Surgical Decompression for Cerebral and Cerebellar Infarcts

BY HENRIQUE S. IVAMOTO, M.D., MITSUO NUMOTO, M.D., PH.D., AND R. M. PEARDON DONAGHY, M.D.

Abstract:
Surgical Decompression for Cerebral and Cerebellar Infarcts

Following an infarction in the right cerebral hemisphere, massive cerebral edema developed in a woman and led to a marked intracranial hypertension and to a progressive "uncal and central herniation." The downhill course was reverted with a decompressive operation. Epidural pressure was monitored during the procedure and in the postoperative period. She showed progressive recovery from the initial motor, sensory, and visual field losses. Among 18 reported patients with cerebral infarction who underwent surgical decompression, 11 presented pupillary asymmetry, 14 were in stupor or coma, and 18 were either hemiplegic or hemiparetic preoperatively. Seven died following the operation. Among four cases of cerebellar infarction who underwent similar procedures, there were three survivors. Due to the lack of a controlled study the data available so far are not conclusive as to the beneficial effects of surgical decompression for cerebral or cerebellar infarcts. Some data of clinical relevance on cerebral edema and intracranial hypertension associated with cerebral infarction are reviewed.

Additional Key Words: cerebral and cerebellar edema, intracranial hypertension, herniation, monitoring of intracranial pressure.

Introduction

It is now well accepted that "edema is a consistent accompaniment of large cerebral infarcts and is a major cause of disability and death." Surgical decompression in completed ischemic stroke is controversial. We recently felt compelled to perform decompression in a case of right cerebral infarction. This prompted us to review the literature.

Case Report

HISTORY
A 49-year-old right-handed housewife began having transient episodes of weakness and numbness of the left hand around February 6, 1973. On the evening of February 12, she became hemiplegic on the left side and slightly obtunded. She was admitted to a hospital where a lumbar puncture revealed an opening pressure of 220 mm Hg and yielded a crystal-clear fluid. On the following night she was transferred to our hospital.

EXAMINATION
On February 13, she was sleepy but easily arousable, with a flaccid hemiplegia, Babinski sign, anoxia, homonymous hemianopia on the left side, and no papilledema. A brain scan demonstrated increased activity in the territory of distribution of the right middle cerebral artery, more densely at the base of the Sylvian fissure.

PREOPERATIVE COURSE
During the morning of February 15 she became stuporous, with decorticate responses on the left side, Cheyne-Stokes respiration, a pulse rate of 44, and blood pressure of 120/75 mm Hg. The right pupil measured 4 mm in diameter reacting sluggishly to light, whereas the left pupil measured 3 mm and reacted well. A "combined uncal and central herniation was developing." After 500 cc of a 20% mannitol solution had been given intravenously, the patient showed some improvement but remained stuporous. A right carotid angiography was performed (figs. 1 and 2). With the diagnosis of cerebral infarction with associated massive cerebral edema she was taken to the operating room.

OPERATION
Surgery was performed under local anesthesia, Innovar® (fentanyl and droperidol), and oxygen administered through a nasal catheter, on the afternoon of February 15. A wide scalp incision was made on the right side. A fiber optic pressure switch, which is a variant of the Numoto pressure switch, was introduced in the epidural space through a burr hole made in the posterior portion of the wound (fig. 3). The initial intracranial pressure was 1,600 mm Hg. The systemic blood pressure then was 130/80 mm Hg. The arterial Pco, was 33 mm Hg, the Po, 242 mm Hg, and the pH 7.47. A large craniotomy and a subtemporal craniectomy were made, followed by a dural incision at the anterior temporal lobe region. Necrotic tissue extruded spontaneously. The intracranial pressure, which had been stable at the initial level, began to drop sharply following the dural incision. The anterior 6 cm of the temporal lobe were found to be necrotic and therefore were removed, causing no bleeding. Replacing the bone flap raised the pressure from 450 to 850 mm Hg. Therefore, the wound was closed without the bone flap. Following elevation of the head and trunk, the pressure declined from 450 to 200 mm Hg.

From the Division of Neurosurgery, University of Vermont College of Medicine, Burlington, Vermont 05401.
POSTOPERATIVE COURSE
The patient began to wake up on the same afternoon, and on the following morning (February 16, 1973) was alert and oriented, with normal pupils and respiration. She was kept initially in a semi-sitting position. The pressure declined progressively to a range of 160 to 300 mm Hg in the supine position. The pressure switch was removed four days after surgery, and on March 9, 1973, she was discharged.

