Steal Phenomenon at Unclamping During Carotid Endarterectomy

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SUMMARY A patient is reported who during vigorous back bleeding after unclamping of the internal carotid artery during endarterectomy had EEG slowing and a postoperative increase in neurological deficit. This phenomenon, an apparent steal, has not been reported and suggests that EEG monitoring is as vital at unclamping as it is after clamping. The patient also raises questions about the risk of early endarterectomy for those who have persistent deficits, even with unobstructed vessels.

EVALUATION by electroencephalography (EEG) of adequate collateral flow while cross-clamping during carotid endarterectomy has become an integral part of operative technique. It helps to make the decision for the need of a vascular shunt. Usually the EEG changes rapidly if collateral flow is insufficient. In a recent patient, opening the internal carotid artery clamp during endarterectomy resulted in profuse backflow and was followed by slowing of a previously unremarkable EEG. The EEG rapidly became normal when the artery was again clamped. This episode suggests a steal phenomenon which, to our knowledge, has not been previously recognized.

Patient Report

The patient is a 54-year-old right-handed male with hypertension, diabetes and hypertriglyceridemia who had 2 episodes of weakness and numbness in his right arm over 2 weeks. On physical examination 3 days after the most recent attack, the neurological examination was normal except for slight weakness of the right hand and patchy areas of decreased sensation over the right hand. No carotid bruit was heard. Transfemoral 4-vessel cerebral angiography showed a long shallow ulcerated plaque in the left internal carotid artery just above the bifurcation. There was no other cerebrovascular lesion. The "watershed" between the middle and anterior cerebral artery circulations was in the mid-convexity. For the most part, the voltage decrease persisted and alternated with brief periods when the activity was unremarkable. The dominant activity on the left remained essentially the same over the entire parasagittal regions.

Anesthesia was maintained with 60 percent nitrous oxide and one percent halothane. The posterior activity was moderately well regulated 8 to 9 cps waves mixed with 5 and 6 cps theta activity. The voltages of these wave forms were similar over the entire parasagittal regions.

After administration of heparin, the external carotid artery was clamped with no visible change in the record. After the common carotid artery and internal carotid arteries were clamped, there was momentary bilateral voltage decrease posteriorly but no change in frequencies and no asymmetry. Throughout the initial period following artery clamping, the dominant posterior activity remained at 9 to 9 1/2 cps.

Seven minutes after clamping the internal carotid artery, the clamp was released in order to extend the endarterectomy distally. There was brisk backflow and no clots were seen. Systemic blood pressure remained stable. An asymmetry in the EEG became apparent at this time with decreased voltage of the faster frequencies in both the left parasagittal and temporal regions. There was some delta activity in the left posterior-temporal, mid-temporal area.

For the next minute this asymmetry showed variable persistence and alternated with brief periods when the left parasagittal activity was similar to that on the right. For the most part, the voltage decrease persisted and could be seen better in the temporal than in the parasagittal region.

The internal carotid artery was then reclamped, after which the asymmetry gradually decreased. The dominant activity on the left remained essentially the same as that on the right even though the voltages were lower. Next, faster frequencies became more apparent in the left temporal region. The degree of asymmetry in the parasagittal area greatly decreased following 14 minutes and 15 seconds after the initial clamping. At this point, 125 mg of pentobarbital was given for added protection against ischemic change.

This was followed by change in the dominant frequencies to a more irregular 8 cps pattern and by 17 minutes after the initial clamping the asymmetry was greatly reduced, although some posterior temporal amplitude attenuation was still visible.

Nineteen minutes and 20 seconds after the initial clamping, the clamps were removed. Seven minutes after the vessels were unclamped, the posterior dominant activity was 10 cps. While there was still slight voltage attenuation in the left temporal area...
FIGURE 1. Angiogram of the left common carotid bifurcation demonstrating a long shallow ulcerated plaque (arrow) at the origin of the internal carotid artery.

