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Neurologic Changes during Carotid Endarterectomy under Cervical Block Predict a High Risk of Postoperative Stroke

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Background: **This study was undertaken to confirm a previous report that patients having neurologic changes with carotid artery clamping were at greater risk of developing permanent postoperative neurologic complications after carotid endarterectomy.**

Methods: **Superficial and deep cervical plexus blocks were performed in 389 patients undergoing carotid endarterectomy. The patients were premedicated and sedated to a level that allowed awake neurologic assessment. Intraoperative neurologic changes were recorded and all patients were examined postoperatively by an independent anesthesiologist to record postoperative neurologic outcome.**

Results: **Trial carotid artery cross clamping resulted in 24% of patients having neurologic changes that usually responded to declamping and shunt insertion. Postoperative permanent neurologic complications occurred in 2.6% of patients, but were more common in patients who had neurologic changes associated with carotid artery cross clamping (6.6% compared to 1.1%, $P < 0.01$). Thrombosis of the carotid artery was the most common finding in patients who underwent reexploration of the carotid artery after developing postoperative neurologic changes.**

Conclusions: **This study confirms that patients undergoing carotid endarterectomy under cervical plexus block who have intraoperative neurologic changes have a sixfold increase in the chance of developing a postoperative stroke. This high-risk group may benefit from antithrombotic therapies to improve their outcome. (Key words: Anesthetics, local: bupivacaine; lidocaine. Anesthetic techniques: cervical plexus block. Complications: stroke. Monitoring: awake neurologic assessment. Surgery, vascular: carotid endarterectomy.)**

REGIONAL anesthesia has been advocated for patients undergoing carotid artery surgery principally because it allows awake neurologic assessment of the adequacy of cerebral blood flow during carotid artery cross clamping.[1–5] Intraoperative neurologic changes have been reported to occur in 2.4–21% of patients,[5–13] but only one report correlates these changes with postoperative neurologic outcome.[8]

We prospectively studied all patients having their first carotid endarterectomy under regional anesthesia from October 1987, through December 1991, to determine whether intraoperative neurologic changes predicted patients who would have a higher risk of postoperative neurologic complications. This study was undertaken to confirm a previous report,[8] because if cross clamping the carotid artery did define a high-risk population, then strategies for improving outcome could be applied in a more rational manner.

Materials and Methods

The patients were assessed preoperatively, gave consent, and were premedicated 1 h before anesthetic preparation. Patients taking antianginal and antihypertensive therapy continued that medication on the day of surgery. Oxygen was administered *via* a face mask during the performance of the cervical plexus block and throughout the surgery. Patients were monitored clinically and with a lead V5 electrocardiogram, intraarterial blood pressure, and pulse oximetry.

Deep and superficial cervical plexus blocks were performed using the technique described by Moore.[14] The superficial cervical block was usually performed with 10–15 ml of 1.5% lidocaine with epinephrine 1:200,000 and the deep cervical block with 5–6 ml of the same solution at C2, C3, and C4. The surgeon supplemented the blocks with local infiltration of 0.5% lidocaine if this was necessary. During the surgery, sedation was provided with diazepam, midazolam, or fentanyl in doses aimed at preserving patient cooperation.

The patients were repeatedly assessed for neurologic changes during a 2-min trial of carotid artery cross

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clamping. If conscious state changed or contralateral grip strength decreased during this trial period, a shunt was inserted. Neurologic observations were continued every 5 min throughout the time of carotid artery clamping.

Cardiovascular management was aimed at maintaining heart rate and blood pressure within normal ranges. Bradycardia was defined as a heart rate of less then 60 beats/min and, if treatment was indicated, titrated intravenous atropine was administered. Tachycardia was treated with intravenous atenolol if the heart rate exceeded 90 beats/min. Systolic blood pressure was maintained between 100 and 180 mmHg, and hypotension was treated with intravenous metaraminol and/or blood volume support. Hypertension was treated with intravenous nitroglycerine or, if associated with tachycardia, intravenous atenolol.

Patients were given intravenous heparin 100 units/kg before cross clamping of the carotid artery, and anticoagulation was reversed with 1 mg/kg of protamine after closure of the arteriotomy. On completion of the endarterectomy, patients were given 500 ml of dextran 40 over 4 h to reduce platelet adhesiveness.

