transient cerebral ischemic attacks. These events usually come to an end in less than an hour and may be the result of ischemia in the retina, in either cerebral hemisphere, in the brain stem, or in the cerebellum. Manifestations may include sudden transient blurring or loss of vision in one eye, weakness or numbness of one side of the body, difficulty with speech, vertigo, diplopia, ataxia, or any combination of these symptoms. Less specific complaints, such as dizziness, fainting, or unexplained loss of consciousness, are not acceptable by themselves as diagnostic of TIA.

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 or over one eye, altered arterial pulsations, or decreased retinal artery pressures, only occasionally will angiography reveal pathology amenable to operative intervention. However, when such a person with TIA does not respond satisfactorily to medical management, arteriography is an appropriate aid to further diagnosis and therapy.

Physical Inactivity. No documented evidence exists that lack of physical activity is a risk factor for stroke. However, because there may be a greater incidence of ischemic heart disease in sedentary persons, and because cardiac disorders are associated frequently with an increased incidence of stroke, further consideration of this possible hazard is justified.

Cigarette Smoking. Data from the Framingham study indicate that cigarette smoking increases the risk of cerebral infarction threefold (see: Section on Epidemiology for Stroke Facilities Planning. Stroke 3: 360-371 [May-June] 1972). However, other investigations have not confirmed this relationship. Because ischemic heart disease, which is associated with the use of cigarettes, does increase the occurrence of stroke, smoking should be considered at least as a secondary risk factor.

Physical Examination

Hypertension. Although information is incomplete at this time, hypertension appears to be the most important alterable factor associated with nonembolic cerebral infarction. From studies available to date (see: Section on Epidemiology for Stroke Facilities Planning. Stroke 3: 360-371 [May-June] 1972), strokes are from two to four times as frequent in hypertensive as in comparable normotensive individuals. There is no evidence of a sharp cutoff point to define hypertension. However, actuarial data indicate that deaths from vascular disease of the nervous system increase as the blood pressure elevates, starting with the lowest levels at which hypertension may be suspected. Although diastolic pressures seem no more reliable than systolic as indicators of risk for stroke, no reliable information concerning persons with systolic hypertension in the absence of diastolic elevation is presently available.

Despite the fact that published surveys show hypertension to be a common disorder in the general population, the condition is untreated in a large percentage of these persons. A study of one county in Georgia with 55% whites and 45% blacks revealed that 20% of persons over age 15 had blood pressures exceeding 160 systolic and 95 diastolic. Seventy percent of those found to have high blood pressure were not receiving any treatment, and only 9% of the hypertensive group were well controlled by drug or other therapy.

A logical question is whether control of existing hypertension lowers subsequent stroke incidence. Both the Veterans Administration Cooperative Study of moderate hypertension and an English investigation have demonstrated significantly more new strokes or recurrent attacks in untreated patients as compared to treated patients. Further extension of the former study indicated that treatment of mild hypertension (<105 diastolic) also resulted in fewer initial strokes of all types.

Sufficient evidence has accumulated to indicate that hypertension is not only a major cause of stroke but also the most important treatable risk factor for this condition. Extensive drug trials in hypertensive populations aimed at preventing cerebral hemorrhage and infarction are justified.

Induced Hypotension. An excessive or prolonged fall in blood pressure from any cause, including shock, severe hemorrhage, surgery, dehydration, medications, and diagnostic procedures, may lead to general cerebral ischemia. In the presence of cerebral arterial disease, the reduction of blood flow may become most apparent in an area served by a focally compromised vessel. During procedures which may cause hypotension, careful monitoring of blood pressure is important.

Cervical Carotid Bruit. A well-localized bruit at the cervical carotid bifurcation, or extending
upward from that point, is usually due to stenosis of the common, internal, or external carotid artery in the neck. Vascular sounds heard over the entire cervical common carotid artery are usually transmitted from a proximal source or they are of hemodynamic origin and unassociated with local narrowing (see: Section on Strokes in Children).

A localized cervical carotid bruit with both systolic and diastolic phases often is associated with a high degree of arterial stenosis and correspondingly reduced retinal artery pressure. Variations in the character or intensity of a localized bruit may imply precarious flow through a severe stenosis, and can be caused by changes in head position or by alterations in hemodynamics at the lesion site secondary to thrombotic material.

Many authorities recommend no further studies or treatment for an asymptomatic patient with normal or even decreased ipsilateral retinal artery pressure who has a localized systolic bruit over the carotid bifurcation. However, other physicians advise angiography and surgery in selected individuals with asymptomatic carotid bruit, particularly those in whom major surgical procedures are contemplated elsewhere in the body. At present, data are insufficient to indicate the risk of stroke in these patients.

Regardless of the retinal artery pressure, if a patient with TIA has a localized systolic bruit, then many physicians believe that angiography should be carried out to determine the location and extent of the cervical carotid artery stenosis, and whether there is an ulcerated atherosclerotic plaque. This diagnostic test should be performed by a person experienced in such procedures and should be designed to visualize adequately both the extracranial and intracranial carotid artery circulations (see: Section on Special Procedures and Equipment in the Diagnosis and Management of Stroke).

The evidence concerning treatment of unilateral carotid stenosis by surgical endarterectomy is incomplete. However, if this operation is performed by an experienced team with full understanding of the cerebral circulation, physiology, and anatomy related to the procedure, the combined risk of death and serious neurological signs is no more than 1% or 2% in the patient who originally had no focal neurological deficit.

Some authors contend that patients with TIA respond favorably to long-term anticoagulant therapy even though physical findings due to unilateral carotid stenosis are present. They believe that if the diagnosis is certain and anticoagulant treatment is chosen as the means of management, arteriography usually is unnecessary.

At this time there are no universally accepted guidelines as to which patients with carotid stenosis and TIA should be treated by endarterectomy, by anticoagulants, or by neither. In general, surgery is indicated most strongly for: (1) symptomatic patients with well-localized unilateral carotid artery stenosis, (2) those having contraindications to anticoagulant therapy, and (3) persons with a bruit which is changing in pitch and intensity. Age is not necessarily a deterrent to surgery if the patient is in good general condition. Many surgeons operate on patients with bilateral carotid stenosis or with stenosis on one side and occlusion on the other. In the latter case, endarterectomy is performed only on the stenosed vessel. Agreement is general that surgery should not be advised for patients with completed strokes and significant neurological residuals, nor should operation be attempted on occluded internal carotid arteries.

In the opinion of some authorities, anticoagulant therapy is usually preferred when patients are relatively old, have widespread atherosclerotic disease, suffer from some life-threatening disorder, or are being treated in locations where teams experienced in angiography and vascular surgery are not readily available. Anticoagulants should be given only in circumstances where the prothrombin time can be monitored carefully. Caution should be exercised in prescribing anticoagulants for patients who are demented or confused, or who are unlikely to follow instructions. When the internal carotid artery is occluded, thromboendarterectomy is usually not recommended, because even if flow can be re-established, there is risk of distal embolization. In this circumstance, anticoagulant therapy is appropriate for patients with TIA. If the occlusion is found incidentally in an asymptomatic patient, no treatment is needed.

