Surgical Treatment of Cerebellar Infarction

Roberto C. Heros, MD

Although carotid endarterectomy surgery plays a significant role in the prevention of ischemic stroke, the treatment of established ischemic stroke is usually medical. However, the one exception in which surgery plays an important therapeutic role is cerebellar infarction. In this issue, Chen and his colleagues present 11 patients with cerebellar infarction who were treated surgically because of progressive deterioration in spite of maximal medical therapy. In the opinion of the attending clinicians, all these patients would have died without surgery. All survived and improved, although three remained functionally dependent.

No previous report has documented so well the late clinical condition of each patient in reference to both the condition on admission and the immediate condition before surgery. However, the results achieved by Chen and coworkers are by no means unique. There have been numerous anecdotal reports of similarly favorable responses to decompressive surgery since the initial reports by Fairburn and Oliver and by Lindgren in 1956. The results of surgery have been so consistently favorable in patients who clearly were progressively deteriorating that it seems fair to say that this is one surgical indication that does not need the scrutiny of a randomized study.

However, the timing of surgical intervention remains controversial. The article by Chen et al appears to suggest that surgery should be considered only as a lifesaving last resort when all medical means fail and death appears imminent. Their results under these circumstances are surprisingly good. However, their Table 1 may be more reflective of their clinical indications than the text. It indicates that, in fact, they did not wait to operate until the patients were moribund, and most patients had Glasgow Coma Scale (GCS) grades of 7 or 8. It should be noted, parenthetically, that there is a problem with using the GCS to evaluate patients with posterior fossa masses. The GCS was designed to evaluate patients with head injuries, most of whom deteriorated from supratentorial pathology with consequent transtentorial herniation. This scale appropriately emphasizes level of consciousness and motor response. It is well known that unresponsiveness and posturing have different prognostic implications when the cause is a posterior fossa mass rather than herniation from a supratentorial mass.

See p 957

Since a decompressive suboccipital craniectomy with removal of infarcted cerebellar tissue is not a formidable procedure and carries relatively little intrinsic morbidity, there is no reason to wait until signs of either downward tonsillar herniation or upward transtentorial herniation occur, as the authors seem to suggest. It is possible that their three patients who remained in a "dependent" state may have fared better with earlier surgical intervention. There is considerable support for proceeding with decompression as soon as signs of direct brain stem compression appear.

The clinical syndrome of cerebellar infarction was well established by several early reports about twenty years ago. Little new has been added since. The early symptoms (headache, dizziness, nausea, vomiting, and loss of balance) and signs (truncal and appendicular ataxia, nystagmus, and dysarthria) are intrinsic to the cerebellar lesion. Later on, as cerebellar edema progresses, there is direct compression of the brain stem, which initially affects the posterior aspect of the pons, leading first to a paresis of the VI cranial nerve and soon to a complete loss of ipsilateral gaze (from compression of the nucleus of VI and the lateral gaze center) and peripheral facial paresis (from compression of the facial colliculus). At this stage, there may also be some confusion and drowsiness from hydrocephalus. It is at this point that if medical measures have failed, we recommend surgical intervention. The results of suboccipital decompression have almost invariably been excellent when the patient is operated on at this stage.

It has been estimated that without surgery, about 80% of patients who have developed signs of brain stem compression will die, usually within hours to a few days. The unoperated patient can progress rapidly to later stages of brain stem compression, with the appearance of Babinski's signs and hemiparesis, profound drowsiness, and small but still reactive pupils. This is followed within hours by coma, posturing, or flaccidity and ataxic respirations. Even though patients who reach this stage rarely make a complete recovery, even with surgery, a surgical decompression is still indicated, provided this condition is of very recent onset. There are several anecdotal reports of patients in profound coma preoperatively who made a useful recovery with prompt decompression. With proper respiratory support, useful recovery can take place in these patients because the comatose state, at least initially, is due to extrinsic compression of the stem without necessarily any irreversible intrinsic damage. Also important is that there is no intrinsic damage to supratentorial telencephalic structures in these patients. Full intellectual and cognitive function can be
recovered, even if there are persistent focal brain stem deficits.

One important clinical point that the authors mention is the frequent coexistence of a lateral medullary infarct and a cerebellar infarct. This is not surprising because most symptomatic cerebellar infarctions are due to vertebral occlusion involving the origin of the posterior inferior cerebellar artery, which also supplies the lateral medullary region.7 The clinical challenge is to distinguish focal brain stem symptoms and signs typical of the lateral medullary infarct (dysphagia, dysarthria, Horner's syndrome, crossed sensory loss, and ipsilateral facial numbness13) from those, already described, that occur later as a result of pontine compression by the swollen cerebellum. The former set of symptoms and signs are usually present at the onset, are not accompanied by a change in sensorium, and, of course, do not call for surgical intervention because they are due to intrinsic brain stem ischemia rather than to secondary compression.

The authors emphasize that the CT scan can be negative or only very subtly positive during the early stages of the clinical evolution of these patients. It takes several hours for the infarct to become visible, but even later, because the infarct can be mildly and diffusely hemorrhagic, it may be relatively isodense. Subtle signs of posterior fossa "tightness," such as disappearance or compression of the cisterns and fourth ventricle, must be sought. Chen et al did not use MRI in their patients, but there is good reason to hope that with increased use of this modality, these infarctions will be recognized earlier.14

In their discussion of etiology, Chen et al mention that cerebellar infarction may result from chiropractic manipulation of the neck. Indeed, the scenario of a patient with chronic neck pain who develops dizziness, nausea, and loss of balance after vigorous chiropractic manipulation of the neck and later deteriorates as a result of cerebellar infarction is familiar to many neurologists and neurosurgeons.12 In these cases, the cerebellar infarct, which may also be accompanied by lateral medullary infarction, is usually due to traumatic dissection of the vertebral artery secondary to forceful abrupt cervical hyperextension, a common chiropractic maneuver.15

In reference to surgical treatment, I agree with Chen et al that the operation of choice is a suboccipital decompression carried through the foramen magnum to relieve tonsillar herniation and with removal by suction of the grossly infarcted cerebellar tissue that usually exudes "like toothpaste" upon opening of the dura.49 I also agree that ventricular drainage should not be used alone without prompt suboccipital decompression because of inadequate relief of the direct compression of the stem and the risk of upward herniation with ventriculostomy.

References
Surgical treatment of cerebellar infarction.

R C Heros

*Stroke*. 1992;23:937-938
doi: 10.1161/01.STR.23.7.937

*Stroke* is published by the American Heart Association, 7272 Greenville Avenue, Dallas, TX 75231
Copyright © 1992 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/23/7/937.citation

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/