Management of Patients with Established ("Completed") Cerebral Infarction

FERDINANDO BUONANNO, M.D. AND JAMES F. TOOLE, M.D.

SUMMARY Management of patients with sudden neurological deficit must be based on complete knowledge of the underlying cause. In about 80% of such patients, a careful history and examination will lead to a precise etiologic and anatomic diagnosis. If the deficit is vascular in etiology, therapy and prognosis depend on its stage of evolution.

Definition of Terms

BY ARBITRARY DEFINITION, focal neurological deficit due to vascular disease which resolves in less than 24 hours is called a transient ischemic attack (TIA); one that exceeds this length of time is attributed to infarction or hemorrhage. Some neurologists distinguish another category, in which a neurological deficit lasting more than 24 hours eventually resolves completely — usually within 3 weeks. This has been termed a reversible ischemic neurological deficit (RIND). It is postulated that this situation results from sustained ischemia not severe enough to produce infarction.

To be considered as established, or completed, an infarction in the brain supplied by the carotid system must be static for at least 24 hours; one in the brain supplied by the vertebrabasilar arterial system, for 72 hours. If new symptoms or signs develop during this period, the original episode is regarded as an evolving (progressing) infarction.

Established or completed refers to the sequence of events and does not imply that a neurological deficit is as severe as it can become, but indicates that infarction has remained unchanged. For example, in a right-handed patient with an established infarction in the distribution of the left middle cerebral artery there may be no asphasia. Because it is still possible for the infarction to extend within this vascular bed, the question is whether prophylaxis following the first episode can prevent this.

These definitions are retrospective and arbitrary but because they form a framework with which to link pathophysiology and clinical events, they are important when one is comparing groups of patients and different types of therapy. TIA and RIND suggest ischemia with preserved viability; established and completed infarction, death of tissue.

Emergency Management

With rare exceptions, patients with an infarction must be admitted to the hospital as emergencies. If the patient is in coma or comatose, management geared towards the maintenance of life must be initiated before he is evaluated for cerebral infarction.*

If there are signs of increased intracranial pressure, the head of the bed should be elevated; otherwise, the patient should lie supine or, if he can tolerate it, prone in the Trendelenburg position with his head turned to one side.

Immediate differentiation between cerebral hemorrhage and infarction is mandatory, because the 2 conditions require entirely different types of management and therapy.

Diagnostic Evaluation

In order to complete all essential tests within 12 hours, the following procedure should be adopted. While the patient is still in the emergency room, urine and blood samples are obtained for the laboratory investigations listed in the table. A 12-lead electrocardiogram is done before the patient leaves the emergency room, and, thereafter, the patient is maintained on an ECG monitor for 24 hours or more to assess cardiac rhythm.*

Electrocardiographic abnormalities are more frequent in patients with intracranial hemorrhage* than in thromboembolic disorders.* Progressive bradycardia may reflect increasing compression of the brainstem, and dysrhythmias may result in diminished cerebral perfusion.*

While in the emergency room, the patient is prepared for an emergency computerized cranial tomographic study (CCT) — the keystone of initial management. If CCT is not available for immediate use, skull radiographs can be obtained to display an unsuspected skull fracture, an enlarged sella turcica,
or a shift of the pineal gland. If the pineal is not calcified, echoencephalography can be used to determine whether midline structures have been displaced. A midline shift is demonstrable at the time of admission to the hospital in 77-85% of patients with intracranial hemorrhage; but only in 4% of patients with cerebral infarctions and then only after edema has developed 6-24 hours after onset.

CCT, performed both before and after the infusion of contrast material, almost always reveals intracranial hemorrhage immediately if the stroke is hemorrhagic. In one series, CCT revealed hemorrhages in 43% of patients with stroke clinically diagnosed as having infarction. CCT may also reveal a subdural hematoma and hemorrhage into a neoplasm as causes of stroke. According to recent estimates, neoplasm is responsible for a significant number of acute "strokes".

Repeated CCT also permits direct evaluation of cerebral infarction. While not detectable for 2 or 3 days after the onset, atherothrombotic and embolic infarcts are thereafter readily visualized on CCT as areas of homogeneous decreased density within a particular vascular distribution. As many as 98% of patients with infarctions have abnormal CCT sometime during the evolution of their disease, but normal CCT 48 hours after the onset of symptoms correlates almost exclusively with TIA.