Orbital Bruit. A bruit over one eye, if not associated with an arteriovenous anomaly, is generally due to stenosis of the intracranial portion.
of the internal carotid artery. In the absence of associated symptoms, no treatment is indicated, but if TIA are present, the therapeutic principles discussed previously should be followed.

**Difference in Brachial Artery Blood Pressures.** A difference of more than 10 mm Hg between right and left brachial artery blood pressures or a decrease in one radial pulse may indicate stenosis or occlusion of the corresponding subclavian artery. If the patient is entirely asymptomatic, continued observation at regular intervals without diagnostic study or treatment is appropriate.

**Evidence of Generalized Atherosclerosis.** When evidence of atherosclerosis is found elsewhere, its presence in cervical or intracranial arteries should be suspected. However, there is no evidence at present that altered pulses or other signs of peripheral vascular disease indicate an increased incidence of stroke. The high stroke risk in patients with cardiac disease is primarily a result of emboli; these may occur early after myocardial infarction, or at any time in persons with valvular disease and/or cardiac arrhythmia, especially atrial fibrillation. In addition, there is some evidence that nonembolic cerebral infarction is more frequent in persons with coronary artery disease even in the absence of the above-named factors.

**Decreased Retinal Artery Pressure.** Retinal artery pressure measurements are unreliable unless done by a trained and experienced observer, usually an ophthalmologist or neurologist (see: Section on Special Procedures and Equipment in the Diagnosis and Management of Stroke). If reliably determined, a reduced pressure is substantial evidence that the ipsilateral internal carotid artery is stenosed or occluded. This finding in an asymptomatic patient who has no cervical bruit does not require further immediate attention, but if bruits or TIA are present, treatment may be desirable (see: Transient Ischemic Attacks [TIA], Cervical Carotid Bruit).

**Retinal Emboli.** The retinal emboli most commonly observed are shiny crystals of cholesterol esters, commonly called cholesterol emboli; they are definite evidence of an ulcerated atherosclerotic lesion in a larger vessel proximal to the ophthalmic artery. More than 80% of persons with this finding have other evidence of cardiovascular or cerebrovascular disease either by history or by physical examination. However, cholesterol emboli are seen occasionally in asymptomatic patients who are otherwise normal, and they usually do not cause overt clinical signs of retinal ischemia. There are no data to indicate the risk of stroke in these persons.

Platelet fibrin emboli in retinal arterioles, either with or without associated cholesterol emboli, also may arise from an ulcerated plaque. They may occur more commonly than cholesterol emboli, but are not observed as often. They are the kind of emboli usually seen when ophthalmoscopy can be done during a transient episode of monocular blindness (amaurosis fugax). Should these emboli originate from a source proximal to the ophthalmic artery, they may occlude cerebral vessels as well; frequency of stroke under these circumstances is unknown.

Treatment for cholesterol emboli is directed toward an associated arterial lesion, usually in the internal carotid vessels. If retinal ischemia also is present, management should be the same as that discussed under TIA with and without carotid stenosis (see: Transient Ischemic Attacks [TIA]).

**Obesity.** Although obesity has long been considered as associated with hypertension, and the latter with stroke, available evidence indicates no increased risk of cerebral ischemia in obese persons in the absence of other risk factors.

**Laboratory Examinations** (See: Section on Laboratory Evaluation of Neurovascular Disease [Stroke]. Stroke 3: 503-526 [July-Aug 1972].)

**Elevated Serum Glucose.** Some authorities have suggested that cerebrovascular disease is no more frequent in diabetic than in normal persons, an opinion based to some extent on evidence from certain prospective studies. However, clinical experience and prospective data from the Framingham study indicate an increased risk of cerebral infarction with even modest impairments of glucose tolerance.
Elevated Serum Lipids. The Framingham study indicates that elevated serum cholesterol in persons under age 50 years moderately increases the risk of ischemic stroke. Although there is no evidence to date that lowering blood lipids lessens this risk, an attempt to reduce high lipid levels in younger persons by dietary means, or by drugs if diet fails, is appropriate. Elevated serum cholesterol found initially beyond 50 years of age, and high triglycerides at any age seem to be unassociated with the occurrence of cerebral infarction. However, treatment of these conditions might be necessary on the basis of other medical considerations.

Increased Blood Volume or Viscosity. The most rapid and immediate control of polycythemia vera, a disease which may be complicated by cerebral thrombosis, is achieved through phlebotomy. Other methods of treatment are discussed in texts on hematology. Some authorities believe that an elevated hematocrit value without polycythemia vera indicates a predisposition to intra-arterial thrombosis; this problem, when due to dehydration, can be prevented by the prompt administration of fluids.

Anemia. Low hemoglobin and/or red blood cell counts are unusual causes of ischemic stroke unless associated with existing focal vascular occlusion in the cerebral circulation. In these circumstances, generalized cerebral ischemia may be manifest overtly in the territory of the affected vessel, and is prevented by treatment of the anemia. The problem of sickle cell anemia is receiving emphasis and should be kept in mind as a risk factor for stroke in the black population (see: Sections on Laboratory Evaluation of Neurovascular Disease [Stroke]. Stroke 3: 503-526 [July-Aug 1972; Strokes in Children).

Abnormal Electrocardiogram (EKG). Prospective studies indicate that any EKG abnormality moderately increases the risk of ischemic stroke. When abnormalities of the Q wave or ST segment are present, this risk becomes much more definite. At present, the only known way of preventing cerebral infarction secondary to heart disease showing EKG changes is by the use of anticoagulants (see: Embolic Cerebral Infarction).

Abnormal Serological Test for Syphilis. Syphilitic vascular disease has long been known to cause ischemic strokes. Prevention is accomplished by prompt treatment of patients with an abnormal serological test for syphilis unless they have already received adequate amounts of penicillin.

Other Laboratory Procedures. The diagnosis of TIA is made by the history and the occasional observation of an attack by the physician; during the intervals between episodes, examination reveals no evidence of neurological disease. To find possible causes for these symptoms, and to rule out other conditions which might mimic TIA, the following diagnostic procedures are available (see: Section on Special Procedures and Equipment in the Diagnosis and Management of Stroke):

Electroencephalographic tracings are normal in patients with TIA unless there is clinically undetected minor residual infarction. Focal seizure activity generally means that the transient symptoms are of epileptic, not ischemic, origin.

Radioisotope brain scans, either by the rectilinear method or by the gamma camera, are normal in patients with TIA unless there is residual undetected infarction.