Patient followup was done by an independent anesthesiologist between postoperative days 2 and 5. Patients in whom surgeons diagnosed a neurologic change were reviewed by a neurologist. Patients developing permanent neurologic changes were serially examined until discharge from the hospital to determine the extent of recovery from that complication. All data were recorded prospectively on a specifically designed data sheet and analyzed on completion of the study.

The data were analyzed using Statview software (Abacus Concepts, Berkley, CA). Continuous data were compared using Student's *t* test. Nominal data were analyzed using chi-square testing with Yates' continuity correction for small numbers when required. A *P* value of less than 0.05 was considered to be significant.

Table 1. Clinical and Demographic Data

No. of patients	389
Mean age (yr) (SD)	67 (8)
Male:female	266:123
Presenting symptoms*	
Transient ischemic attacks	200 (51%)
Amaurosis fugax	135 (35%)
Cerebrovascular accidents	67 (17%)
Reversible ischemic neurologic deficit	22 (6%)
Asymptomatic	52 (13%)

* Some patients had multiple presenting symptoms.

Table 2. Premedication, Local Anesthetic, and Sedation Used

	No. of Patients (%)
Premedication	
Papaveretum/hyoscine	165 (42)
Temazepam	96 (25)
Other	121 (31)
Nil	7 (2)
Local anesthetic	
Lidocaine, mean dose 477 mg (SD 79)	361 (93)
Bupivacaine, mean dose 159 mg (SD 34)	28 (7)
Sedation	
Midazolam	119 (31)
Diazepam	41 (11)
Fentanyl	12 (3)
Midazolam and fentanyl	58 (15)
Other	11 (3)
Nil	139 (36)
General anesthesia	9 (2)

Results

Three hundred and eighty-nine patients having their first carotid endarterectomies under cervical plexus block were included in the study. Relevant clinical and demographic data are presented in table 1. The majority of patients had symptomatic disease and all the asymptomatic patients had stenosis of greater than 70%. The premedication, dose of local anesthetic used, and type of intraoperative sedation is shown in table 2. Nine patients required conversion to general anesthesia because of restlessness (five patients), claustrophobia (two patients), prolonged surgery (one patient) and distress associated with neurologic change (one patient).

During the operation, 24% of patients had neurologic changes after trial carotid artery cross clamping. The nature of the changes are shown in figure 1. Loss of consciousness was the most common change, but some patients had loss of grip strength without a change in conscious state. One patient had a seizure at the time of removal of a shunt, and this was treated by reestablishing the carotid circulation with a side clamp. Fourteen patients were not able to be assessed because nine had general anesthesia and five were too sedated for effective neurologic assessment.

In the majority of these patients, the changes were treated by intravascular shunt insertion (88 patients). The blood pressure was increased in three patients, reversing the change, and the management of the patient who had a seizure is described above. All the neurologic changes were reversed with this treatment except those in one patient. In 80 of the patients, the

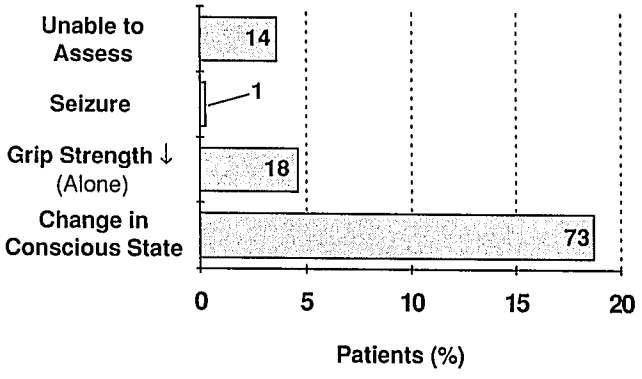

Fig. 1. Intraoperative neurologic changes after trial carotid artery cross clamping. Number of patients is shown in each column.

changes occurred within 2 min of trial cross clamping, but in 6 they occurred between 5 and 10 min after cross clamping, in 4 between 10 and 20 min after cross clamping, and in 2 more than 25 min after cross clamping. No significant changes occurred in the blood pressure to explain these late changes. Patients in whom the neurologic state could not be assessed had shunts inserted to restore carotid circulation during carotid surgery.