The results of CCT are central to decisions regarding management. If edema is present, consideration must be given to placement of an intracranial pressure monitor, and to appropriate medical or neurosurgical management. If there is a hemorrhage, arteriography and possible evacuation of clot are the pivotal issues. If the clinical picture and CCT do not reveal the etiology, the cerebral spinal fluid is examined for further clues for diagnosis and always before initiating anticoagulant therapy.

A multitude of non-invasive techniques has been developed to help distinguish between extra- and intracranial arterial disease. These tests are not performed as emergencies but on a sequential basis some days after the patient's clinical state has stabilized. Although their value has diminished since the advent of CCT, radionuclide techniques retain a place in the evaluation of patients with cerebrovascular disease. On rectilinear scanning, the classic picture of an ischemic infarction is an area of increased radionuclide uptake within a particular vascular distribution. Neoplasms usually have a different appearance but in situations where distinction is not evident differential uptake of diverse isotopes has been used. Studies on small numbers of patients cast doubt on the ability of 18F-fluorodeoxyglucose to differentiate cerebral infaracts from neoplasms. Recent studies, however, yielded more encouraging results. In one study, brain scans with both Tc-99m-pertechnetate and Ga-67 citrate were performed on 93 patients with cerebral neoplasms and on 70 others with cerebral infarction or hemorrhage. While tumor detection was better with Ga (96%) than with Tc (85%), infarctions either did not concentrate Ga (67%) or revealed a much lower concentration than Tc (27%). In another series, Tc-99m-(SM)-diphosphonate demonstrated 15 of 22 infarcts better, 3 less well, and 3 equally well as pertechnetate, while the latter demonstrated 11 of 12 primary or metastatic tumors better than Tc-99m-(SM)-diphosphonate.

Neoplasms are generally evident on brain scan when neurological deficit begins but cerebral infarction is usually not detectable until 2 to 14 days later. Welch et al. have reported abnormal brain scans 48 hours after infarction in 27% of 162 patients. The abnormality is due to increased vascularity of "luxury perfusion" surrounding an infarct and is more often associated with embolic rather than with occlusive disease.

The diagnostic yield increases if static brain scans are coupled with dynamic studies. In 143 patients with stroke cerebral radionuclide dynamic ("flow") studies were abnormal in more than 80%, but static scans were positive in only 42%. In contrast, the percentage of abnormal CCTs in recent infarctions was 40-55% after 2 or 3 days and reached a maximum of 66% at 3 weeks. While the percentages are roughly equal for both techniques, the results of the 2 studies in a given patient may differ. In one recent series, the 2 methods yielded complementary findings in 16 (55%) of 29 patients.

Coupling CCT and radionuclide scans is useful as an aid to making decisions regarding etiology and management. If the CCT shows an abnormality suggestive of infarction, a radionuclide brain scan is performed. If this is normal, we conclude that the blood-brain barrier is intact and that angiography, with a view to reconstructive surgery, can be safely performed at once. If the scan is abnormal, suggesting vascular breakdown, angiography is delayed for at least 3 weeks for gial repair to occur as a prelude to reconstructive surgery. During this time the patient is maintained on anticoagulant therapy.

Arteriography is the definitive diagnostic procedure in patients suspected of having cerebrovascular dis-
order. Indications include: 1) uncertainty of the diagnosis; 2) deterioration in the clinical condition of a patient with a previously stable deficit; and 3) the possibility of performing reconstructive surgery.44

Our morbidity with angiography ranges between 1 and 2%, the incidence of permanent neurological deficits being about 0.5% and the mortality about 0.2%. This, coupled with the risk of surgery, make it particularly necessary to pre-select carefully patients for arteriography.

Because emergency arteriography and/or surgery during an ictus carries a higher morbidity and mortality,45 angiography is not performed in the acute stages unless brain scan is negative or the diagnosis is in doubt. If the patient makes a good functional recovery, arteriography performed during convalescence may indicate the need for surgery to prevent recurrence or extension of infarction.

**General Principles of Management**

Fastidious general management of the patient is an important factor in determining the outcome. Apart from those patients who die before therapy can be instituted, the majority of deaths from stroke are not directly due to the cerebral insult but to the ensuing complications.