FIGURE 2. Angiogram demonstrating the "watershed" between the anterior and middle cerebral arteries high in the mid convexity region (arrow).

FIGURE 3. Two minutes and 40 seconds after the carotid artery was clamped there is normal symmetrical electrical activity.

FIGURE 4. a. When the internal carotid clamp was released for retrograde flushing, loss of voltage and underlying delta waves were observed in channels 3, 11, and 12 that were not seen in comparable areas on the right. b. Four minutes later, 11 minutes after initial clamping, there was continued asymmetry and delta activity, seen best in channels 11 and 12.
pared to the right there was no significant asymmetry (fig. 5a).

Once the patient awoke it was apparent that there was a marked weakness of the right arm. Within a few hours this weakness only involved hand movements. At that time a repeat EEG was done which showed intermittent temporal slow activity in the theta range, although the dominant frequencies on the two sides were symmetrical (fig. 5b). Within a few days the patient's strength was almost normal.

**Discussion**

Because this patient had had two apparent embolic episodes in fairly rapid succession, it was feared he might soon develop a major stroke. It was therefore decided to perform the endarterectomy early, even though he had some persistent neurological deficits. But it was recognized that the dangers of early surgery in such a patient are not fully understood. After a major completed stroke due to carotid occlusion, revascularizing infarcted brain may cause hemorrhage, and this risk may persist for 4 to 5 weeks. Patients with less marked neurological deficits do not tolerate early endarterectomy well even if their carotid arteries are not significantly obstructed. It is generally suggested that surgery be delayed 2 to several weeks under these circumstances. Certain risk factors might be related to the severity of neurological deficits, such as the degree of stenosis, rCBF, CT, EEG, and timing of operation, but this subject has never been systematically studied. Interest in very early surgery for acute and progressive strokes is being revived. The problem of early surgery is related to the degree of injury to a poorly functioning portion of the brain and its susceptibility to intraoperative ischemia during carotid occlusion. The fact that this patient's signs were relatively new and had not completely cleared indicated that his brain had not completely recovered and possibly explained his sensitivity to an apparent brief ischemic episode.

Several techniques have been used to try to predict whether a patient will tolerate carotid occlusion. Angiographic definition of collateral flow through the circle of Willis and determination of back pressure have been shown to be of variable value. EEG monitoring is considered by many, though not all, to be reliable. EEG changes generally occur early and reflect lowering of rCBF to ranges where there are abnormalities of electrical transmission but not necessarily infarction, and moderate decreases of flow may be tolerated for hours. This would explain the reversibility of both the EEG changes and clinical deficits in our patient.

The cause of this ischemic episode appears unique. Late EEG changes have been reported in one to 7 percent of patients and have been previously attributed to decreases in systemic blood pressure or to emboli. In this patient there was no change in systemic blood pressure, and no clots or debris were seen during back bleeding. Since the episode of ischemia occurred during release of the distal clamp, it would appear to be secondary to a steal phenomenon. The region of the brain most involved clinically was the hand area of the motor strip which was in the "watershed" between the anterior and middle cerebral circulation as demonstrated on the angiogram, and would logically be the area most affected in such a steal.

As soon as the changes in the record were reported, the clamp was replaced and the EEG changes began to diminish. It was also hoped that the administration of barbiturates would protect the ischemic brain. It has been reported that pentobarbital hastened the reduc-
tion of the asymmetry of EEG changes after carotid occlusion in one case. By the end of the procedure the asymmetry, although still evident, was quite subtle and was not felt to represent a significant EEG finding. The patient's right arm was weak postoperatively, but, as expected, rapidly improved.

This patient demonstrated the value of EEG monitoring during endarterectomy and helped document an unexpected ischemic event. This experience suggests that there is a second "high risk" portion of the procedure during unclamping of the internal carotid artery. Reducing the chance of emboli by opening the distal clamp and allowing back bleeding is an integral part of the procedure, but when back flow is initiated, it is important to be alert to the possibility of reduced cerebral blood flow and to carefully monitor the EEG.

References

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