CAROTID ARTERY bifurcation endarterectomy is now well established in the treatment of patients with monocular or hemispheric transient cerebral ischemic attacks. Conversely, it is also thought that carotid surgery is contraindicated for patients with acute severe strokes because of the risk of making the neurological deficit worse. However, a small percentage of patients have thromboembolic cerebral ischemia manifested by unstable neurological deficits of only mild to moderate degree. There is no consensus on the approach to these patients, but many neurologists and vascular surgeons have considered them as acute strokes and avoided angiography and surgery. Dissatisfied with the outcome in several such patients, we have recently adopted a more aggressive plan of management. The purpose of the present report is to describe this approach and the successful results of its use.

Definitions

The following definitions, adapted from the Handbook of Clinical Neurology,

**Transient ischemic attacks** — Cerebral hemispheric or monocular episodes of transient ischemia, usually lasting a few minutes to a few hours, leaving no residual signs and symptoms between attacks. **Crescendo** transient ischemic attacks are those attacks abruptly increasing in frequency to at least several per day.

**Stroke in evolution** — Several symptom patterns can be included in this term, which is also known as progressing stroke or incomplete stroke. An acute neurological deficit of modest degree may, within hours or days of the initial event, progress in a sequential series of acute exacerbations to a major stroke. Alternatively, after the initial episode the neurological deficit may improve temporarily, only to reappear later, often with more widespread involvement, leading to a pattern of waxing and waning of signs and symptoms that occurs over hours to days with an incomplete recovery.

Clinical Material

In the past three years approximately 180 patients have undergone carotid thromboendarterectomy at the San Francisco Veterans Administration Hospital. The primary indications for angiography and operation were transient ischemic attacks (hemispheric or monocular) and asymptomatic carotid artery stenosis in patients requiring other major cardiovascular surgery. During the same time period 26 patients were encountered who underwent emergency evaluation, including angiography and emergency carotid endarterectomy. The indications for emergency arteriography in this group were crescendo transient ischemic attacks in 8 patients and stroke in evolution in 18 patients. None had severe, devastating neurological deficits or depressed levels of consciousness.

Angiographic Management

All patients underwent carotid arteriography via the transfemoral route, using the Seldinger technique and rapid film changer. In several of the patients, the studies were incomplete because the procedure was terminated once a critical arterial lesion was identified. Thus, often only one carotid artery was examined, but in all cases at least two projections of the neck vessels and ipsilateral intracranial vessels were taken. Selective carotid injections using small catheters and small amounts of contrast medium are important details of the angiographic management of these cases. Injections to visualize the vertebral arteries and the aortic arch are no longer performed routinely. None of the patients in this series suffered any complications from arteriography.
lesion appropriate to the side of the cerebral symptoms. Atherosclerosis was the etiology in each case. An angiographic example of one of the arterial lesions is shown in fig. 1.

Management

Because of unstable neurological symptoms, the clinical evaluation of each of the patients in this series was done on an urgent basis, including emergency carotid arteriography. All of the stroke-in-evolution patients received heparin anticoagulation prior to angiography, and the remainder received it as soon as the critical arterial lesion was identified. Emergency carotid bifurcation endarterectomy was then performed as soon after completion of angiographic studies as possible.

The surgical and anesthetic management used in these cases has been well described in previous publications and includes general anesthesia (Halothane) and maintenance of normal arterial PCO₂ and preoperative blood pressure. Our usual practice is to use a temporary internal shunt during carotid endarterectomy only in selected patients, namely, in those whose internal carotid artery back pressure is below 25 mm Hg (under conditions of normocarbia and normotension) and in those who have had a previous cerebral infarction on the side of operation regardless of the level of carotid back pressure. In conformity with this policy, a shunt was used in 2 of the 8 patients with crescendo transient ischemic attacks because of low back pressure and in all 18 patients in the stroke-in-evolution group because of inability to exclude the presence of cerebral infarction. Intraoperative arteriograms at the conclusion of each procedure confirmed a satisfactory technical result.

Results

Eight of the 26 patients in this series had crescendo transient ischemic attacks. Eighteen had stroke in evolution, of whom 10 exhibited the slowly progressive pattern and 8 the waxing and waning or stuttering onset pattern. As noted above, each had a critical arterial lesion at the carotid bifurcation appropriate to the symptoms. Each of the 26 patients made a dramatic, complete, and so far permanent neurological recovery following operation. There was no morbidity from either the angiographic or surgical procedures.