The patient's room should be quiet, and he should be sedated, preferably with barbiturates. Barbiturate-augmented hypothermia has been reported to decrease intracranial pressure and cerebral metabolic rate for oxygen46 but its clinical utility is still unproven.

Aggressive treatment of hypertension is indicated for patients with hypertensive encephalopathy or subarachnoid hemorrhage. For patients with thrombotic occlusions, on the other hand, moderate elevations may be necessary to maintain cerebral perfusion, and lowering the blood pressure may increase cerebral ischemia. Dysautoregulation has been demonstrated in hypertensive patients and — even without the loss of autoregulation associated with a stroke — a reduction of blood pressure can jeopardize perfusion. Hence, hypotensive therapy must be used judiciously, if at all. Some47 have recommended the use of norepinephrine to elevate blood pressure during the acute phase of some ischemic episodes. Aside from the treatment of vasospasm or other neurological deficits related to angiography,48 such therapy has not been fully evaluated in controlled studies in patients with stroke.

Fluids should be restricted enough during the first 4 or 5 days following a stroke to keep the patient slightly dehydrated without significantly increasing blood viscosity and further reducing cerebral blood flow.49 Proper fluid management requires weighing the patient at least every other day, determining serum and urinary electrolytes q.o.d., and maintaining accurate records of fluid intake and output. Daily fluid requirements can be satisfied by the administration of about 1500 mL of 5% glucose in 1/2 N saline containing 20 mEq/L of potassium. If oral liquids are not tolerated, a nasogastric tube is passed and tube feed-
treatment affect the platelet phase of the coagulative process, in the "cascade" proper, or in the thrombolytic phase? "Full anticoagulation" is desirable and is initiated with heparin and followed by maintenance coumarin drugs. No consensus has been reached regarding the utility of such treatment for patients with established or completed infarction.68-71 Except when a condition contraindicates anticoagulant therapy (e.g., pregnancy, hemorrhagic diatheses, active peptic ulcers, uremia, pericarditis, bacterial endocarditis, hepatic disease)72 intravenous heparin is used in patients with early infarction. The initial dose (5000 U/500 ml saline) is often begun in the emergency room, an infusion pump being used to control the rate of administration. Heparin is continued for at least 72 hours, or until definitive decisions about long-term management are made. Full anticoagulation generally should be given for at least 2 months following thrombosis because this much time is required for the thrombus to adhere to the vessel wall and to be covered with endothelium.73 The dosage of heparin is regulated by the clotting time. Whether the Lee-White method for determining whole-blood clotting time, or the activated partial thromboplastin time (aPTT) is used, the clotting time should be kept within a range of one and a half times that of normal controls. The patient's own blood cannot be used as the control because thrombosis may accelerate the clotting time. The method most frequently used is the Lee-White clotting time which is less sensitive but has the advantage that it can be performed at the bedside. Advantages of the aPTT are that it can be performed on anticoagulated blood at the laboratory's convenience, and that it has a very rapid end-point.

Platelet Antiaggregants

During hemostasis, platelets undergo adhesion, release reaction, aggregation, and consolidation. Drugs affect different steps in this sequence, but most of the "antiplatelet" agents employed interfere with adhesion or aggregation. Antiaggregant effects have been demonstrated for non-steroidal anti-inflammatory agents (aspirin, phenoprofen, phenylbutazone, soudoxicam, sulfinpyrazone) and for the pyrimidopyrimidine compounds (dipyridamole, papaverine, pyridinolcarbamate); similar actions have been reported for numerous other compounds (antihistamines, barbiturates, clofibrate, cyproheptadine, halofenate, propranolol, prostaglandin PGE, tricyclic antidepressants, and antipsychotic drugs).74

A significant increase in platelet adhesiveness has been observed in 43 patients with ischemic infarction, 88% of whom were below the age of 40.80 Platelet aggregation is significantly greater in young patients with stroke than in controls.80,81 This difference is not so evident in older patients because platelet aggregability is thought to increase with age.82 Some studies83 have shown that dipyridamole reverses abnormalities of platelet aggregation in patients with cerebrovascular disease, but it was found that aggregated platelets returned to normal 10-40 days after acute cerebral ischemia even in the absence of aspirin or dipyridamole therapy.84

Although the numerous agents listed above have not been extensively tested, there is no present evidence that platelet antiaggregants are useful for completed infarction. One report of the effect of such drugs for reducing morbidity, mortality, or post-infarction TIAs in patients with previous strokes,85 showed that the survival rate was improved by the long-term administration of sulfinpyrazone, 600 mg/day.

