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Case Report
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# Seizures after a Carotid Endarterectomy are not due to Hyperperfusion but to Vascular Steal Activity

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## Abstract

This paper advances the idea that carotid endarterectomy operations can cause seizures that occur after the operation, but the cause of the seizure is not due to hyperperfusion but apparently to an inter cerebral steal phenomenon that develops between non-ischemic and hypoxic neurons. Cerebral hyperperfusion begins immediately after a carotid endarterectomy, but within days there is a return to normal cerebral perfusion. Of interest is that so-called hyperperfusion seizures do not occur early after carotid surgery even though hyperperfusion is at its height during this time period. A hypothesis is presented based on Bernoulli's Law that cerebral blood flow after a carotid endarterectomy is disproportionate in the volume of blood flow to normal areas of the brain compared to chronically ischemic locations. This allows for a continued disparity between the blood flow between non-ischemic and neuro-electrically unstable hypoxic neurons. Hypoxic neurons which are known to be neuro-electrically irritable appear to be the source of seizure activity suggesting, therefore, that seizures following a carotid endarterectomy are not due to hyperperfusion but to vascular steal activity.

**Keywords: Carotid endarterectomy; Hyperperfusion; Vascular steal action**

## Introduction

A seizure that occurs after a carotid endarterectomy is a rare but serious post-operative complication. It is generally believed that these seizures result from post-operative hyperperfusion. This paper advances the idea that carotid endarterectomy operations can cause seizures that occur after the operation, but the cause of the seizure is not due to hyperperfusion but apparently to an inter cerebral steal phenomenon that develops between non-ischemic and hypoxic neurons. This hypothesis is based on physiological principles and the personal observation of a stroke patient who underwent omental transposition for brain revascularization [1].

## Case Presentation

The patient was a 65-year-old Catholic nun who developed a left middle cerebral artery occlusion which resulted in a severe speech deficit, a marked right hemiparesis and the inability to read. To improve these neurological disabilities, the patient underwent omental transposition to her left cerebral hemisphere two-and-a-half years after her stroke. On the sixth post-operative day, she suddenly became completely aphasic for two hours at the end of which time her speech abruptly returned. On the following day, she again developed instantaneous and complete aphasia. This disorder continued for six days after which her speech abruptly returned. Of marked interest was the fact that even though the patient was totally aphasic for six days, she could communicate during this period if she sang the words she wished to express. It was learned that this phenomenon is the basis for musical into national therapy, which is used by speech pathologists treating aphasic patients. It appears that a cerebral location becomes functional for the singing of words in a cerebral area distant from the major but non-functional speech center.

Over a thirteen-year post-operative period the patient experienced no aphasia and had excellent subjective and objective improvement in her walking ability and in her speech. In addition, she regained her ability to read, which she had been unable to do for two and a half years following her stroke.

For this Catholic nun, being able to read once again was as important to her as her speech improvement. She was one of five stroke patients treated by the author who had been unable to read for years following their strokes, but within two to six weeks after omental transposition to the brain, all five could read.

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### Post-operative hyperperfusion

Seizures following a carotid endarterectomy have generally been regarded as due to cerebral hyperperfusion. If hyperperfusion is the basis for these seizures, certain physiological events must be explained. Powers and Smith using transcranial Doppler techniques showed that the post-operative blood flow velocity within cerebral vessels ipsilateral to a carotid endarterectomy increased up to 400% [2]. This increased blood flow velocity, however, returned to normal by the second post-operative day. Sundt et al. reported a greater than 100% increase in CBF in their patients who developed seizures following carotid endarterectomy [3]. Shroader et al. reported even greater than 200% CBF levels following carotid endarterectomy, but the hyperperfused state markedly receded within days of surgery and had returned to a normal blood flow state by the end of the first post-operative week [4]. If post-operative seizures following a carotid endarterectomy are the result of cerebral hyperperfusion, it would seem that seizures would likely occur during the early post-operative days when cerebral hyperperfusion is at its maximum rather than occurring most commonly during the second post-operative week by which time CBF has returned to normal.