In May, 1973, she was readmitted and had the bone flap replaced. She was fully oriented, with left homonymous hemianopia, slight to moderate weakness of the left lower extremity, marked weakness of the left upper extremity, slight weakness of the left side of the face, and impaired sensory functions on the left side, particularly position sense. She had been using a walker. A psychological evaluation revealed a verbal IQ of 129, a performance IQ of 110, and a full scale IQ of 121, which placed her at the 92nd percentile of the general adult population. She was depressed because of her physical condition, showed some impairment of adaptive abilities and some constructional dyspraxia.

In July, 1973, occasional mild seizures developed which involved the left side.

In September, 1973, a continuous mild recovery of motor, sensory, and visual functions was noted. A radiograph of the skull showed a partial reabsorption of the bone flap. She still needed a walker, but had been doing some housework and bookkeeping.

Discussion
EDema AND INTRACRANIAL HYPERTENSION RELATED TO CEREBRAL INFARCT
Shaw et al.4 concluded that swelling of the infarcted brain is manifest in the first day, reaches a maximum between the third and fifth days, and disappears after about two weeks. Death during the first week is usually directly related to this swelling.

Plum5 found, in a series of 106 consecutive cases of acute hemispheric infarction producing major neurological deficits, 14 (13.2%) who had unequivocal clinical signs of significant swelling and an additional eight (7.6%) who presented possible signs of significant swelling. Most presented the central syndrome of progressive brain stem dysfunction and only a few had uncal herniation. Among the 14 cases mentioned above, six (44%) died. Three of the six also suffered from potentially fatal systemic illness.

Ng and Nimmanitya6 found a large infarction, severe brain swelling, and marked herniation in 45 (13%) of 353 consecutive cases of supratentorial cerebral infarction, who underwent autopsy. Twenty-seven (7%) could be considered to have died from acute swelling with transtentorial herniation.

Lascelles and Burrows,7 investigating 59 cases of occlusion or stenosis of the middle cerebral artery, found hemisphere displacement in six, the degree varying from 3 to 14 mm. All six patients died within 15 days of the onset. Hemisphere displacement was felt by the authors to be the most valuable radiological feature from the prognostic viewpoint. Similar cases may survive.

Experimental investigations have demonstrated that gradients of pressure develop within the intracranial space, being higher on the side of the infarct.
than on the opposite side, and in the posterior fossa. Brock et al. found that, although an asymmetrical elevation of the intracranial pressure was always present, the higher pressure could be either on the side of the infarcted hemisphere or on the opposite side.
The pressure switch was implanted in the epidural space before the craniotomy was performed.

Cerebral perfusion is reduced with very high intracranial pressures, especially with mass lesions and with cerebral edema. Cerebral perfusion pressure in the presence of intracranial hypertension can be calculated by subtracting the mean intracranial pressure from the mean systemic arterial pressure. Intracranial pressure can be measured at different structural levels within the intracranial space. Ventricular fluid pressure has been found to represent from approximately 80% to 100% of the epidural pressure measurements. In our case, the epidural pressure was 115 mm Hg initially, which would correspond to a ventricular fluid pressure anywhere from about 92 to 115 mm Hg. The mean systemic arterial pressure at that time was 96 mm Hg. Therefore, the cerebral perfusion pressure was very low, at least in the right hemisphere.

OUTLINE OF THE PATHOPHYSIOLOGY
Cerebral infarct associated with cerebral edema and marked elevations of the intracranial pressure causes ischemia of the neighboring tissues and may conceivably cause further infarction. Shifts of the brain through the various dural compartments can cause ischemia and infarction in other areas. Ischemia, infarction, and hemorrhage may occur in the brain stem. Shifts also can aggravate the intracranial hypertension by distorting and blocking cerebrospinal fluid pathways. Intracranial hypertension favors shifting. Eventually, a vicious cycle may be formed, as outlined in figure 4.

SURGICAL DECOMPRESSION FOR CEREBRAL INFARCTS
We decided to try a surgical decompression in this case because of a progressive herniation that did not respond satisfactorily to medical treatment. We found 17 cases of cerebral infarct with edema causing pressure effects who underwent decompressive surgery, reported by King, Schneider and Lemmen, Scarcella, and Greenwood. A case reported by Smiley (case 1) was not included here because the possibility of encephalitis was not ruled out.