A double attack on the coagulative mechanisms has been suggested by Sullivan et al.,86 who conducted a study in patients with prosthetic heart valves. Those who were treated with a combination of dipyridamole and warfarin compounds had fewer embolic episodes than those treated with warfarin alone. No major hemostatic problems have been reported in patients receiving both anticoagulants and antiplatelet drugs, although the incidence of bleeding disorders should be theoretically increased by such therapy.

Because abnormalities of the fibrinolytic system have also been demonstrated in patients with stroke,87 plasmin-pathway activators (acetylcholine, adrenalin, nicotinic acid, streptokinase, urokinase) have been suggested or employed by some investigators, but the data are still inadequate.

Anticonvulsants

Generalized or focal seizures occasionally initiate or accompany cerebral infarction. Seizures are more frequent in embolic infarction and in cortical venous occlusions. Two retrospective studies have been conducted on autopsy-proven cerebral infaracts — and hence on populations selected for the most severe lesions. In one, seizures were reported in 12.5%, in the other 6.6% and in a third,88 the incidence of seizures was 7.7%. In 33 of these 77 patients with post infarction seizures, convulsions occurred at the onset of stroke or within the first 2 weeks; in only two of these 33 patients did recurrent seizures develop. Of the 27 patients whose seizures developed after the second week, 22 became epileptics. Of interest is the fact that anticonvulsants were ineffective in the patients whose seizures developed early, whereas late-onset seizures were easily controlled.

This study throws doubt on the value of "prophylactic" anticonvulsant therapy for patients with non-embolic cerebral infarction.

Barbiturates

The potential value of barbiturate sedation for stroke management has been mentioned. The concept of using these drugs to provide pharmacologic protection for the ischemic brain has some experimental support89-92 but has not been tested clinically.

Blood Gas Therapy

Hypocapnia. It has been suggested that prolonged hyperventilation might benefit stroke victims by reducing PCO, causing vasoconstriction, and shifting
blood to an ischemic area, and by reducing intracranial pressure, but there are numerous reports of adverse effects produced by such therapy. No significant difference was noted between patients with severe infarction treated with hypocapnic ventilation and those treated with normocapnic active ventilation.

Hypercapnia. Because it is the most powerful cerebral vasodilator, carbon dioxide in varying concentrations has been administered to patients with evolving infarction. In both animals and patients with brain infarctions, an increase in regional cerebral blood flow (rCBF) has been demonstrated after the addition of 5% CO₂. However, no rigorous studies documenting its therapeutic effect on human beings are available.

An objection to its use is that it might induce an intracerebral "steal"; i.e., with the non-reactive vessels in an ischemic brain already maximally dilated, dilatation of the remaining normal vessels might shift (steal) blood from ischemic areas. Two studies, however, have shown that vasodilator response was reduced or impaired in only 25% of patients with cerebral circulatory disturbances. It is generally agreed that the steal response occurs only during the acute stages of severe and extensive cerebral infarction, and then only rarely.

Hyperoxia. The use of hyperbaric oxygen therapy to increase the oxygenation of ischemic brain is an appealing idea. Patients with infarction so treated show clinical improvement initially, but the effects are not sustained when the patient leaves the hyperbaric chamber. The vasoconstricting effects of O₂ reduce CBF to such an extent that tissue oxygenation may not change. This effect may be circumvented by the concomitant use of decarboxylase or carbonic anhydrase inhibitors, which cause vasodilatation.

Vasodilators

Studies on the effects of vasodilators other than CO₂ are also conflicting and inconclusive. In normal subjects, oral or parenteral papaverine increases CBF and cerebral oxygenation, but McHenry et al. found no benefit in 6 patients. Furthermore, although Meyer et al. reported a slight improvement in 27 patients, the control and treated populations were not comparable and embolic events were grouped with thrombotic infarctions. Data regarding the utility of other vasodilators (e.g., dehydroergonovine, nylinidrin, betahistine, beta-blockers, cyclandelate, hexobendine) — are even less conclusive.

