

# Cervical Carotid Aneurysm Presenting As Recurrent Cerebral Ischemia with Head Turning

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**SUMMARY** Extracranial carotid artery aneurysms are uncommon lesions with protean manifestations. This report describes a patient in whom the presenting symptom of a right carotid aneurysm was recurrent right hemisphere ischemic attacks when he turned his head to the left. The angiographic and operative findings explained the mechanism(s) of his symptoms. The importance of such symptoms is that they should suggest a mechanical etiology and that the probability of a surgically correctable lesion exists. Arteriography is the only reliable means of making a definitive diagnosis and should be considered early in the evaluation.

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ANEURYSMS of the carotid artery in the neck are an uncommon disorder, but not rare.<sup>1-3</sup> Although the most frequent presenting complaint in patients with these lesions is an uncomfortable mass in the neck,<sup>4</sup> the clinical picture may be quite variegated. Headache, neck and facial pains,<sup>4-8</sup> subjectively audible bruits,<sup>1, 9</sup> hoarseness,<sup>10</sup> upper airway obstruction,<sup>4, 11</sup> dysphagia,<sup>11-13</sup> hemoptysis, and epistaxis,<sup>4, 12, 13</sup> have all been reported as initial symptoms of these lesions. Neurological symptoms are also not uncommon as initial presentations. Frank stroke, amaurosis fugax, transient cerebral ischemia, dizziness, syncope, and coma have been described.<sup>1, 3, 4, 8, 10, 12, 14, 15</sup> We have recently encountered a patient in whom recurrent transient ischemic attacks of the right hemisphere, precipitated by turning the head to the left, was the presenting symptom of a dissecting aneurysm of the cervical internal carotid artery. To our knowledge, this is the first report in the English literature of such a presentation. Our case is described. The mechanism and the importance of this uncommon presentation for this uncommon disorder is discussed.

## Case Report (W.T.)

A 69-year-old, hypertensive, white male was admitted to the hospital in March, 1977, for complaints of recurrent episodes of dizziness and fainting spells, associated with numbness and weakness of his left face and arm for one year previously. These attacks were typically precipitated by turning his head to the left and would last for only seconds to minutes. The patient's complaints had initially been felt to represent orthostatic hypotensive episodes. However, there had been no improvement in his symptoms after his anti-hypertensive medications had been stopped. He had been given a diagnosis of "Atypical TIAs" until he presented with the sudden onset of a left hemiparesis one week prior to his admission to our hospital. The left hemiparesis had lasted almost 24 hours and had also been precipitated by turning his head to the left.

On admission to the hospital the patient was found to be a generally healthy male with blood pressures of

180/100 in each arm. There was a mild hyperreflexic left hemiparesis and a right carotid thrill and bruit. No palpable masses in the neck were noted. The patient would allow his head to be turned only slightly to the left by the examiner and cautiously maintained a "face-forward" position. The remainder of the examination was unremarkable. Three days after admission, the patient suddenly became densely paretic in the left face and arm when he inadvertently turned his head to the left while taking a shower. He markedly improved over the next 72 hours and arteriography was performed.

Complete cerebral angiography, using a femoral catheter technique, revealed a lobulated and dissecting aneurysm at the origin of the right internal carotid artery (fig. 1). With the patient's head turned cautiously to the left, just short of the point past which he knew that his symptoms would be precipitated, a repeat right common carotid injection was performed. In this oblique view the lumen of the proximal right internal carotid artery was seen to be almost 99% obstructed by the dissecting aneurysm (fig. 2). Distal to the obstruction in the neck the carotid was well opacified and normal in appearance throughout. The left carotid and both vertebral arteries were normal at their origins and throughout their respective courses. There was no demonstrable contribution from these vessels to the right internal carotid distribution.

The following day the right carotid bifurcation was explored. A thin-walled, bluish-tinged, 3.0 × 4.0 cm aneurysmal dilatation of the distal common carotid and proximal internal carotid arteries was found (fig. 3). A large fungating, ulcerative, and partially calcified atheroma was found to bulge into the lumen of the internal and common carotid arteries circumferentially. A subintimal dissection had begun along the posterior wall of the internal carotid ostium with resulting aneurysmal dilatation of its walls and rostral progression of the dissection into the proximal 2-3 cm of the vessel. The lumen of the internal carotid was severely compromised. A common carotid to internal carotid shunt was utilized intraoperatively while an endarterectomy was performed. The aneurysmal sac was excised and an angioplastic repair of the vessel was performed. No grafts were necessary.