Thirty-four patients had postoperative neurologic complications (fig. 2). Twenty-four of these were temporary and resulted in full recovery, while the remaining ten patients had permanent neurologic complications. In 1 patient, the complication occurred during the operation; in 15, they occurred in the recovery room within 2 h of completion of surgery; and in 18, they developed 1–5 days postoperatively in the ward. Twelve patients with temporary neurologic changes had transient ischemic attacks and had full recovery, while the remaining 12 had neurologic changes that necessitated reexploration resulting in complete recovery from the complication. Thrombosis at the arterectomy site was found in nine of these patients and treated with thrombectomy and patch grafting. Three patients had patent carotid arteries with no obvious explanation for the neurologic problem. The patients who developed permanent neurologic changes had hemiparesis (four patients), monoparesis (five patients), and dysphagia (one patient). In six of these patients, reexploration of the surgical site occurred; four patients had thrombosis and two patients had no cause found. These reoperations did not result in full neurologic recovery.

Figure 3 shows the correlation of the intraoperative neurologic change with the postoperative neurologic outcome. Patients developing an intraoperative neurologic change were at a statistically significantly greater incidence of developing either temporary (11%) or permanent (6.6%) neurologic complications when compared with those patients who had no intraoperative neurologic change, in whom the risks were 5% and 1.1% respectively ($P < 0.05$ for temporary, $P < 0.01$ for permanent.)

Discussion

The development of neurologic changes after internal carotid artery cross clamping depends on the extent of atherosclerosis in the other extracranial vessels and the extent of the collateral circulation at the level of the Circle of Willis.[1] Postoperative stroke rate has been shown to increase from 0.6 to 28.4% in patients with contralateral carotid artery occlusion, persistent pre-

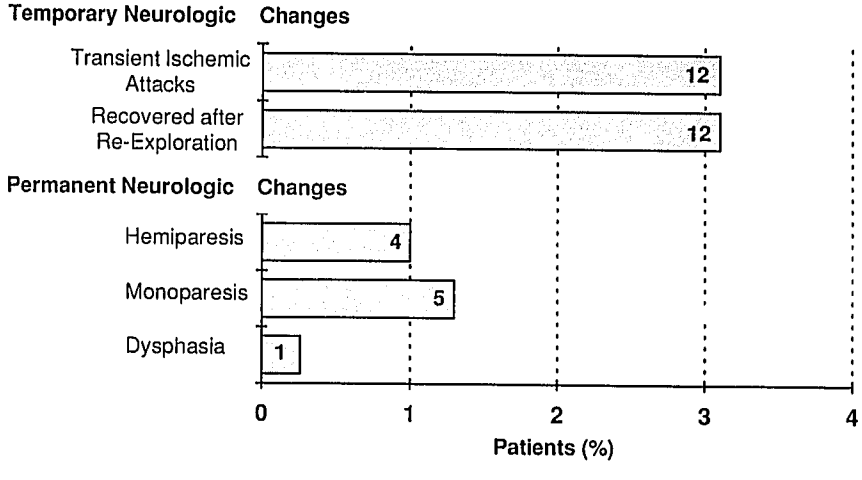

Fig. 2. Postoperative neurologic complications. Number of patients is shown in each column.