Dehydrating (Antiedema) Agents

Cerebral edema accompanying ischemia is both cytotoxic and vasogenic in origin. In the early stages, impairment of cellular metabolism leads to cytotoxic edema. The vasogenic component, which arises later, is presumably secondary to cell death, to loss of integrity of the blood-brain barrier, to the extrusion of osmotically active particles into the extracellular space, with a consequent increase in the extracellular water content. A theory, which has not been proven, is that edema is self-perpetuating because it a) overtaxes the cell's Na-K pump so that an accelerating deficit accrues, and/or b) has undesirable effects on the microcirculation, reducing blood flow further and causing additional cellular damage. Cerebral edema may be sufficient to cause brain herniation and death.

Hyperonotic Solutions. Urea, mannitol, and glycerol have been used to reduce increased intracranial pressure and, more recently, for cerebrovascular insults. These agents act by increasing osmotic gradients, thus promoting the egress of water from areas of cytotoxic edema and from normal brain. They share the ability to act rapidly and to decrease blood viscosity, but may produce some complications such as severe dehydration with electrolyte imbalance and intracranial hemorrhage. Glycerol does not seem to produce the complications associated with the other 2 compounds, and its effect lasts nearly 48 hours, as compared to 6-8 for urea and mannitol. Another advantage of glycerol is that it acts even in nephrectomized animals and is less toxic to both kidneys and liver than urea and mannitol. Theoretically, glycerol, alone of the 3 agents, provides a metabolic substrate and improves cerebral metabolism as well as increases in cerebral blood flow.

Meyer et al. reported reduction of mortality in 36 patients treated with glycerol during acute cerebral infarctions. Mathew et al. demonstrated improvement in neurological status 14 days after an acute cerebral infarction in patients treated with intravenously administered glycerol. Gilsanz et al. confirmed the favorable effect of glycerol and noted that it was more efficacious than dexamethasone. Fritz and Werner noted no improvement in patients with either a minimal or severe deficit, but did note a significant improvement in the moderately-impaired patients. However, the patients were not followed long enough to determine whether such therapy decreased disability or changed life expectancy.

In a retrospective study of 227 patients with infarction treated with various dehydrating agents, Candelise et al. found no improvement. Gelmers evaluated 100 patients and could not demonstrate beneficial effects of glycerol during the 4 weeks after an acute infarction, confirming similar results obtained by Larsson et al.

Glycerol may theoretically have an adverse effect on normal brain, but this has not been documented. The hyperosmolarity produced by glycerol does not begin to approach that found in human hyperosmolar states. It has been suggested that an osmolar gradient of about 30 mOsm is desirable for achieving an adequate dehydrating action, yet the improvement in hemispheric blood flow is noted at 14-20 mOsm. Dehydrating agents have also been criticized as having
only short-lived efficacy,78,128 but Newkirk et al.137 were able to demonstrate control of intracranial pressure continuously for 3 weeks using oral glycerol.

Because of its ease of use, lack of toxicity, and rapidity of action glycerol is the leading agent for the control of brain edema—both massive, life-threatening and peri-infarct edema. Beneficial secondary effects of glycerol derive from the enhancement of metabolism,114,128 increase in blood flow,126 platelet antiaggregation,129 and ADH inhibition.180 If given as a bolus, glycerol produces rebound pressure;181 hence it must be given slowly.129,131,132 A 10% solution is given by slow intravenous drip over 4 to 6 hours twice daily. If intravenous glycerol is not available, it can be given orally at 2-hour intervals, the total daily dose being 2 gm/kg body weight. Glycerol should be administered for about 4 days and complemented by the use of adrenocorticosteroids. Glycerol minimizes the early cytotoxic component of edema, while the steroid (which has maximum effect after 6–48 hours) should counter the later vasogenic component of edema accompanying brain ischemia.

