

Rotational Vertebral Artery Occlusion: A Mechanism of Vertebrobasilar Insufficiency

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OBJECTIVE: Symptomatic dynamic changes in blood flow secondary to vertebral artery compression with rotational head motion are evaluated in a series of patients as a cause for posterior circulation transient ischemic attacks. These cases are classic examples of rotational vertebral artery occlusion and allow for the discussion of the anatomic basis, angiographic features, and treatment options.

ILLUSTRATIVE CASES: In our series, symptoms of vertebrobasilar insufficiency were reproducible with rotational head movement. Compression of the vertebral artery was demonstrated angiographically. The correct site of occlusion of the vertebral artery was apparent only by dynamic angiography with progressive head rotation. All of the patients presented in the illustrative cases had occlusion at the C2 level; however, one patient had been previously misdiagnosed and another had an additional site of occlusion. The anatomic course of the vertebral artery is described in addition to the sites of rotational occlusion.

CONCLUSION: Rotational vertebral occlusion is an important cause of vertebrobasilar symptoms, which may lead to permanent neurological deficit if left undiagnosed. Dynamic angiography is the established method of diagnosis. Great care must be taken to avoid misdiagnosing the site of occlusion or missing a second occlusive site. For this reason, it is crucial to have a thorough understanding of the anatomic course of the vertebral artery and the muscular and tendinous insertions, which may cause rotational occlusion. The decision for treatment must be based on the site of occlusion as well as the assessment of the patient as a surgical candidate. A review of the literature reveals that surgical treatment is effective and must be considered to avoid further morbidity. (*Neurosurgery* 41:427-433, 1997)

Key words: Cerebral angiography, Vertebral artery, Vertebrobasilar insufficiency, Vertigo

Vertebrobasilar insufficiency is a syndrome characterized by diplopia, dysarthria, visual disturbances, vertigo, and sensory or motor symptoms. Symptoms relate to ischemia from atherosclerotic lesions, thromboembolic events, and mechanical obstruction. In three patients, symptomatic dynamic changes in blood flow secondary to vertebral artery compression with rotational head motion are evaluated as a cause for posterior circulation transient ischemic attacks. The purpose of these case reports is not to define a new neurological disorder but rather to serve as examples for discussion of the appropriate diagnostic evaluation and treatment options to avoid some of the common pitfalls encountered in patients with vertebrobasilar insufficiency.

ILLUSTRATIVE CASES

Patient 1

This patient, a 49-year-old man, presented with a 1-year history of dizziness when turning his head to the far right. The results of a computed tomographic scan of the brain were normal. An arch aortogram was obtained and demonstrated a normal appearing left vertebral artery up to the C2 vertebral body. The right vertebral artery was hypoplastic. During a selective left vertebral artery injection with the head turned to the right, contrast flowed to the level of the C4-C5 interspace, which was thought to be the cause of the symptoms (*Fig. 1A*). The patient underwent a posterior decompression of the foramina transversaria and surrounding soft tissues at the level of C4-C5. Postoperatively, there was no improvement