The patient's postoperative course was uneventful and he went home on the tenth postoperative day. Upon discharge he was neurologically intact save for a

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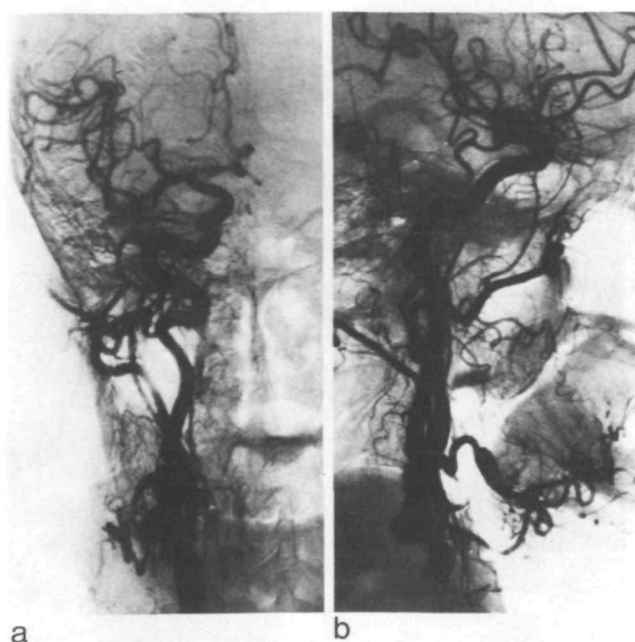


FIGURE 1. Preoperative right AP and lateral carotid angiogram. Lobulated dissecting aneurysm of the stenotic internal carotid artery is seen.

mild left central facial paresis and a left side reflex preponderance. Postoperative angiography before discharge demonstrated good patency and normal flow through the right carotid (fig. 4). The patient has remained free of symptoms to date and enjoys an unrestricted range of motion of his neck.

### Discussion

The effects of head turning upon the vertebral-basilar circulation are well documented.<sup>16</sup> The effects of head turning on internal carotid artery flow, however, are less well appreciated. It has been clearly demonstrated in cadavers,<sup>17</sup> as well as in patients,<sup>18</sup> that turning the head to one side may obstruct flow in the contralateral internal carotid artery. The mechanism by which carotid artery flow is altered by head rotation in these situations is thought to result from extrinsic compression of the vessel by the lateral mass of the atlas.<sup>19</sup> This may sometimes result in intimal fractures and subsequent thrombosis of the vessel,<sup>20</sup> and possibly aneurysm formation as well.

In our patient the clinical course of transient cerebral ischemia precipitated by head turning is explained by the angiographic and operative findings. The firm mass of the aneurysm, which in our case involved the origin of the internal carotid artery well below the atlas, compressed the already severely compromised lumen of the vessel when the ipsilateral sternocleidomastoid muscle contracted. In view of the fungating and ulcerated luminal surface of the atheromatous plaque, it is also possible that embolic debris was liberated when the aneurysm fundus was compressed. Compression by and/or embolism from the aneurysm itself best explains the symptoms in this