**Steroids.** No matter how rational such a scheme might seem, there is scant evidence that adrenocorticosteroids affect the sequence of events in patients with cerebral ischemia and infarction. Earlier investigations attributing beneficial effects to steroids were either not controlled or lacked adequate statistical analysis. In the widely misquoted study by Patten et al.124 in which the effect of 220 mg of dexamethasone given over 17 days was evaluated in 31 acute stroke patients, 3 patients in the control group had cerebral hemorrhage. With or without these 3 patients there was significant improvement (p = 0.02) in the group presenting the greatest neurologic deficit. The authors concluded that dexamethasone can be a useful adjunct in the therapy of patients with severe stroke. Studies on the effect of mega-doses of steroids (1000 mg/24 h) in patients with evolving infarction have not been reported. Other reports cast doubt on the efficacy of steroids in the treatment of stroke. Dyken and White146 found that death of patients with acute cerebral infarction occurred as frequently whether or not they were treated with 300 mg of cortisone per day, but the investigators themselves indicated that their data were not statistically significant. Hetzel et al.148 evaluated the effect of cortisone “in the dosage recommended” in a double-blind fashion in only 12 patients. In each group 3 patients died, 2 were better, and one remained the same. The authors concluded that “large doses of cortisone” were of no value in the immediate treatment of stroke. The same conclusions were reported by Bauer and Tellez149 in their double-blind study of the effect of 12 mg of dexamethasone daily for 10 days. However, the treated and control groups were not comparable because the placebo patients were more ill on admission. It was reported that dexamethasone treatment did not alter prospects of discharge home or death rate at 3 months in 247 patients with hemiplegia;150 these patients received either 48 mg of dexamethasone over 3 days, or 112 over 7 days, but many had hemiplegia for 7 days before medication was administered. Because edema is most likely to occur during the first 2 to 4 days after the onset, therapy needs to be initiated earlier, as was done by Norris.182 In his study, 53 patients with acute cerebral infarction were treated with 140 mg dexamethasone or placebo for 12 days and evaluated at intervals for 28 days. Norris concluded that the patients treated with steroids fared slightly worse with 2 of the 5 patients in the placebo group dying of cerebral edema, compared to 3 of 7 in the steroid group, while infections, gastrointestinal hemorrhage, and exacerbation of diabetes mellitus occurred more commonly in the steroid-treated group. This contrasts with the study of Bauer and Tellez149 where 3 patients developed gastrointestinal tract bleeding while on the placebo regimen but there were no complications in the steroid-treated group.

In Norris’ study the 2 populations were not comparable since the steroid treated group was more seriously ill and there were different etiologies for stroke.

The effect of coupling steroid therapy with other anti-edema agents has not been adequately evaluated. In a double-blind study of 40 patients given 215 mg of dexamethasone over 15 days, plus 7500 ml of low-molecular weight dextran over 3 days starting 24–48 hours after the onset of symptoms, Kaste et al.140 could not find a reduction in mortality or morbidity at the end of a 28-day observation period. The simultaneous administration of steroids and diuretics, a combination effective in animal models, has not been tried in humans.

**Surgical Measures**

Two major surgical techniques have been used in suitable patients with cerebral infarction. These are: 1) endarterectomy or thrombectomy for lesions in the cervical carotid artery, and 2) extracranial-intracranial anastomosis to bypass inaccessible arterial lesions.

Re-establishing flow and pressure during acute carotid occlusion accompanied by neurological deficits is likely to produce intracerebral hemorrhage.141–148 The dangerous period lasts a minimum of 2 weeks after ischemia. A mortality of 42% in 50 patients has been reported for patients operated upon during this period, as compared to a 20% mortality in nonoperated patients.144 Surgical treatment has been recommended for patients with mild completed stroke and for reversible ischemic episodes.

The 4-year mortality reported in groups of patients with RINDs varies from 17% to 40%; during the same period, up to 55% deteriorate neurologically. Twenty-nine–40% of patients with established infarction will die of another stroke within 3 years.144 Elective thromboendarterectomy is indicated in patients whose carotid is stenosed but not occluded, and for patients with a mild to moderate neurological deficit. For patients of this type, Bauer144 reported significant improvement in the 42-month survival rate after surgery. In that report, as in others,141–146 patients with established in-
Most commonly, this procedure has involved anastomosing the superificial temporal artery to a branch of the middle cerebral artery (ST-MC bypass). The procedure carries some morbidity and mortality but in all large series patency rates of more than 80% have been achieved. At present, suggested indications include generalized low perfusion syndromes (e.g., multiple large vessel occlusive disease) as well as focal low perfusion syndromes. The latter include situations where collateral circulation is inadequate to compensate for internal carotid occlusion, as well as stenosis of the carotid or middle cerebral artery, and occlusions inaccessible with previous vascular techniques. Some authors suggest that at least a 25% reduction, focal or generalized, of cerebral blood flow should be present before ST-MC bypass is contemplated.