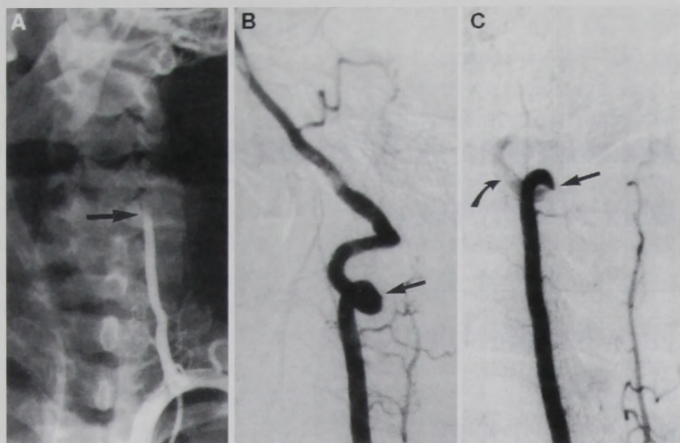


FIGURE 1. *A*, angiogram of the left subclavian artery, obtained before surgery (anteroposterior projection, head rotated to the right), demonstrating flow up the left vertebral artery to the C5 level (arrow). The contrast material reaches only the level of C5, the site at which the laminectomy was subsequently performed. This does not represent the site of arterial occlusion, which is higher at the C2 level. *B*, angiogram of the left vertebral artery, obtained after laminectomy (lateral projection, head in a neutral position), showing the left vertebral artery coursing through the foramen transversarium of the C2 vertebral body (arrow). As the artery crosses through the foramen transversarium, there is a curve in the vessel but no stenosis. *C*, angiogram of the left vertebral artery (lateral projection, head turned to the right). With head turning, there was progressive stenosis of the left vertebral artery at the foramen transversarium of the C2 vertebral body (arrow). A small amount of contrast courses beyond the stenosis into the more distal vertebral artery (curved arrow). This stenosis is caused by an ossified or fibrous band across the foramen transversarium at the C2 level.

in the patient's symptoms. He presented to our institution for further evaluation.

The findings of a physical examination included immediate onset of dizziness, leading to loss of consciousness, when the patient turned his head 90 degrees to the right. Extreme neck extension also reproduced the symptoms. There were no cervical bruits, and the remaining neurological examination revealed no focal deficits.

Digital subtraction angiograms were obtained of the anteroposterior, Towne's, and Caldwell projections, with the patient's head turned to the right sufficiently to produce dizziness but not loss of consciousness. The left vertebral injection demonstrated a stricture of the left vertebral artery as it entered the C2 foramina. With further head turning, the stenosis became an occlusion (Fig. 1, *B* and *C*). The patient refused further surgical treatment, preferring to restrict his head movement to avoid vertebrobasilar insufficiency.

Patient 2

This patient, a 62-year-old man, presented with a 2-month history of symptoms. He reported that while working on his car, he felt as though he were going to pass out when he extended his head backward. After that, he experienced daily events of near fainting when he turned his head backward and to the right. The events rapidly resolved whenever he turned his head to a neutral position. His medical history was notable for hypothyroidism and cervical spondylotic

degenerative disease. The results of a physical examination were noncontributory.

Transcranial doppler ultrasonography indicated normal flow and velocity in both vertebral arteries and the basilar artery with the head in neutral, far right, and extended positions. Vertebral angiography demonstrated a hypoplastic right vertebral artery and a normal left vertebral artery with the head in a neutral position. When the patient's head was turned to the right, there was significant stenosis of the left vertebral artery at the foramina transversaria at the C2 vertebral body (Fig. 2, *A* and *B*). With head rotation, there were no changes in the right vertebral artery. This patient also elected to forego surgical treatment and has remained asymptomatic with voluntary restriction of head movement.

Patient 3

This patient, a 39-year-old man, developed episodic tinnitus, haziness, and blackouts, which were associated with turning his head to the right. There were no focal neurological deficits.

The results of angiography of the cervical portions of both vertebral arteries were normal when the patient's head was held in a neutral position. Dynamic angiography with head rotation to the right demonstrated that the left vertebral artery was occluded at the C2 level (Fig. 3*A*). His right vertebral artery, which was normal in the neutral position (Fig. 3*B*), occluded at the C7 level (Fig. 3*C*) with head rotation to the right. A lateral carotid arteriogram with the patient's head turned sharply to the right showed normal filling of the anterior circulation but no filling of the posterior circulation, indicating that