Skull x-rays may show calcification of the carotid artery near the sella turcica. If these calcifications are dense, they may indicate the presence of stenosis. Alternative causes for transient neurological symptoms should be considered when additional abnormalities, as prominent vascular channels or other types of calcification, are present.

Cerebrospinal fluid and echoencephalographic abnormalities are usually due to causes other than TIA.

Other Factors

Antihypertensive Agents. As noted previously (see: Induced Hypotension), excessive blood pressure drops may cause generalized cerebral ischemia and focal ischemic symptoms, a possible danger in treatment of hypertension. Persons receiving antihypertensive drug therapy should have periodic blood pressure determinations made in both the supine and standing positions. Ischemic stroke due to antihypertensive drugs is rare.
Oral Contraceptives. Numerous reports claim a greater than normal occurrence of cerebral infarction in young women taking oral contraceptives. However, at the present time no evidence has been published to establish that ischemic arterial strokes occur with any greater incidence in women taking oral contraceptives than in the general population of young women.

EMBOLIC CEREBRAL INFARCTION

Historical
Embolic cerebral infarction usually presents as a single, abrupt, monophasic event reaching its peak in minutes, but sometimes this condition develops more slowly and in a deliberate manner.

Recent advances in the prophylaxis and therapy of cardiovascular diseases have changed the incidence of stroke associated with these disorders. For instance, cerebral infarction following the insertion of a prosthetic heart valve is one of the common neurological problems now encountered by cardiovascular specialists. Most authorities agree that the frequency of strokes from this source can be reduced with postoperative anticoagulant therapy. In addition, the use of prophylactic antibiotics has reduced the incidence of rheumatic heart disease and subacute bacterial endocarditis and secondarily, therefore, has reduced the incidence of cerebral embolism.

Physical Examination
White calcific emboli originating from calcium deposits on the aortic valve may be detected in retinal arteries. They usually are asymptomatic, and rarely lead to visual or neurological deficits. Their discovery, however, should alert the observer to the probable presence of calcific aortic stenosis.

Palpation of the precordium may give significant information about the possible sources of cerebral emboli. A diffuse right ventricular lift or a palpable pulmonary outflow thrust may indicate the presence of mitral valvular stenosis. A paradoxical apical rocking motion may be the only finding associated with left ventricular aneurysm, a common precursor of embolic stroke. A presystolic filling wave (double apical pulse), felt best in the left lateral decubitus position, indicates that the diastolic left ventricular resistance is elevated and that cardiac function is impaired. The anterior chest wave transmitted from atrial contraction is absent in atrial fibrillation. Cardiac conditions in which a presystolic filling wave may be observed and which are associated with a significant risk of emboli to the brain include aortic valvular disease, left ventricular failure, and cardiomyopathy.

A diastolic apical rumble, heard in left atrioventricular (mitral) valve abnormalities, is due either to mitral stenosis or, much more rarely, to a left ventricular myxoma, both of which are possible causes of embolic cerebral infarction. About one-third of all cases of rheumatic mitral valve disease in which postmortem examination is performed have evidence of systemic thromboembolism, with cerebral infarction occurring in approximately half of these. Severe, and even moderate, mitral stenosis may cause left atrial thrombosis, especially when atrial fibrillation also is present. Embolic strokes result more frequently from mitral stenosis than from aortic stenosis and mitral insufficiency.

When a patient with mitral stenosis and a history of thromboembolic disease, atrial fibrillation, and symptoms of pulmonary vascular congestion has evidence of valve pliability (an opening snap), a mitral commissurotomy and cardioversion to a sinus rhythm should be done. In the event that the patient has calcific stenosis of the mitral valve, replacement of the valve with a prosthesis should be considered, although the risk for stroke remains following valve replacement. Carefully administered anticoagulant therapy may be indicated in patients with calcific or noncalcific mitral stenosis and atrial fibrillation.

Left ventricular failure, a condition often causing thromboembolism in bedridden patients, may cause a diastolic gallop sound occurring during rapid ventricular filling. Short-term anticoagulant therapy should be considered in these cases.

Delayed left ventricular emptying may be suspected upon hearing a paradoxical splitting of the second heart sound; this condition, which occurs with disturbances of conduction through the left bundle and with myocardial disease and decreased cardiac output, is found most often in painless myocardial ischemia, acute angina pectoris, or chronic left ventricular failure. When paradoxical splitting is present, in the absence of a left bundle branch block, frequency of endomural thrombosis may be increased.
CLINICAL PREVENTION OF STROKE

The highest correlation between embolic strokes and prior cardiac rhythm disturbance occurs with atrial fibrillation. Friedman et al. noted that strokes were six times as frequent in patients with chronic atrial fibrillation as in a control group. Those with intermittent atrial fibrillation also are stroke-prone. Continued anticoagulant therapy should be considered for all patients with long-standing atrial fibrillation, especially in the presence of valvular cardiac disease.

While ventricular and atrial gallop sounds, paradoxical splitting of the second heart sound, tachycardia, and pulmonary rales are physical signs of left ventricular failure familiar to most physicians, cardiac overactivity is a less well known but subtle and important clue to the presence of a poorly functioning ventricle that may contain endomural thrombi. The latter condition is appreciated when the observer palpates the chest and notes that the heart is overactive.

When an acute myocardial infarction occurs, there are no specific detectable physical abnormalities indicative of endomural thrombi, unless the patient presents with clinical evidence of an embolus to the brain or elsewhere. Although anticoagulant therapy may decrease the frequency of strokes during acute infarction of the heart, this form of treatment may be without benefit, or even contraindicated, if hemiparesis or other focal neurological disorder already has occurred. Myocardial infarction is recognized occasionally after the occurrence of a stroke. Furthermore, as left ventricular contractility increases during recovery from this condition, cerebral emboli can appear. Despite the fact that left ventricular thrombi were found in 44% of autopsied cases of myocardial infarction, in only 2% of these instances were strokes the primary cause of death.

Persons with prosthetic mitral valves, who are receiving anticoagulant therapy, have a 35% cumulative frequency of thromboembolic disease in a five-year period; most commonly the lesions are cerebral infarcts. These patients are less responsive to anticoagulant therapy than are those with aortic valve prostheses who, if not given anticoagulant therapy, also will have a 35% cumulative frequency of thromboembolism. In the latter group, however, well-regulated anticoagulant therapy (sufficient to prolong the prothrombin time in seconds to approximately two and one-half times the control value) will reduce the frequency of complications to 5%. Therefore, continued, well-controlled anticoagulation is vital for all patients with Starr-Edward or similar prosthetic valves; when human homograft valves are used, this type of treatment is unnecessary.

Even with modern antibiotic therapy, approximately 25% of patients with bacterial endocarditis have major neurological symptoms and one-third of these die of cerebrovascular and cardiac complications. Bacterial endocarditis usually produces small cerebral emboli which cause infarction, mycotic aneurysms, hemorrhage, or abscess. Most authorities believe that anticoagulant therapy may have an adverse effect on patients with active bacterial endocarditis. Antibiotic prophylaxis is essential in all patients with significant cardiac murmurs (including those arising from ventricular septal defects) and prosthetic valves, particularly prior to and after all dental procedures, and with respiratory, urinary tract, and other infections.