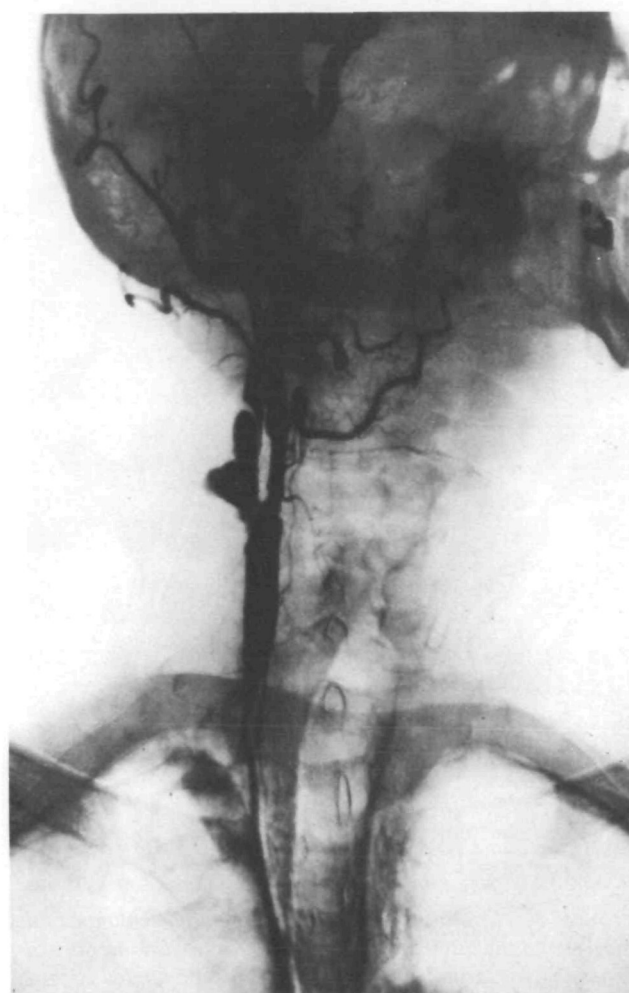


FIGURE 2. Preoperative right common carotid angiogram (oblique view). Patient's head is turned slightly to the left and 2 aneurysmal dilatations of the internal carotid origin are seen. Lower lobulated portion is directed laterally and posteriorly. More elongated portion is directed upwards as well as laterally and is separated from the lumen of the internal carotid by an intimal flap. The internal carotid lumen is almost completely obliterated by the dissecting aneurysm when head is turned.

case rather than atlantal compression. The complete relief of symptoms after surgery lends further support to this contention.

Aneurysms of the extracranial carotid arteries may be located on the common carotid artery, its bifurcation, or on the internal carotid artery from its origin up to the base of the skull.<sup>2, 3, 21</sup> At each site various shapes and sizes may occur which probably accounts for the various signs and symptoms of this disorder. Atherosclerosis and trauma are now considered to be the most common causes of these lesions,<sup>1, 2, 4, 8, 14, 15, 21</sup> and in most cases they are amenable to surgical correction.<sup>1, 4, 8, 15, 22, 23</sup> Although rupture of these aneurysms is apparently uncommon,<sup>3, 4, 12, 15</sup> neurological catastrophes are frequent sequelae. Moreover, in the case of the dissecting

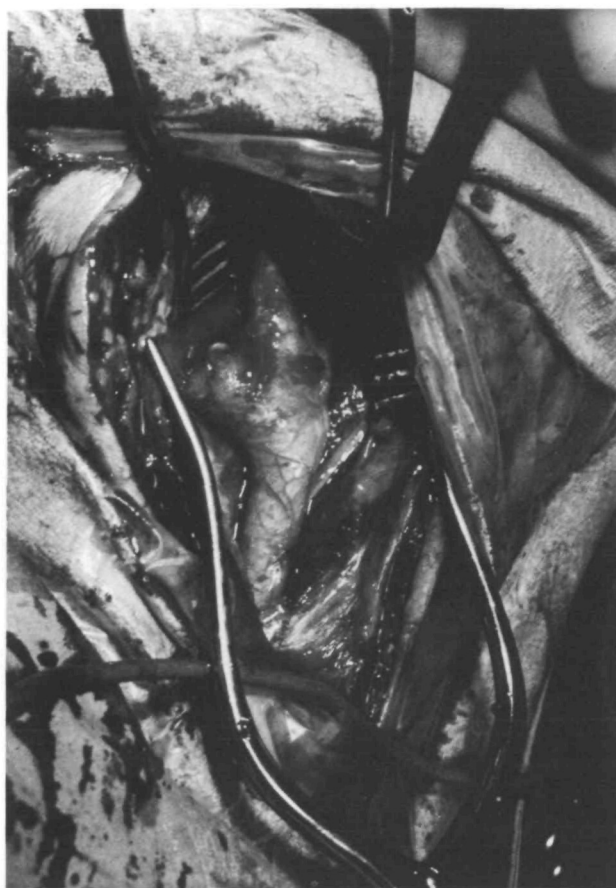


FIGURE 3. Operative exposure. The sternocleidomastoid muscle and the internal jugular vein are retracted laterally to expose right carotid bifurcation. Aneurysm fundus is projecting laterally and posteriorly from origin of the internal carotid artery to lie between the sternocleidomastoid muscle and the transverse process of C5.

