Intracranial Hemorrhage Following Surgical Revascularization for Treatment of Acute Strokes

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In the evaluation of the results of vascular reconstructive operations for cerebral vascular insufficiency it becomes necessary to divide the clinical syndromes of cerebral insufficiency into two broad categories. The category of patients for whom revascularization seems most helpful is represented by those individuals whose totally or partially occlusive lesions produce transient ischemic attacks or chronic low-grade insufficiency in the apparent absence of cerebral infarction. In this category, when occlusive lesions are present in operable extracranial arteries, improvement in cerebral function following successful operations ordinarily is subject to clear evaluation on the basis of definable changes in neurologic symptoms or findings. An early unfavorable neurologic result may be considered to be caused largely by an error in performance of operation.

The other category is represented by those patients whose occlusive lesions have caused frank cerebral infarction resulting in a fixed or progressive neurologic deficit. The numerous variations in the natural history of recovery of cerebral function in this group make it difficult to define the extent of improvement that properly may be credited to a successful arterial operation, particularly when operation is performed a few hours or days after the onset of a stroke. When improvement follows operation upon a patient with a recent acute stroke it may be difficult to determine whether this result represents a natural phase of recovery or whether it has occurred because of increased arterial flow to partially ischemic brain tissue at the periphery of the infarcted zone. Conversely, should further cerebral deterioration develop following operation it is similarly difficult to assess the extent to which the operation may have been a contributing factor. The vascular surgeon, confronted with a patient whose cerebral status worsens after operation may take refuge in the assumption that the unsatisfactory outcome was the result of widening destruction from the original infarct and that the operation itself failed only in that it was performed too late.

The purpose of this report is to present the clinical and pathologic observations on 5 patients with acute infarction in whom ultimately fatal brain damage appeared to be the direct result of the restoration of normal cerebral blood flow.

Of 179 arterial reconstructive operations performed at the University of California Hospital for the treatment of cerebral vascular insufficiency, 9 were performed upon patients who had had a recent stroke with varying degrees of neurologic residual. In 5 of these 9 patients, sudden, progressive and ultimately fatal cerebral deterioration appeared within 2 hours to 3 days after operation. In each, death apparently was caused by fresh cerebral hemorrhage. Following are the case reports of these patients.

Case Reports

Case 1. A 50-year-old man was admitted 3 days after the onset of an acute stroke. The first symptom was sudden and complete loss of consciousness. Partial consciousness was regained after 3 hours at which time flaccid paralysis of the left arm and leg was apparent. During the following 3 days there was a gradual decrease of somnolence and slight return of motor function.

Examination. At entry a conscious but drowsy man exhibited a partial left hemiparesis. Although speech was slurred, he appeared to comprehend questions. His blood pressure was 138/92. The

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sclerotic lesion was removed from the orifice of the right internal carotid artery. A fresh thrombus was extracted from the distal internal carotid artery. Brisk back-bleeding developed immediately. After closure of the arteriotomy and release of the occluding clamps a normal pulsation appeared in the distal internal carotid artery.

Course. Slight improvement in motor function and further lessening of somnolence was evident for the first 36 hours after operation. At the end of this time, rapid return of lethargy associated with an increase in the degree of paralysis of the left side occurred. Within a few hours the patient became comatose. The right pupil was dilated. Ophthalmodynamometry showed equal pressures in the vessels of each retina. Lumbar puncture revealed red blood cells in the spinal fluid and a pressure of 300 mm. H₂O.

Hypothermia to 31°C. was instituted; respiratory paralysis was treated by intubation and a respirator. On the 7th postoperative day the patient died.

Autopsy showed a large area of infarction and softening in the right hemisphere with massive fresh hemorrhage at that site (Fig. 2).

Case 2. A 56-year-old man entered with a 48-hour history of paralysis of the right arm and leg, facial asymmetry and expressive aphasia. The patient appeared alert but was unable to speak.

Examination. Blood pressure was 150/100. There was a right-sided flaccid hemiparesis and a fairly complete right homonymous visual field defect. Ophthalmodynamometry showed blood pressure in the right eye of 115/52 and in the left of 64/31. Carotid arteriogram showed complete occlusion of the left internal carotid artery.

Operation. Two days after entry a left carotid thromboendarterectomy was performed under local anesthesia. A completely occluding arterio-

optic disks were flat. An arteriogram performed under local anesthesia showed complete occlusion of the right internal carotid artery (Fig. 1).

Operation. A right carotid thromboendarterectomy was performed on the day of entry under local anesthesia. A completely occluding arterio-

![Fig. 1. Case 1. Carotid arteriogram showing complete occlusion at orifice of right internal carotid artery.](Image)

![Fig. 2. Case 1. Gross section of brain showing massive hemorrhage and infarction in right hemisphere.](Image)