Laboratory Examination
An abnormal EKG, as broadly defined in the Framingham study, may occur in 85% of patients with acute stroke. Confirming the presence of a myocardial infarction, especially in the stroke patient, may be difficult, but daily 12-lead EKG often are helpful. EKG tracings made at previous times should be sought for comparison. If atrial fibrillation or flutter is present, cardioversion should not be performed, except occasionally for control of acute left ventricular failure, as the reversion of cardiac rhythm may cause further embolization. In patients with embolic strokes from associated myocardial infarction, intravenous heparin, which can be given conveniently through a plastic needle with a stylet placed in a peripheral arm vein, should be administered every four hours if there are no contraindications, such as evidence of intracerebral hemorrhage.

Cardiomegaly alone does not warrant long-term administration of anticoagulants, but this treatment is justified when ventricular aneurysm, cardiomyopathy, or chronic atrial dysrhythmias also are present.
**Joint Committee for Stroke**

**Procedures**

**Elective Cardioversion.** To prevent thromboembolism, many authorities believe anticoagulants should be administered for three weeks prior to elective cardioversion of atrial fibrillation.

**Cardiac Angiography and Catheterization.** A review of central nervous system complications associated with cardiac angiography and cardiac catheterization revealed that focal cerebral complications are usually secondary to displacement of clots or atheromatous material from the heart or aorta. Only ten instances of cerebral embolic infarction were noted among 12,367 patients. The injection of contrast media per se did not appear to be a significant cause of embolic cerebral infarction, although in a few patients hyperosmolality from this material or from fluid loss appeared to contribute to the development of stroke. The risk of stroke is moderately increased in patients with ventricular failure undergoing left heart catheterization.

**Cardiac Surgery.** Under a number of circumstances, cerebral embolization may occur during cardiac surgery, either directly from the heart, or as a consequence of material accumulating in the blood during cardiac bypass. Some authorities recommend using special filters for the blood during bypass procedures, but these devices have not had universal acceptance by cardiac surgeons. A detailed discussion of techniques used to prevent embolization during cardiac surgery is inappropriate here.

**Cerebral Venous Thrombosis**

The role of infection in causing cerebral venous thrombosis is well documented. Thrombotic occlusion of dural venous sinuses or their parenchymal cerebral venous tributaries accounts for less than 1% of thromboembolic strokes. More than half are the consequence of local suppuration (mastoiditis, meningitis, brain abscesses), while the second most common cause is cachexia and dehydration accompanying terminal illness. Other etiological factors include head trauma, particularly penetrating injuries from automobile accidents or war wounds, and operative craniotomy. Blood dyscrasias, especially polycythemia, the leukemias, and the systemic thrombotic states associated with certain abdominal carcinomas (notably pancreatic), are occasional causes. Dural sinus thrombosis is also a well-recognized postpartum complication, occurring generally two to eight days after delivery.

In Aronson's series of 38 autopsied cases at Kings County Hospital (written communication, September 1970), all had "associated lesions," mostly in the categories noted above. One each of bronchial asthma, bronchopneumonia and diabetes, and a gunshot wound of the neck were more remotely associated diseases.

Johnson et al. described cerebral venous thrombosis in one patient with paroxysmal nocturnal hemoglobinuria, noting that, "Although many patients with PNH apparently die as a result of cerebral venous thrombosis, this has only rarely been documented in the literature."

Puerperal thromboembolism is three times more common among women whose lactation is suppressed by estrogens than among those who breastfeed, according to Daniel et al. and Jeffcoate et al.. Millar and Littlepage emphasized differences in smoking habits among such patients: 28% of breastfeeding smokers as opposed to 37% of those with suppressed lactation. For women over age 30, these frequencies were 18% and 77%, respectively. The last authors thought that this factor should be considered before ascribing the cause of puerperal thromboembolism to oral contraceptives.

Regarding the use of estrogens per se, Atkinson et al. described five autopsied cases of cerebral venous thrombosis in women aged 23 to 49 years who had been taking estrogens before the onset of sudden illness; a sixth fatality was documented as an addendum to their report. They also recorded a 23-year-old who survived cavernous sinus thrombosis. No other similar cases were reported to the Committee on Safety of Drugs for Great Britain over a five-year period, but it is important to note that the diagnosis was unsuspected antemortem in all instances and the condition would have remained undiscovered without autopsy. Sissons and Hall reported a 22-year-old woman who survived presumptive cortical venous thrombosis which occurred while she was taking oral contraceptives.

Thus, it is not certain at this time whether postpartum cerebral venous thrombosis (as...
opposed to systemic venous thromboembolism) can be related validly to breastfeeding, cigarette smoking, or estrogen usage.

The following factors seem important in recognizing and/or preventing cerebral venous thrombosis:

1. Avoidance of dehydration and hypercoagulable states, particularly at the extremes of age,
2. Appropriate management of polycythemia,
3. Recognition of abdominal malignancy leading to diffuse thrombosis,
4. Early antibiotic treatment for all penetrating head injuries and purulent infections of middle ear, paranasal sinuses, and other paranasal structures; appropriate attention to dental sepsis and cervicofacial furunculosis,
5. Recognition that purulent meningitis, brain abscess, metastatic brain tumor, or metastases to skull or meninges all may cause cerebral venous thrombosis, and
6. Appreciation of the possibility of a real risk for systemic thromboembolism with the use of oral contraceptives; possibly the cerebral veins may be susceptible also, though available data are insufficient to quantify this risk.

Recognition of the likely precipitants, and early diagnosis and treatment of the condition itself could serve to reduce greatly the morbidity and mortality from cerebral venous thrombosis, and

Primary Intracerebral Hemorrhage

Introduction

Since this discussion is concerned principally with the prevention of primary cerebral hemorrhage, presently untreatable risk factors such as racial predilection and increasing age are not discussed further. The best evidence concerning the frequency of cerebral hemorrhage suggests that it comprises some 10% to 15% of new strokes, or an annual incidence rate of less than 25 per 100,000 population.\(^{22}\)

The term "primary cerebral hemorrhage" excludes other causes of bleeding into the brain such as trauma, hemorrhagic tendencies (drug-induced or due to blood dyscrasias), saccular aneurysms, arteriovenous malformations, and brain tumors. Margolis et al.\(^{33}\) have called attention to minute intracerebral vascular malformations or hamartomas as a cause of massive intracerebral hemorrhage. Goldberg and Leeds\(^{34}\) presented five instances of spontaneous intracerebral hemorrhage from ruptured collaterals of occluded cerebral arteries.