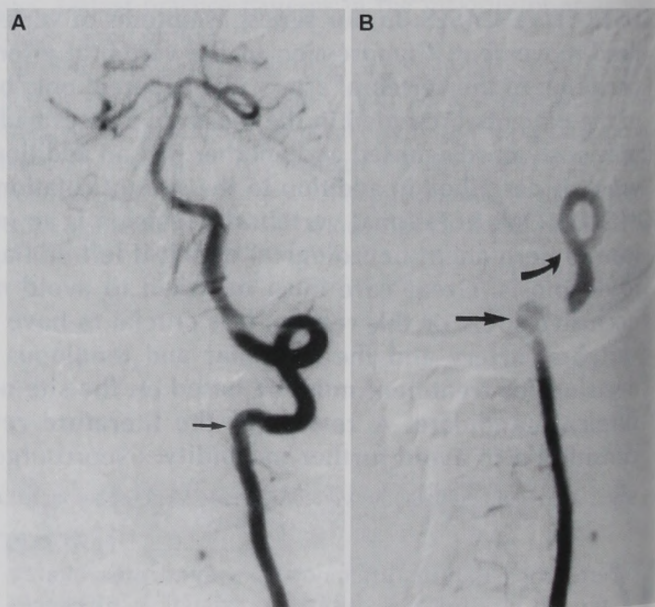


FIGURE 2. *A*, angiogram of the left vertebral artery (anteroposterior projection), demonstrating normal left vertebral and basilar arteries. The vertebral artery, at the point at which it enters the foramen transversarium of the C2 vertebral body (arrow), shows the normal anatomic caliber change without significant stenosis. *B*, angiogram of the left vertebral artery (anteroposterior projection, head turned to the right), showing compression of the left vertebral artery (arrow), producing markedly decreased flow through the more distal vertebral artery (curved arrow). In the more proximal vertebral artery, there is stasis of contrast, accounting for the increased density.

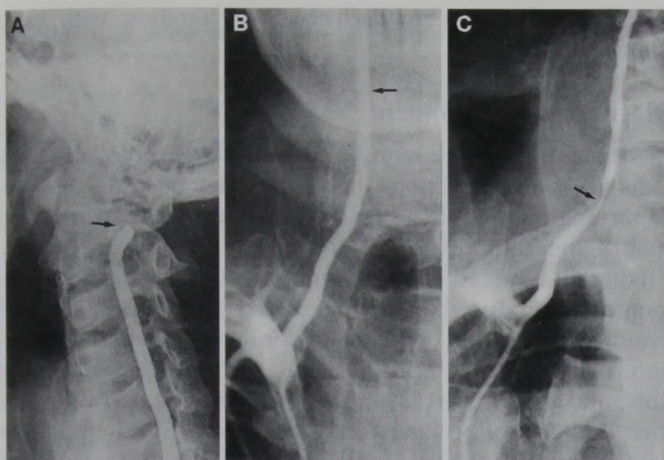


FIGURE 3. *A*, angiogram of the left vertebral artery (lateral projection, head rotated to the right), showing occlusion of the artery at the foramen transversarium of the C2 vertebral body (arrow). *B*, angiogram showing right subclavian artery injection (anteroposterior projection, head in a neutral position) and demonstrating flow in the right vertebral artery to the C5 vertebral body (arrow). *C*, with head rotation to the right, a high-grade stenosis of the right vertebral artery developed at the C7 level (arrow).

there was no flow through the posterior communicating arteries. The patient reported having symptoms during that part of the examination. He elected to treat this conservatively and has remained asymptomatic.

DISCUSSION

What was referred to as cervical vertigo (17) in 1955 was a syndrome characterized by vertigo, dizziness, and blurred vision with head rotation. Tatlow and Bammer (24) postulated, in 1957, that head rotation compressed the vertebral arteries, which led to these symptoms of vertebrobasilar insufficiency. In studies performed on a single cadaver, compressions of the vertebral arteries were identified at the C1–C2 and the C5–C6 levels when the head was rotated. These observations were based on work performed 20 years earlier by DeKleyn and Versteegh (4), who described a decrease in vertebral circulation on one side when the head was turned backward and to the opposite side. Toole and Tucker (25) later observed, in a study of 20 cadavers, that 75% of the cases demonstrated significantly compromised blood flow in one or both vertebral arteries with head rotation.