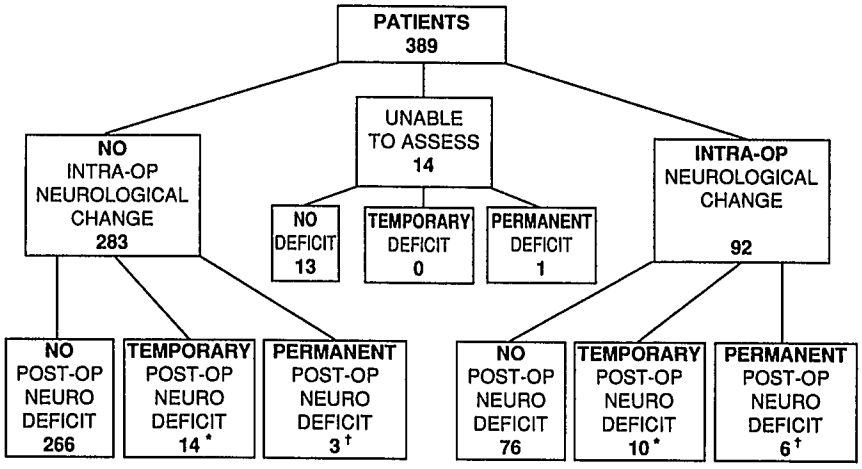

Fig. 3. Correlation of intraoperative neurologic change and postoperative neurologic outcome. Number of patients is shown in each box. *$P < 0.05$ for temporary neurologic complication. †$P < 0.01$ for permanent neurologic complication.

operative neurologic deficit, and carotid artery clamping intolerance.[8] This study confirms that patients anesthetized with cervical plexus block who have a neurologic change after cross clamping the internal carotid artery are at a greater risk of developing both temporary and permanent postoperative neurologic changes. The cause of these complications is multifactorial, but thrombosis at the surgical site is the most common.[15] Seventy-two percent of the patients in this study who were reexplored were found to have thrombosis in the carotid artery, which suggests that methods aimed at preventing thrombosis may be appropriate to reduce postoperative stroke rate. Nonreversal of the intraoperative heparin or full anticoagulation of patients developing clamping neurologic changes may be appropriate. The major concern with this approach would be the increased risk of neck hematoma.[16] Dextran 70 and 40 have been used to prevent thrombosis by decreasing platelet adhesiveness,[17] and have been shown to reduce deep venous thrombosis and arterial thrombosis.

Patients who develop neurologic changes after carotid artery cross clamping must have the poorest collateral circulation and, therefore, if thrombosis occurs at the endarterectomy site, they are more likely to develop neurologic deficits. Postoperative thrombosis may well have occurred in patients who tolerated intraoperative clamping, but this did not cause a neurologic deficit because of the adequate collateral circulation. Inability to tolerate carotid clamping readily identifies the high-risk group, and the high incidence of thrombosis justifies early surgical reexploration if a neurologic problem occurs.

Patients in this study had a 24% incidence of neurologic changes with carotid artery cross clamping compared with 9%,[5] 21%,[6] 19%,[7] 7.6%,[8] 14%,[9] 9.7%,[10] 9%,[11] and 2.4%[12] in other reports. Our greater incidence could be explained by a different severity of cerebrovascular disease, different cardiovascular management, or variations in approaches to intraoperative sedation.

Regional anesthesia for carotid endarterectomy is well accepted by patients; a previous report by our group[13] noted that 92% of patients would have the same anesthetic for a future carotid endarterectomy. These patients are fully alert after the operation; therefore, any neurologic changes are immediately apparent and surgical reexploration can be carried out without delay. Regional anesthesia for carotid endarterectomy has been shown to decrease length of stay in postoperative intensive care.[2] The use of regional anesthesia may also allow for selective admission to intensive care. Patients developing intraoperative neurologic change justify admission based on the high risk of stroke, but those that do not have neurologic change have a much reduced chance of stroke and may not require intensive care admission. This could contribute to cost saving with carotid endarterectomies in a cost-sensitive environment.

The increased incidence of stroke in the patients having intraoperative neurologic changes may be explained by the use of shunts in this group. However, shunt insertion reversed the intraoperative neurologic changes and thrombosis was found to be the commonest cause of postoperative neurologic problems in patients who were reexplored. The mechanism of neurologic complications caused by shunts is either embolic or carotid artery dissection,[1,4,7,8] but thrombosis could possibly occur as a result of intimal damage by the shunt.

This analysis of carotid surgery under regional anesthesia identifies patients at greater risk of developing permanent neurologic deficit. Patients who had neurologic change after carotid artery clamping had a 6.6% risk of permanent postoperative neurologic complications, whereas patients who did not have this intraoperative change were six times less likely to develop permanent neurologic complications. The outcome of this high-risk group may be improved by antithrombotic therapy, selective admission to intensive care, and early surgical reexploration.

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