Hypertension and Cerebral Hemorrhage

In Rochester, Minnesota, the stroke experience for the years 1945 to 1954 has been summarized by Whisnant et al.\(^{26}\) Hypertension, defined as a diastolic pressure of 90 mm Hg or more, was present before the hemorrhage (within five years) in 62% of those with infarcts, 74% of those with subarachnoid hemorrhage, and 89% of those with cerebral hemorrhage.\(^{3-15,21}\) Dyken\(^{36}\) found hypertension in 30 of 40 patients with cerebral hemorrhage (17% of his stroke patients), five times the frequency in his matched controls. Both series had a devastating fatality of 83%, occurring within one month in Rochester, and while the patient was still hospitalized in the Indiana group. In Göteborg, Sweden, Aurell and Hood\(^{37}\) asserted that not one patient in their large series of fatal cerebral hemorrhages had received adequate hypertensive therapy. They claimed a significant decline in cerebral hemorrhage death rate over the decade when antihypertensive agents came into wide usage. However, in examining these statistics, Kurtzke\(^{38}\) found that there were no valid mortality differences between the decade in question and previous ten-year periods for any age group or for the total. Other studies also have claimed similar changes in mortality over time, but evidence is insufficient to document any real decline in cerebral hemorrhage death rates.\(^{39}\)

The Framingham study has provided valuable data on precursors of vascular disease. For stroke as a whole, Kannel et al.\(^{28}\) record a strong positive correlation between initial blood pressure levels and later cerebrovascular disease. The most recent information from this study\(^{40}\) indicates that hypertension preceded both cerebral hemorrhage and infarction with equal frequency. In a group of patients who were hypertensive at initial examination, six cerebral hemorrhages occurred subsequently when 2.9 were expected.
Among those initially normotensive, there were nine cerebral hemorrhages, with 12.1 anticipated (morbidity ratio 74). For those with infarcts, the morbidity ratios were 183 for hypertensives (16 observed versus 8.8 expected) and 64 for the normotensives (23 observed versus 30.2 expected). It is noteworthy that the absence of hypertension as previously defined for this study does not preclude the occurrence of stroke.

From the data of the Build and Blood Pressure Study of the Society of Actuaries, Stamler showed that with progressively elevated blood pressure levels, the risk for strokes of all types increases concomitantly. There were four times as many stroke deaths in hypertensive as in normotensive males.

Hypertension has a high prevalence in the population at risk for stroke. According to a Baltimore survey, 21% of persons age 35 to 64 and 41% of those age 65 and over had high blood pressure. The first report of the Veterans Administration Study on Antihypertensive Agents has substantiated that treatment of severe hypertension will, in fact, prevent its complications, about one-third of which are cerebrovascular disease. The second report showed that even less severe hypertension—in those who present to hospital with chiefly cardiovascular symptoms—also was amenable to therapy, and treatment averted the usual consequences (including stroke). This was a randomized, prospective, double-blind study, the results of which are summarized below. There were 20 strokes in the controls and five in the treated patients.

<table>
<thead>
<tr>
<th>Stroke Type</th>
<th>Control</th>
<th>Treated</th>
</tr>
</thead>
<tbody>
<tr>
<td>Fatal cerebral hemorrhage</td>
<td>3</td>
<td>0</td>
</tr>
<tr>
<td>Fatal subarachnoid hemorrhage</td>
<td>1</td>
<td>0</td>
</tr>
<tr>
<td>Fatal cerebral thrombosis</td>
<td>3</td>
<td>1</td>
</tr>
<tr>
<td>Nonfatal subarachnoid hemor-</td>
<td>1</td>
<td>0</td>
</tr>
<tr>
<td>rhage</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Nonfatal cerebral thrombosis</td>
<td>4</td>
<td>0</td>
</tr>
<tr>
<td>“Severe”</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Other cerebral thrombosis</td>
<td>8</td>
<td>4</td>
</tr>
<tr>
<td>or TIA</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Total with stroke</td>
<td>20</td>
<td>5</td>
</tr>
</tbody>
</table>

Information is lacking as to whether strokes can be prevented by treating asymptomatic hypertensive patients discovered at casual examination or in a population survey. However, at present, it appears justified to recommend permanently maintained antihypertensive treatment (if tolerated) for all ages when elevated blood pressure is found, in the hope of preventing primary intracerebral hemorrhages and other strokes.

**Risk Factors Other than Hypertension**

Hypertension is the only condition generally accepted as a risk factor in hemorrhagic stroke. Other factors may be unrecognized, however, because cases are relatively few—from 10% to 15% of all strokes. In addition, data are incomplete since some studies have not attempted to differentiate the several types of stroke. There are, however, no published claims for risk factors in cerebral hemorrhage other than high blood pressure, blood dyscrasias, age, and race.

**Subarachnoid Hemorrhage**

There are no known ways to select from the general population persons who are likely to have subarachnoid hemorrhage. Occasionally, one or more unruptured saccular aneurysms are found in patients who have had angiography performed for some other reason. Even if there were an absolutely safe angiographic technique to detect intracranial aneurysms or arteriovenous anomalies, it still would be impossible to predict which of the lesions are likely to bleed.

However, some patients with serious subarachnoid hemorrhage will have had previous minor bleeding episodes, often manifested only by the abrupt onset of a generalized or occipitocervical headache and mild nuchal rigidity, and occasionally by an isolated third nerve palsy. If these patients are seen within a few days after the onset of the above symptoms, cerebrospinal fluid examination is appropriate to detect the presence of blood or xanthochromia.

Focal seizures without other obvious cause may be the first symptom of an arteriovenous malformation. However, without previous subarachnoid bleeding, the lesion may be unsuspected. Evaluation of patients with focal seizures should include a careful neurological examination, skull x-rays, EEG, brain scan, and probably an arteriogram.

Unless an intracranial aneurysm is large enough to cause direct pressure on a cranial nerve or some other intracranial structure, evidence of its presence cannot be detected on physical examination. Cranial bruits are rarely,
If ever, associated with intracranial aneurysms but may be heard over the skull or eyes when there is an arteriovenous malformation. Since spinal cord vascular malformations also may present with subarachnoid hemorrhage, diagnostic efforts should be directed to this site if intracranial studies are negative.

Neither physical nor laboratory examinations can predict the probability of subarachnoid hemorrhage, even when there is a known lesion such as an arteriovenous malformation. Patients with bleeding tendencies or those taking anticoagulant drugs may develop subarachnoid hemorrhage, but in these conditions subdural and intracerebral hematomas are encountered more commonly than subarachnoid hemorrhage.

**Management of Risk Factors for Stroke at the Community Hospital**

**INTRODUCTION**

For this discussion, the community hospital is defined as a general medical and surgical facility operating without the services of interns, residents, or medical students, but designed to meet the needs of a patient referral area. This category includes facilities ranging in capacity from 50 to 500 or more beds.