### Physiological consideration

Levels of pCO2 and pO2 are important factors that control local CBF. When a particular region within the brain increases its neuronal activity, CBF and pO2 levels rise. A pO2 increase triggers a compensatory elevation of pC02 produced by auto regulatory mechanisms within the brain. This increased pCO2 normally results in increased vasodilation of cerebral blood vessels resulting in an increased blood supply that can be delivered into areas of decreased cerebral activity. However, if there has been longstanding cerebral ischemia, as would be expected in a patient with a tight carotid artery stenosis that is the basis for a need for carotid endarterectomy, blood vessels in ischemic areas of the brain area already maximally dilated and can accept only a limited blood supply. Therefore, hypoxic cerebral neurons which already have a need for additional blood flow may prove instrumental in subsequent seizure activity following a carotid endarterectomy.

Bernoulli's law of physics written in 1,738 states, "fluids readily flow to and through areas of least resistance". Based on this physical principle, cerebral hyperperfusion that occurs immediately after a carotid endarterectomy results in a post-operative rush of blood into areas of the brain with the least vascular resistance, i.e., normal brain tissue. By Bernoulli's law, a smaller volume of blood flows to brain areas with greater vascular resistance within ischemic brain tissue.

Cerebral hyperperfusion occurs within 24 h to 48 h after carotid endarterectomy, but seizures do not occur during this period. If hyperperfusion is the basis for seizure activity, it seems reasonable to believe that seizures would occur shortly after surgery, but they do not. Some patients after carotid endarterectomy develop vasogenic edema seen on CAT scan that is diffuse and patchy. This edema has a slowing effect on the neuro-electrical activity of hypoxic neurons reaching its maximum intensity by 48 h following carotid endarterectomy. However, the edema is absorbed by the end of the first post-operative week [5].

According to Bernoulli's Law following a carotid endarterectomy, CBF enters the entire brain with the major increase occurring predominantly into well-perfused locations with less blood flowing into hypoxic neuronal areas. This results in the inability of hypoxic neurons to accept the increased blood flow needed by these neurons that are known to be electrically unstable and have the potential to excite seizure activity. That hypoxic neurons are neuro-electrically irritable was well demonstrated by Electroencephalography (EEG) in patients who did develop post-operative seizures following a carotid endarterectomy. Mayo Clinic investigators reported that seizures following a carotid endarterectomy demonstrated Period Lateralizing Epileptiform Discharges (PLED) on the side of the brain ipsilateral to the carotid endarterectomy. These PLEDs were characterized as being "periodic discharges on one side of the brain which denote an acute localizing cerebral focus of irritability that are usually transient and resolve fully" [6].

## Discussion

Cerebral hyperperfusion begins immediately after a carotid endarterectomy, but within days there is a return to normal cerebral perfusion. Of interest is that so-called hyperperfusion seizures do not occur early after carotid surgery even though hyperperfusion is at its height during this time period. If increased blood supply is needed by non-ischemic neurons because of heightened neuronal activity after a carotid endarterectomy, CBF is apparently diverted after surgery from ischemic cerebral regions and transferred to non-hypoxic locations which are requiring an increase in blood supply in effect, an intra cerebral steal reaction. A steal phenomenon occurs when a vascular location steals blood from another region within the organ. This is apparently what happens following a carotid endarterectomy when non-ischemic neurons post-operatively require an additional blood supply for functional activity. This appears to be the stimulus for seizures following a carotid endarterectomy—a need for vascular steal action.

Based on Bernoulli's Law, a vascular steal activity would result in cerebral blood flow following a carotid endarterectomy to be disproportionate in the volume of blood which flows to normal areas of the brain compared to chronically ischemic locations. This in effect leads to an intra cerebral steal phenomenon in which hypoxic neurons, known to be neuro-electrically irritable, would allow blood flow to be transferred to neurons which are non-hypoxic resulting in subsequent seizure activity.

## Conclusion

A hypothesis is presented based on Bernoulli's Law that cerebral blood flow after a carotid endarterectomy is disproportionate in the volume of blood flow to normal areas of the brain compared to chronically ischemic locations. This allows for a continued disparity between the blood flow between non-ischemic and neuro-electrically unstable hypoxic neurons. With increasing post-operative neuronal activity following a carotid endarterectomy, additional amounts of blood required by non-ischemic neurons lead to vascular shunting from hypoxic neuronal areas resulting in an intra cerebral steal phenomenon. Hypoxic neurons which are known to be neuro-electrically irritable appear to be the source of seizure activity suggesting, therefore, that seizures following a carotid endarterectomy are not due to hyperperfusion but to vascular steal activity.

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