Reports on the use of this new surgical approach are difficult to evaluate because of the wide variation in the time elapsing between the onset of stroke and surgery (one day to 7 weeks or more), differences in the classification of patients, and short follow up periods.

Rehabilitation

Rehabilitation seeks to help the patient with completed stroke achieve as great a degree of independence as possible despite the amount of neurologic deficit. Rehabilitation should begin as soon as possible after the onset of a stroke. The initial goals are to avoid increased disability from immobility by employing daily passive exercise with all weakened extremities being put through a complete range of motion. This avoids muscle shortening and joint fixation which are difficult to treat when the acute phase of stroke is over and function begins to return.

As function begins to return the patient is educated to strengthen it, use it and use his uninvolved extremities to carry out activities of daily living which make for independence. Programs for this phase of treatment are ideally carried out in specialty hospitals with disability oriented units. In such institutions for those patients not neurologically devastated, partial to complete independence can be expected in 60-80% of patients. Treatment can be successfully carried out in nonspecialty hospitals provided there is interest in the problem of stroke rehabilitation and there is personnel skilled in physical and occupational therapy.

Poor outcome from programs of rehabilitation for patients with stroke may be expected when there is severe paresis, left sided neglect, dementia and global aphasia. Good results can be expected in patients with mild to moderately severe paresis even with expressive-receptive dysphasia. Outcome is also improved when patients have families able to care for them at home despite disability. General function may progressively improve over 12-18 months after the onset of stroke despite lack of improvement of neurologic deficits in the involved extremities. This largely is related to the patient’s ability to learn to use his uninvolved extremities for improved function.

References

1. Millikan CH (Chairman): Classification and outline of cerebrovascular diseases II. Stroke 6: 594-616, 1975
16. Lavy S, Yaar I, Melamed E, Stern S: The effect of acute stroke on cardiac function as observed in an intensive stroke care unit. Stroke 5: 775-780, 1974
24. Pearce JMS: If I had a transient ischemic attack at the age of
by guest on October 15, 2017 http://stroke.ahajournals.org/ Downloaded from

STROKE

VOL 12, NO 1, JANUARY-FEBRUARY 1981

44. Toole JF: Diagnosis and Management of Stroke, American Heart Association, 1979
46. Michenfelder JD, Theye RA: Cerebral protection by thiopental during hypoxia. Anesthesiology 39: 510-517, 1973
64. Mustard JF, Packham MA, Platelets, thrombosis and drugs. Drugs (Basle) 9: 19-76, 1975
76. Enger E, Boyesen S: Long-term anticoagulant therapy in...


82 Couch JR, Hassanein RS: Platelet aggregaton, stroke, and transient ischemic attack in middle-aged and elderly patients. Neurology (Minneapolis) 26: 888-895, 1976


95 Christensen MS, Paulson OB: Prolonged artificial hyperventilation in severe apoplexy: Clinical results and CSF findings in a controlled study. Panminerva Med 13: 201, 1971


100 McHenry LC Jr. Formal discussion of paper by Fieschi C, Ref. 99, Ibid., pp 139-141


116 Mathew NT, Cherry JZ, River VA, Mathew NT: Treatment of cerebral edema due to acute cerebral infarction. Lancet 2: 1027-1029, 1971

117 Gilsanz V, Rebollar JL, Buencuerpo J, Chantrcs MT: Con-


Management of patients with established ("completed") cerebral infarction.
F Buonanno and J F Toole

*Stroke*. 1981;12:7-16
doi: 10.1161/01.STR.12.1.7

*Stroke* is published by the American Heart Association, 7272 Greenville Avenue, Dallas, TX 75231
Copyright © 1981 American Heart Association, Inc. All rights reserved.
Print ISSN: 0039-2499. Online ISSN: 1524-4628

The online version of this article, along with updated information and services, is located on the World Wide Web at:
http://stroke.ahajournals.org/content/12/1/7