The principles basic to the prevention of stroke are detailed elsewhere (see: Management of Risk Factors for Stroke at the Teaching Medical Center). The application of these principles, however, will vary considerably, depending upon the levels of professional and supportive skills, and the facilities available at a given location.

**Nonembolic Cerebral Infarction**

"Stroke proneness" should be a consideration in evaluating all patients in a community hospital regardless of diagnosis or reason for admission.

A detailed family history concerning possible stroke-related deaths in relatives should be obtained. Because treatment of even mild hypertension reduces the incidence of stroke, physician and patient education is necessary, particularly at the community level, in order to emphasize the importance of constant vigilance to detect and treat high blood pressure. Hospital personnel should be prepared to screen all patients for hypertension, obtaining blood pressure readings several times daily during different physical and emotional states so that a true blood pressure baseline may be established.

Auscultation for bruits should be a part of every physical examination as discussed elsewhere (see: Cervical Carotid Bruit; Orbital Bruit). In addition, blood pressure measurements in both upper extremities should be made, atherosclerosis should be sought in other portions of the body, and ophthalmoscopy should be performed with particular attention to retinal emboli, since these suggest a possible ulcerated plaque in proximal blood vessels. The further evaluation of any abnormal finding depends upon a history or identification of TIA.

Routine laboratory examinations can aid in detecting stroke-prone patients. Fasting and two-hour postprandial blood glucose determinations are indicated for all patients because diabetes is related to the development of vascular disease, and a relatively large number of asymptomatic persons have elevated blood glucose levels. If serum cholesterol and triglyceride levels are abnormal, phenotyping and subsequent appropriate dietary or medical management should be instituted. Polycythemia and anemia occasionally may be associated with cerebral ischemia and often can be suspected or identified by means of routine blood counts. The urinalysis and blood urea nitrogen may show abnormal values in renal disease associated with hypertension. A serological test for syphilis should be obtained to exclude luetic vasculitis.

Attention should be given to the possible dangers of hypotension related to anesthetic procedures or induced by antihypertensive drugs.

Patients should be questioned regarding the occurrence of focal neurological dysfunction whether fixed or transient. Particular attention should be paid to complaints of transient neurological abnormalities compatible with TIA (see: Transient Ischemic Attacks [TIA]). Upon encountering this type of history, the diagnostic procedures discussed previously should be used to assist in differential diagnosis. However, tests are inadequate substitutes for experienced clinical judgment, and neurological consultation, including advice obtained by telephone, should be utilized in reaching a decision.
Clinical and laboratory evaluations permit the identification of most patients with TIA (see: Section on Laboratory Evaluation of Neurovascular Disease [Stroke]. Stroke 3: 503-526 [July-Aug] 1972). If the diagnosis is established on a clinical basis in patients without associated physical findings, as a localized bruit, altered pulses or decreased retinal artery pressures, medical therapy may be pursued, even though cerebral angiography has not been performed (see: Transient Ischemic Attacks [TIA]). If the diagnosis is in doubt, if TIA occur despite appropriate medical therapy, or if there are physical signs of extracranial carotid disease, it is proper to perform angiography locally if experienced personnel and facilities are available. Otherwise, the patient should be referred to a medical center where personnel have the required diagnostic experience and where these procedures can be done with less risk.

Many authorities believe that a significant percentage of patients with TIA can be diagnosed adequately and treated medically following a clinical evaluation, and that angiography and consideration for vascular surgery become necessary in a relatively small proportion of cases. Other authorities, however, have recommended that angiography be performed in all patients thought to have TIA.

EMBOLIC CEREBRAL INFARCTION
An adequate clinical cardiac evaluation is available ordinarily in community hospitals, including the identification of abnormal rhythms and heart sounds which indicate the sites from which the majority of cerebral emboli arise. Management varies little from that outlined previously in the section on the teaching medical center. Laboratory facilities adequate to ensure the safe control of anticoagulant therapy are necessary.

When appropriate, the need for a cardiac pacemaker or for surgical correction of valvular deformities must be considered. The operation may be performed locally, if experienced physicians are available; otherwise, referral is made to another medical facility.

CEREBRAL VENOUS THROMBOSIS
Prevention of cerebral venous thrombosis is the obligation of the community hospital physician, following guidelines previously described for the teaching medical center.

JOINT COMMITTEE FOR STROKE

PRIMARY INTRACEREBRAL HEMORRHAGE
Adequate and continued therapy of hypertension is the major factor in preventing intracerebral hemorrhage. Certain other predisposing conditions can be identified by detecting bleeding tendencies through use of the prothrombin time, platelet count, and tests for other clotting factors.

SUBARACHNOID HEMORRHAGE
Spontaneous subarachnoid hemorrhage may be associated with intracranial aneurysms or arteriovenous malformations. A history of previous intracranial bleeding should be sought. A cranial or carotid bruit and areas of calcification on plain skull x-rays may be detected in association with arteriovenous malformations. The sudden onset of severe diffuse or focal headaches, especially if persistent or accompanied by any degree of nuchal rigidity, suggests a subarachnoid hemorrhage, particularly in a patient without prior or family history of headaches. The only diagnostic study which can identify an intracranial aneurysm is cerebral angiography, and if this condition is suspected there should be adequate evaluation by a neurologist or neurosurgeon.

MANAGEMENT OF RISK FACTORS IN THE PHYSICIAN’S OFFICE OR CLINIC

INTRODUCTION
Earlier in this report, a detailed discussion of risk factors for the several types of cerebrovascular disease is presented in reference to their management at a teaching medical center; these same comments are relevant to the stroke-prone patient wherever he may appear.

The major source for identification of persons susceptible to cerebrovascular disease is in the office of the primary care physician, who must appreciate the importance of the risk factors discussed previously so that he can recognize and manage properly the potential stroke patient. Some of the points mentioned in the following paragraphs are applicable equally to the general physician and to the neurological specialist in office practice.

NONEMBOLIC CEREBRAL INFARCTION
History and physical examination should be carried out as noted in the first pages of this Section. Special emphasis should be placed on a history of possible previous TIA.
pressure recordings in both arms, with the patient in supine and standing positions, auscultation for bruits and a careful search for neurological deficits are of prime importance. All hypertensive patients should have pulses palpated and/or blood pressures measured in the legs also. The fundi in all patients should be visualized adequately. For those with a carotid bruit or a history of TIA, regardless of apparent localization of either, ophthalmodynamometry may be helpful.

In patients with TIA, a blood count, EKG, serum glucose, serum lipids, and urinalysis should be determined. If a simple routine test for the hypercoagulable state becomes available in the future, this also would be appropriate.

EMBOLIC CEREBRAL INFARCTION
The historical and physical data discussed previously should be obtained. Referral for cardiopulmonary evaluation may be necessary.