Adding our case to these, we have a total of 18 cases. The average age is 50 years, ranging from 24 to 64. The etiology was spontaneous cerebral infarction in 15 cases, aneurysm surgery in two, and traumatic occlusion of an internal carotid artery in one. Eleven cases showed pupillary asymmetry, 14 were in stupor or coma, and 18 were either hemiplegic or hemiparetic.
SURGICAL DECOMPRESSION FOR CEREBRAL AND CEREBELLAR INFARCTS

CEREBRAL INFARCTION

SWELLING

HERNIATION

INTRACRANIAL HYPERTENSION

ISCHEMIA, HEMORRAGES

ISCHEMIA

DEATH

FIGURE 4

Events that may follow cerebral infarction.

prior to surgery. Shift of the midline structures was present in 16 cases and unverified in two. The degree of shift, reported in 14, varied from 4 to 15 mm, with an average of 6 mm. The operative results are summarized in table 1. The mortality rate was 39%. The survivors were either moderately or severely disabled. It must be stressed that there were variations in surgical techniques, etiology, and general condition of the patients.

The experiences of the others and our own suggest that a large craniotomy and removal of all necrotic tissue are important. Examination of the tentorial edge has been advised for all cases in which the temporal lobe is involved. Greenwood stated “a continuous, intracranial pressure monitoring system is desperately needed.” Lundberg briefly mentioned the usefulness of recording ventricular fluid pressure in stroke. The device we used is very easily implanted in the epidural space through a single burr hole, is safe, and can be removed quickly with no need for a reoperation. In the present case it was particularly helpful in planning the size of the craniotomy, indicating the need to leave the bone flap out, and in following up the pressure during the postoperative period. The value of an epidural pressure monitoring device as a guide during decompressive surgery will be discussed in another communication.

SURGICAL DECOMPRESSION FOR CEREBELLAR INFARCTS

Four patients with cerebellar infarct and pressure effects who underwent surgical decompression were reported by Fairburn and Oliver (case 2), Lindgren (cases 1 and 2), and Greenwood. Case i presented by Fairburn and Oliver was regarded as having recovered spontaneously and therefore was not included here.

The average age was 49 years, ranging from 33 to 69. The results are summarized in table 2. One of the four patients died of pulmonary embolism. One of the three survivors made a complete recovery from the intracranial lesion but died from coronary thrombosis four months after the operation.

Conclusions

The old concept of cerebral infarct as a nonexpanding lesion has been repeatedly proved to be incorrect. Large cerebral infarcts are associated with a significant cerebral edema which may cause severe pressure effects. A device to monitor the intracranial pressure could be used throughout the acute phase in such cases because it would enable the physician to follow that important parameter and estimate accurately the effects of his treatments. Due to the lack of a study including a control group the data available so far are not conclusive as to the benefits of surgical decompression for cerebral or cerebellar infarcts.

References

4. Shaw CM, Alvord EC Jr, Berry RG: Swelling of the brain following

---

TABLE 1

Results in 18 Patients With Cerebral Infarction With Significant Brain Edema Who Underwent Surgical Decompression

<table>
<thead>
<tr>
<th>Preoperative consciousness</th>
<th>Died</th>
<th>Survived</th>
</tr>
</thead>
<tbody>
<tr>
<td>Stupor or coma (14)</td>
<td>6</td>
<td>8</td>
</tr>
<tr>
<td>Alert (4)</td>
<td>1</td>
<td>3</td>
</tr>
<tr>
<td>Total (18)</td>
<td>7</td>
<td>11</td>
</tr>
</tbody>
</table>

TABLE 2

Results in Four Patients With Cerebellar Infarction With Significant Brain Edema Who Underwent Surgical Decompression

<table>
<thead>
<tr>
<th>Preoperative consciousness</th>
<th>Died</th>
<th>Survived</th>
</tr>
</thead>
<tbody>
<tr>
<td>Alert (1)</td>
<td>0</td>
<td>1</td>
</tr>
<tr>
<td>Stupor (3)</td>
<td>1</td>
<td>2</td>
</tr>
</tbody>
</table>


Surgical Decompression for Cerebral and Cerebellar Infarcts
HENRIQUE S. IVAMOTO, MITSUO NUMOTO and R. M. PEARDON DONAGHY

Stroke. 1974;5:365-370
doi: 10.1161/01.STR.5.3.365

Stroke is published by the American Heart Association, 7272 Greenville Avenue, Dallas, TX 75231
Copyright © 1974 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/5/3/365

Permissions: Requests for permissions to reproduce figures, tables, or portions of articles originally published in Stroke can be obtained via RightsLink, a service of the Copyright Clearance Center, not the Editorial Office. Once the online version of the published article for which permission is being requested is located, click Request Permissions in the middle column of the Web page under Services. Further information about this process is available in the Permissions and Rights Question and Answer document.

Reprints: Information about reprints can be found online at:
http://www.lww.com/reprints

Subscriptions: Information about subscribing to Stroke is online at:
http://stroke.ahajournals.org//subscriptions/