Cervical spondylosis provided the initial model for vertebral artery compression. Sheehan et al. (19), in 1960, published reports of a series in which 5 of 26 patients with cervical spondylosis and symptoms of vertigo and blurred vision with head rotation demonstrated vertebral artery compression as revealed by angiography. Powers et al. (16) then described a similar syndrome related to intermittent compression of the vertebral artery by the anterior scalene muscle. Since then, there have been more than 100 cases of symptoms related to compression of the vertebral artery, which has recently been named *bow hunter's syndrome* (22).

At this time, the number of causes of this syndrome has grown to include many activities. Vertebral artery compression has resulted from chiropractic manipulation, surgical positioning, rheumatoid subluxation, a multitude of physical activities, and sports. There have also been numerous accounts of embolic complications related to vertebral compression. However, the true incidence of this disorder is not known. In an early study of 43 asymptomatic male volunteers with a mean age of 47 years, Farris et al. (5) observed angiographic evidence of vertebral artery occlusion with head rotation in 4 of the volunteers (9%).

Anatomic relationships

We do know that the vertebral arteries are susceptible to mechanical compression with head rotation because of their anatomic relationship with neighboring bone, muscle, ligaments, and fascia. As each vertebral artery branches from the first part of its respective subclavian artery, it ascends and enters the foramina transversaria of C6. At this entry point, the artery must pass near tendinous insertions of the longus colli, scalenus anterior, and scalenus medius muscles (Fig. 4). Fibers arising from the lateral rim of the vertebral canal cross medially to join the longus colli. In addition, fibers from the medial rim cross laterally to join the scalenus anterior and medius. Powers et al. (16), in 1960, were the first to publish reports of a series of patients whom they treated by performing scalenotomies. Reports of more than 50 cases have been published relating to external compression of the vertebral artery by ligamentous insertions or deep fascia at the "scalenovertebral angle" (3, 8, 10–12).

The artery next passes through the foramina transversaria of the cervical vertebrae at C6–C2. At that point, the artery is susceptible to compression by osteophytes and other degenerative changes of cervical spondylosis (Fig. 5). There have been more than 30 published reports of cases of vertebral compression by osteophytes at all levels (1, 13, 19, 21). When the head is rotated toward the side with the osteophyte, the ipsilateral vertebral artery is compressed against the osteo-

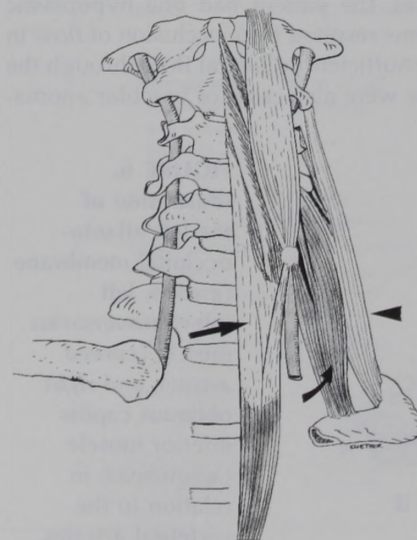


FIGURE 4. Illustration of the left vertebral artery as it passes near the tendinous insertions of longus colli (arrow), scalenus anterior (curved arrow), and scalenus medius (arrowhead).

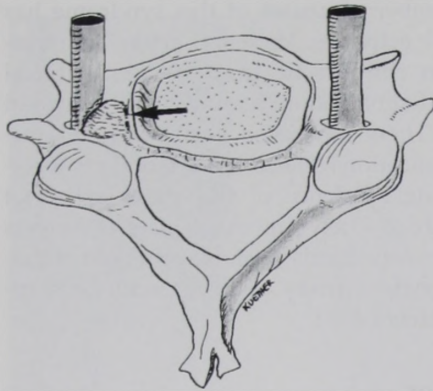


FIGURE 5. Illustration of the left cervical osteophyte (arrow) showing compression of the vertebral artery at the foramina.

phyte and the contralateral artery is compressed at the foramina.