CEREBRAL VENOUS THROMBOSIS
The risk factors already mentioned should be identified, when appropriate, by history and/or physical examination. Cerebral venous thrombosis should be suspected in patients with focal neurological complaints and any of the following factors: recent trauma or infections about the head, puerperal state, oral contraceptives.

PRIMARY INTRACEREBRAL HEMORRHAGE
The relevant historical and examination data have been discussed earlier. Hypertension is unquestionably the most important risk factor, while drugs or disorders leading to extensive bleeding are a distant second.

SUBARACHNOID HEMORRHAGE
As previously indicated, a subarachnoid hemorrhage gives little warning unless a previous bleeding episode has occurred. A nonbleeding aneurysm or arteriovenous malformation will be discovered by appropriate investigations if there are signs and symptoms of a focal neurological deficit.

SUMMARY
For all categories of cerebrovascular disease, the emphasis in the physician's office for identification of the potential stroke patient should be based upon: (1) historical evidence of transient or persistent focal neurological deficit, sudden and unusual headache, recent head trauma or infection, cardiac dysfunction, (2) examination findings (aside from the standard neurological assessment) referable to the vascular tree in terms of hypertension, differential blood pressures (arm-arm, arm-leg, erect-supine), bruits (cervical, orbital, supraclavicular, rarely cranial), fundi (retinopathy, emboli), and heart (rate, rhythm, size, and murmurs), and (3) judicious use of laboratory tests which often provide important help toward the solution of these problems.

The only predictors for stroke in the individual patient are evidences either that cerebrovascular disease is already present or that diffuse vascular disease (including hypertension) already exists. If the physician encounters problems for which he desires additional advice, he may wish to seek information over the telephone or refer the patient to a consultant.

Detection of Risk Factors for Stroke at a Screening Facility

INTRODUCTION
The rational basis for screening a population for any disease should be our knowledge of its epidemiology and natural history; however, much more information is needed about these factors in cerebrovascular disease. Nonetheless, it is unnecessary to wait for all points to be clarified completely before developing screening methods applicable to persons who are at higher than average risk.

Screening methods for an apparently normal population are relatively simple and could be performed by nonphysicians who have been properly trained. These procedures should identify persons requiring attention to specific elements, as careful neurological evaluation or certain additional diagnostic techniques.

Two aspects of the screening process, data collection and problem identification, can be carried out by a physician's assistant. The physician decides what information is to be collected and the assistant is then trained to obtain these data accurately. In most instances, the information will be part of a uniform data base used for detecting a wide range of problems. Both a self-administered questionnaire and personal questioning of the patient are utilized.

The individuals with specific problems which relate to cerebrovascular disease can be identified either by the physician himself or by
a physician's assistant who has been taught to recognize factors that indicate the need for referral to a physician for definitive diagnosis and management. Specific instructions can be written to guide the physician's assistant in referring those who are at risk for stroke. The following material deals with the data to be collected for identification of persons at high risk for stroke and also establishes guidelines for referral to a physician.

**DATA COLLECTION**

**Historical**

*Age and Other Factors.* Age, sex, race, and socioeconomic status are basic data for all patient histories. For stroke risk, age is by far the most important.

*Family History.* Family members who have had stroke should be listed and the information obtained should always be confirmed, if possible. Specific questions in regard to focal neurological deficits should be asked.

*Personal History.* Past or present focal neurological symptoms, whether transient or longer lasting, need to be sought by specific questions couched in lay parlance. Questions concerning past or present cardiac dysfunction, diabetes, syphilis, medications, and the use of alcohol, drugs, and tobacco also are relevant.

**Observations**

*Focal Neurological Deficits.* Observations for focal limb weakness, ataxia, hypertonia, tremor, dysfunction of speech or mentation, visual or auditory disturbances, nystagmus, facial or extracocular muscle weakness, and gait abnormalities should be part of the screening examination.

*Blood Pressure.* If a random blood pressure determination made in one arm with the person seated is 140/90 or higher, then the pressure should be taken in both arms, and the dorsalis pedis and/or femoral arteries should be palpated.

*Bruits.* A physician's assistant may be trained to detect bruits over the cervical and supraclavicular regions, over the eyes, and over the cranial cavity. With the exception of supraclavicular bruits, these sounds are likely to be related to cerebrovascular disease when heard in the adult. In the event that a bruit is detected, the patient should be referred to a physician.

**Heart Disease.** Percussion and auscultation of the heart may be taught to the physician's assistant in terms of determining size, rate, rhythm, and the presence of murmurs. The lungs can be auscultated for rales, and dyspnea and pedal edema identified.

**Laboratory Examination**

(See: Section on Laboratory Evaluation of Neurovascular Disease [Stroke]. Stroke 3: 503-526 [July-Aug] 1972). The following tests should be part of a comprehensive screening examination for cerebrovascular disease:

1. Hematocrit or hemoglobin,
2. Urinary glucose and albumin,
3. EKG,
4. Chest x-ray,
5. Serological test for syphilis,
6. Blood urea nitrogen,
7. Two-hour postprandial serum glucose,
8. Serum cholesterol and triglycerides.

**GUIDELINES FOR PHYSICIAN REFERRAL**

In an effort to identify those who are stroke-prone, the assistant should refer a person to a physician when any of the following are present:

1. History of transient ischemic attacks,
2. History of stroke,
3. History of heart disease,
4. Blood pressure greater than 140 systolic or 90 diastolic,
5. Cervical or orbital bruit,
6. Abnormalities of rhythm, motion, or sounds of the heart,
7. Abnormal EKG.

Persons with the following abnormalities also should be referred to a physician even though these findings are considered less important to stroke than the items listed above:

1. Elevated serum cholesterol or serum triglycerides,
2. Abnormal hematocrit,
3. Abnormal urinalysis,
4. Elevated two-hour postprandial glucose,
5. Elevated blood urea nitrogen.

No one individual will show all of the characteristics enumerated above. However, it is likely that the larger the number of risk factors present in one person, the greater is the potential hazard. The characteristics noted are not specific for stroke. Nevertheless, in the general population, where the incidence of stroke is approximately two per thousand per
year, the above list can be useful in identifying a reasonable number of high-risk patients for further attention and evaluation by a physician.

SCREENING AND PREVENTION
The main objective of screening should be to identify patients at greatest risk quickly and to institute corrective measures as soon as possible. Major problems associated with developing and maintaining screening programs include their promotion with initial enthusiasm far in excess of existing ability to alter the course of disease, and failure to provide the manpower and funds necessary to carry out these functions effectively. Current preventive measures are limited largely to the treatment of hypertension, the investigation and treatment of TIA, and the elimination of sources of emboli. Screening for stroke preferably should become an integral part of a screening program for other diseases, and should be considered as both a research and a service resource.