carotid aneurysm, the progression of signs and symptoms may increase at an alarmingly rapid pace.<sup>8, 22</sup>

Our experiences with this patient, combined with our review of the literature regarding these lesions, lead us to several conclusions. In patients with internal carotid ischemic attacks which are precipitated by head turning, a mechanical etiology should be highly suspect. The cause of the symptoms in these patients may be the result of extraluminal compression of the carotid artery by the lateral mass of C1, or possibly by an aneurysm of the vessel in the neck. Carotid sinus massage,<sup>24, 25</sup> deep palpation of the neck, and vigorous head turning in the evaluation of these patients is certainly contraindicated. These lesions causing mechanical compression of the carotid artery are most often amenable to surgical correction. However, if unrecognized and untreated the potential for neurological catastrophe is great. Arteriography is the only reliable means of making a definitive diagnosis and should be considered early in the evaluation of patients with these symptoms.

#### Acknowledgments

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FIGURE 4. Postoperative right brachial angiogram. The common carotid and its bifurcation are widely patent and normal in contour.

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## Neurologic and Cardiovascular Effects of Hypotension in the Monkey

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**SUMMARY** Thirty monkeys were exposed to controlled systemic hypotension of different magnitudes and durations to determine factors leading to brain injury or cardiovascular failure. Fourteen monkeys developed brain injury. Of these, 6 survived indefinitely and 8 were sacrificed or died within 12-62 hours due to neurologic deterioration accompanied by respiratory failure. Sixteen animals did not develop brain injury, but 9 of these died within 24 hours from documented cardiovascular failure while the remaining 7 survived indefinitely. A highly reproducible threshold for the development of brain injury was found at a mean arterial blood pressure (MABP) of 25 mm Hg. Maintenance MABP was  $\leq 25$  mm Hg in 13 of 14 lesioned monkeys and  $> 25$  mm Hg in 15 of 16 non-lesioned monkeys. Maintenance MABP averaged  $20.1 \pm 1.1$  mm Hg in lesioned and  $32.1 \pm 1.7$  mm Hg in non-lesioned animals ( $p < 0.001$ ). Among the non-lesioned animals, death from delayed cardiovascular failure ensued when MABP was maintained between 27 and 35 mm Hg for 90 min or longer. Animals exposed to this range of hypotension for  $< 90$  min or to MABP exceeding 35 mm Hg for as long as 3 h survived intact. EEG changes occurring during hypotension most accurately predicted neurologic outcome. The threshold MABP required to produce cerebral electric silence was 21-22 mm Hg. Monkeys developing marked brain injury had  $> 25$  minutes of EEG flattening, while slightly injured animals had it for 5-15 minutes and those without injury for  $< 5$  min. Changes in acid-base state, common carotid artery blood flow, and cerebral uptake of glucose and oxygen during hypotension also correlated with neurologic and cardiovascular outcome. Hypoxemia and hypercarbia were not contributory factors in the production of brain injury in this study.

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**THE RELATIVE** contributions made by hypoxemia, systemic acidosis, hypotension associated with reduced cerebral blood flow, and altered brain intermediary metabolism to the development of brain injury as a consequence of hypoxic exposure remain uncertain. This lack of precise knowledge of the pathogenesis of hypoxic brain injury is particularly

unfortunate since exposure to hypoxia constitutes one of the common causes of brain injury and death in man.

We have considered 3 questions of fundamental importance to the clinician and experimentalist alike. First, can hypotension and reduced cerebral blood flow be studied independently and assigned a role in the development of brain injury separate from the hypoxemia and systemic acidosis that commonly accompany hypotension? Second, can the threshold value of systemic hypotension that leads to brain injury be delineated with precision? Finally, why does exposure to hypotension cause brain injury in some instances and death from cardiogenic shock in others?

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