Cranial Nerve Paralysis Following Carotid Endarterectomy

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SUMMARY During the past seven years 347 patients have been entered into a data bank at the Duke University Medical Center for evaluation of transient neurologic ischemia. One hundred fifty eight of these patients underwent carotid endarterectomy for transient ischemic attacks. Another complication which merits additional investigation is the occurrence of peripheral neurologic deficits that may lead to functional and cosmetic defects. Surprisingly little emphasis has been given to this problem and this has prompted the present investigation of cranial nerve symptoms in a group of patients undergoing carotid endarterectomy for transient ischemic attacks.

Methods and Results

Since 1973 the Division of Neurology of the Duke University Medical Center has maintained a computerized data bank of patients with symptoms of transient neurologic ischemia. Certain patients among the group had carotid endarterectomy if they had appropriate extracranial lesions. Three hundred forty seven patients with focal transient cerebral ischemic symptoms were entered into this program from 1974 to 1978, and one hundred fifty eight of these patients underwent carotid endarterectomy. Neurological follow-up revealed that 24 (15.1%) of the operated patients had 26 (16.4%) peripheral cranial nerve palsies. Injury to the peripheral portion of the hypoglossal nerve was noted in 13 patients, to the cervical branch of the facial nerve in five and to the recurrent laryngeal nerve branch of the vagus in eight. Complete recovery of nerve function usually occurred within four months but residual deficit was present at one year in two patients with facial nerve and four with hypoglossal nerve involvement. Even though these complications of carotid endarterectomy are generally benign and transient, the frequency of occurrence can be reduced if careful attention is given to anatomic localization of the cranial nerves during surgery.

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year period in this selected group of 24 patients; four myocardial infarctions and two strokes.

Among the patients with hypoglossal nerve palsy, eight had complete resolution of symptoms and four had residual signs at six and twelve months follow-up. Two patients complained of mild dysarthria and decreased ability to use the tongue. Three of the five patients with cervical branch of the facial nerve involvement had complete resolution but two had residual deficit in movement of the lower face one year after surgical procedures. All of the patients with vocal cord paralysis had complete resolution of hoarseness within six months of operation.

A review of the operative procedures revealed no correlation between subsequent cranial paresis and aberrant location of nerves or the presence of distal carotid disease requiring high carotid dissection with unusual retraction.

Discussion

The frequency of occurrence of distal cranial nerve palsies in this series is similar to that previously reported (table 1). Hertzer, et al found 30 patients among 240 operations (12.5%) experienced 38 cranial nerve injuries (15.8%) consisting of recurrent laryngeal nerve in 14, hypoglossal in 13, marginal mandibular branch of facial nerve in six and superior laryngeal in five. All but three resolved in twelve months. Matsumoto, et al reported 16 cases (12.3%) in 130 consecutive carotid endarterectomies. They observed 11 cases of hypoglossal nerve palsy, three cases of vocal cord palsy and two cases of facial nerve paralysis. Although some of their patients required two to three months to clear, all resolved. Evans, et al described motor speech deficits in 20 of 86 patients following endarterectomy (23.2%) with involvement of nerves IX, X, and XII. Follow-up up to six weeks showed some deficits remained. In our series, four of the 12 cases of hypoglossal nerve paralysis had demonstrable residual deficit at one year follow-up. Among the five patients with facial nerve involvement, two cases showed residual paralysis. However, all of the eight patients with vocal cord paralysis showed resolution.

Since these cranial nerve palsies are related to trauma, operative technique has bearing on the incidence and severity of this complication. Surgeons must have an exact understanding of the position and the function of peripheral cranial nerves in the neck before undertaking such operations (fig. 1).

The vagus nerve usually lies posterior and lateral to the internal and common carotid but may be positioned differently in the carotid sheath. During carotid artery dissection, trauma may occur if the dissection is not kept close to the wall of the artery. The recurrent laryngeal nerve can also be injured if it follows an anomalous nonrecurrent course, traversing behind the common carotid. In both cases vocal cord palsy is the result. If dissection around the carotid bifurcation and superior thyroid artery is not kept close to the arterial wall, trauma to the superior laryngeal nerve may occur, resulting in difficulty in swallowing, and change in the quality of the voice due to cricothyroid muscle dysfunction.

The hypoglossal nerve descends along the course of the internal carotid passing medially over the more superficial external carotid. If this structure is not mobilized correctly, traction on the nerve can cause tongue deviation to the side ipsilateral to the endarterectomy. The ansa hypoglossus originating at various

<table>
<thead>
<tr>
<th>Table 1</th>
<th>Reports of Cranial Nerve Palsies Following Endarterectomy</th>
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<tbody>
<tr>
<td>Number of endarterectomies</td>
<td>Total number of palsies</td>
</tr>
<tr>
<td>Matsumoto et al (1977)*</td>
<td>130</td>
</tr>
<tr>
<td>Hertzer et al (1980)*</td>
<td>240</td>
</tr>
<tr>
<td>Evans et al (1982)*</td>
<td>86</td>
</tr>
<tr>
<td>Massey et al (1982)</td>
<td>158</td>
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*Only motor speech deficits examined
positions along the nerve course may lie in the field. Injury to the ansa is inconsequential.

The carotid sinus nerve, originating from the glosso-pharyngeal nerve lies in the tissue between the carotid bifurcation. Dissection in this region can result in stimulation of the nerve leading to hypotension and bradycardia. Mediated through the vagus nerve, this reflex is transient and no significant peripheral paralysis results.

The cervical branch of the facial nerve lies beneath the platysma inferior to the angle of the jaw. In some individuals it sends twigs to the mandibular branch of the facial nerve so that if the cervical branch is divided, ipsilateral lower lip paresis may result. The marginal mandibular nerve itself may be injured if the incision is carried too cephalad. Injury to the nerve can be avoided by posterior displacement of the incision as one approaches the angle of the mandible.

As indicated in this series, occurrence of distal cranial nerve palsy is a risk of surgery and patients should be informed of this possibility. Residual deficits can lead to functional and cosmetic difficulties that detract from the benefits of carotid endarterectomy. The fact that most of the deficits are transient indicates that they likely arise from a reversible injury following stretch, retraction or clamping of nerves rather than permanent division of these structures. Such brief deficits are well tolerated if they are unilateral.

References
Cranial nerve paralysis following carotid endarterectomy.
E W Massey, A Heyman, C Utley, C Haynes and J Fuchs

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