Public Education for Prevention of Stroke

IDENTIFIED RISK FACTORS IN THE INCIDENCE OF STROKE
Public education should be instituted in terms of the known risk factors for cerebrovascular disease in order to facilitate early medical care of the potential patient. In addition, those persons who are stroke-prone should be urged to have proper evaluation and treatment. A number of studies relevant to individuals and their health behavior have been made, and the following summary covers those that have pertinence to stroke.

THE PUBLIC'S KNOWLEDGE ABOUT HEALTH
Wade reports that extensive national surveys conducted at Stanford University indicate that health knowledge increases with the individual's education and income and decreases with age after adulthood. Such knowledge is greater in persons engaged in managerial and professional occupations than in blue collar workers. Women usually know more about health than do men. However, health action is not necessarily related to understanding even when symptoms are apparent. The survey revealed that printed material is more likely to be the source of public information about health than is television.

RISK TAKING
Psychological research on the concept of risk demonstrates that people vary in their patterns of response to hazardous situations. The person who does not seek care when ill may be weighing different competing risks. The average individual assumes that most preventive health measures are excessively expensive and the one with low risk of acquiring a certain disease may not wish to pay the cost involved in avoiding the potential disorder. He has not considered, however, that its treatment may be more expensive than the preventive care he could have obtained originally.

DELAY IN SEEKING HEALTH CARE
Battistella noted factors which are associated with delay in the initiation of physician care among patients in their late adulthood. Delay occurred most commonly among persons with an attitude of disregard toward their health and of disbelief in the efficacy of medical care. Relevant factors included cost for those whose economic status was just above accepted indigency levels. Contrary to expectation, delay was found not to occur commonly among persons of low socioeconomic status.

HEALTH BEHAVIOR
Health action is dependent upon many variables which include learned behavior, fear of illness, and threats to life. It may be instigated as a response to health-related motives, as a reaction unrelated to health, or as a result of established habits; several motives may exist simultaneously. A person must be psychologically ready, and conditions must be opportune before he will take any action toward seeking examinations designed for detection of potential disease.

The patient and his family are most susceptible to health education when he is ill. At that time, all aspects of predisposing causes are reviewed to learn how to avoid another incident and to follow suggestions which may lead to recovery.

METHODS OF APPROACH TO PUBLIC EDUCATION FOR THE PREVENTION OF STROKE

Fear Media
Fear-arousing communication tends to inhibit rather than to stimulate action. There is a difference between realistic concern and fear. Professional workers have been successful in influencing individuals or groups through the process of asking questions that arouse
JOINT COMMITTEE FOR STROKE

The public is much more aware of scientific developments now than in the past, largely as a result of science writers, television and radio commentators, and newspapers and magazines. Trained reviewers and abstractors who are able to evaluate medical publications are in short supply; education of additional competent experts in this field is necessary.

Educational Media
Mass media may influence the public, temporarily at least. Brief television interviews with physicians on symptoms of heart attacks have proved effective. Similarly, interviews on the subject of risk factors for stroke are likely to be valuable. Health agencies and professionals should pursue all avenues of entry to health education via television and radio, including spot announcements. Entertainment programs involving medical care subjects are sources for health education.

Publications for patient education should be available in all areas of the health care system, including doctors' offices, clinics, waiting rooms, outpatient departments in hospitals, and health departments. Different media should be developed for specific ages, and for various educational, cultural, and language groups. Articles on health in popular magazines are a major weapon, e.g., "Nine Steps to a Longer Life," published in Reader's Digest, October, 1970. Comic strips are a commendable means of education by which the reader may be made aware of significant risks to health.

Health Education in the School
New curricula concerning basic consumer education in health-related services should be initiated for children in kindergarten and should continue through college. The aim would be to develop a program of instruction in the maintenance of health and preparation for family life tailored to prepare young people for marriage, parenthood, family living, and prevention of disease. All-out support from the Federal Government and general acceptance of the program in the schools are being sought.

Employment Settings
Business and industry are interested in protecting the health of their executives and employees, thereby lessening absenteeism, and maintaining productivity. Hence, these organizations should be concerned with promoting education programs which have value in keeping the health of their employees at a high level.

Health Advocates or Aides
Health aides and advocates have been taught to survey and organize community groups, make home visits, conduct interviews, make referrals, lead film discussions, and write reports. The aides make regular door-to-door visits, bringing to the attention of area residents those health services that are readily available.

Health aides function in three principal roles: (1) helping to dispel local superstitions and beliefs which impede modern health practices, (2) helping people to overcome apathy, encouraging adoption of good health practices, and assisting them in taking advantage of existing health facilities, and (3) helping professionals in performing some routine tasks related to health.

Counseling
The most effective health teaching is carried out by personal counseling in whatever setting the individual is encountered. When given an opportunity to describe his health problems, concerns, and needs to someone he can trust, the person is apt to respond to the guidance offered. Such behavior and health action are noted in family practice clinics where the whole family is well known and personal...
CLINICAL PREVENTION OF STROKE

attention is given to individual family members. This is equally true in private or group practice when a nurse skilled in communication and interpersonal relationships participates in total health care.

The period during which he is receiving health care presents an unusual opportunity to evaluate the patient's attitudes, values, life style patterns, and knowledge of his health. Whether the patient proves to be symptomatic or without medical problems, this is a favorable time for judging his needs and determining the degree of his interest in obtaining more insight into his own and his family's health requirements.

Community workers who meet family members in the home environment can assess their strengths, and determine the problem areas where assistance is needed in taking further steps toward better health care. With the aid of social workers and public health and/or community nurses, reasons for delay in seeking medical attention can be documented. Often, positive action can be taken to improve health conditions. Community workers have been most successful in contributing to families who do not have access to health information through newspapers, magazines, and other printed media.

In a recent study of the utilization of the public health nurse in a two-hour clinic held weekly in an apartment house for senior citizens, requests for assistance or counseling were found to increase tenfold over a two-month period. In instances where medical care had been long delayed, there was documented evidence that fear in seeking health care was lessened and the first steps taken toward obtaining medical diagnosis and treatment by attending the clinic.

Group Interaction

Group interaction may play a critical role in development of the person; therefore, health educators often use group sessions as an educational method, with many benefits as a consequence.

1. Individuals are given an opportunity to verbalize their feelings,
2. An atmosphere is provided where individuals can clarify potential or real areas of disagreement,
3. Various individual points of view are brought together,
4. Efficiency in educating large numbers of people is increased as compared with one-to-one communication.

Many health educators believe that group sessions provide a valuable opportunity for individuals to find creative and practical solutions for various health-related issues and problems.

SUMMARY

1. Every educational program must have its goals and objectives identified.
2. The population group to whom it is directed must be determined.
3. The content of the educational program must be developed.
4. Risk factors known to be significant in causing stroke must be emphasized.
5. The public should be made aware of available diagnostic measures and treatment.
6. The public should learn about the limitations of knowledge in regard to stroke prevention.
7. Emphasis should be increased on responsibility of the individual for changing his health habits, particularly in relation to the prevention of illness.

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