Discussion

Excellent results from therapeutic carotid artery surgery can be obtained in patients with transient hemispheric or retinal ischemic attacks whose neurological condition is stable. The 8 patients in the present series with crescendo transient ischemic attacks, although neurologically unstable by virtue of an abrupt increase in the frequency of their symptoms, can be included in this group in which nearly negligible morbidity and mortality should be expected. On the other hand, the surgical mortality for operations performed to restore flow in an occluded carotid artery during an acute cerebral infarction ranges from 40 to 50%. This unacceptably high figure, reported by Wylie and associates as well as by the Joint Extracranial Arterial Occlusive Disease (EAO) Study, forms the basis for the belief that carotid artery operations for acute stroke are contraindicated. Many vascular surgeons tend to place in the same high risk category any patient with a neurological deficit that is not completely reversible, that is to say, not a bona fide transient ischemic attack. It has been postulated that the mechanism responsible for the deterioration in this situation is the restoration of flow and pressure into an ischemic area of the brain as a result of the arterial reconstruction thereby converting an ischemic into a hemorrhagic infarct and eventually into a massive hemorrhage in the area of infarction. It is beyond the scope of this report to give a critical analysis of the clinical and experimental data upon which this theory is based. Suffice it to say, it is not incontrovertible. Cerebral embolization, with or without operation,
can cause hemorrhagic cerebral infarction. A number of our patients, as well as those reported by others, had a fresh soft thrombus present at the carotid artery bifurcation that could easily have dislodged and traveled downstream during the performance of an operation, and this may be as significant a factor as restoration of flow in the production of hemorrhagic infarcts. Although the occurrence of hemorrhagic infarction has been well demonstrated by several authors, most of the reported patients had acute, profound neurological deficits, totally occluded carotid arteries, and hypertension. The patients in the present series differed in that the deficits were not fixed and were of only mild to moderate severity and the arteries operated on were not occluded. In addition, we carefully monitored and controlled the blood pressure throughout the periproductive period.

The pathological correlate of acute stroke is acute cerebral infarction, and in general, the more extensive the infarction, the worse the clinical stroke. Unfortunately, the pathological process responsible for the symptoms in our patients is not known. The fluctuating deficits preoperatively and their prompt and total resolution postoperatively suggest that infarction had not occurred or, if it had, that it was small. In spite of these excellent results, we believe the risk of emergency operation in these circumstances is probably higher than is elective operation in patients who are neurologically stable. However, the risk of not operating may even be greater for the prognosis of stroke in evolution is poor. Of 204 consecutive cases reported by Millikan in 1972, 69% became hemiparetic, 5% monoparetic, 14% died, and only 12% returned to normal by 14 days after the onset of symptoms. We adopted the more aggressive approach herein described after encountering several patients in whom the usual conservative plan of management allowed progression to a severe, fixed neurological deficit.

The primary objective of treatment in patients such as these is the prevention of cerebral infarction or the worsening of infarction after the pathological process begins. Since the very nature of progressing stroke allows only a limited amount of time in which to make initial observations and start treatment, the timing of surgical intervention, when appropriate, is of the utmost importance. The reluctance of neurologists and vascular surgeons to subject neurologically unstable patients to angiography does not appear to be supported by hard data. The risk of angiography is acceptably low in hospitals with skilled and experienced radiologists. Even in the Joint EAO Study, four vessel angiography had a grave complication (stroke) rate of 0.5% over-all and only 1% in patients with severe neurological deficits. Since that study is 10 years old and was done early in the experience of most of the participating angiographers, it is reasonable to assume that the risk is even lower at the present time.

On the basis of our initial experience with a smaller group of patients, we adopted an aggressive diagnostic approach in patients whose neurological status was rapidly changing or was atypical and not readily explained. Our subsequent experience has supported this approach. It includes patients with, as defined above, stroke in evolution, waxing and waning neurological deficit, and crescendo transient ischemic attacks. Figure 2 outlines our protocol for evaluating patients with neurological instability. The diagnostic workup should proceed on an urgent basis. Lumbar puncture, brain scan (static, flow, and computerized axial tomography), supraorbital directional Doppler, oculoplethysmographic, and EEG studies may be included, but the decision for arteriography should be based primarily on the clinical course. If a critical, unstable arterial lesion is found, emergency carotid bifurcation endarterectomy should be performed. If a noncorrectable or noncritical lesion is identified, heparin anticoagulation should be initiated since it appears to be associated with a reduction in the progression of infarction in these circumstances. We do not advocate angiography or operation in patients with severe fixed neurological deficits, especially when associated with depressed levels of consciousness. Presumably, cerebral infarction has already occurred in these individuals. Similarly, we do not advocate carotid endarterectomy to reestablish flow in occluded internal carotid arteries regardless of whether or not there are symptoms of cerebrovascular insufficiency.

References


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**Figure 2. Outline of recommended approach to patients with acute unstable neurological deficits.**

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