The vertebral artery then passes obliquely to the C1 foramina. At the atlantoaxial joint, the artery can be compressed by fibers from the obliquus capitis inferior muscle, intertransversarius muscle, membrane hypertrophy, or vertebral subluxation (7) (Fig. 6). Selecki (18) demonstrated, using cadavers in 1969, that kinking and stretching of the contralateral vertebral artery can be observed at this site with 30 degrees of head rotation and becomes well marked at 45 degrees. Beyond 45 degrees, kinking and stretching at the foramina transversaria was seen in both arteries.

After passing through the C1 foramina, the artery bends posteriorly at right angles and winds around the superior part of the lateral mass of the atlas. The vertebral artery pierces the posterior atlanto-occipital membrane. At that point, the artery may finally be compressed (15, 26). The arteries then pierce the dura mater and arachnoid to enter the subarachnoid space of the cerebellomedullary cistern and unite to form the basilar artery.

One last hypothesis concerning the cause of these vertebrobasilar symptoms involves the formation of thrombus within the vertebral arteries during the time when the flow is occluded. These thrombi then embolize to result in either transient ischemic attacks or cerebral vascular accidents (6, 15, 20, 23).

In many of these cases, the patient had one hypoplastic vertebral artery. Symptoms resulted from occlusion of flow in the normal artery with insufficient collateral flow through the opposite vertebral. There were also cases of vascular anoma-

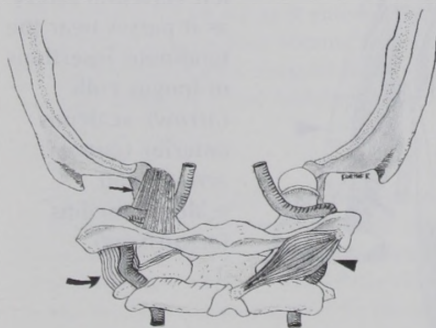


FIGURE 6. Illustration of the left atlanto-occipital membrane (arrow), left intertransversarius muscle (curved arrow), and right obliquus capitis inferior muscle (arrowhead) in relation to the vertebral arteries.

lies that led to cerebral ischemia or infarction with head rotation (9).

Diagnosis and Treatment

Although there are many causes of vertebrobasilar symptoms with head rotation, these cases demonstrate that dynamic angiography is necessary for diagnosis. One must visualize the compression of the artery with head rotation. Credibility is added to the examination if the patient's symptoms are reproduced. In any case, great care must be taken to avoid diagnosing an occlusion lower than the actual constriction. If the patient's head is turned, producing occlusion of flow, and the contrast has not yet reached the occlusive site, then the level of occlusion may be incorrectly identified, as demonstrated in the first case. For this reason, it is imperative to see the contrast extend to the level of the foramen magnum before making a final decision regarding the location of the stenosis.

Once an accurate diagnosis is made of rotational vertebral artery occlusion, then the patient has the option of conservative or surgical treatment. The conservative approach consists of anticoagulation or neck immobilization, either by instructing the patient to refrain from head turning or by the use of a collar. Surgical treatment must be tailored to the likely cause of the compression. Simple exploration with fascial decompression may be all that is needed. For obstructions relating to foraminal compression, either a midline approach with discectomy and osteophyte removal or a posterior lateral approach with partial transversectomy and unroofing of the foramina may be performed. Fusion has been advocated by some individuals but has not been shown to comparatively improve the postoperative course (1, 21). There are certainly those cases, such as vertebral compression related to subluxation, in which fusion is indicated.

Outcome

Regardless of the surgical procedure, a postoperative dynamic angiogram is required to substantiate that the rotational compression has been corrected. Listed in Table 1 is a review of the published reports of cases of rotational vertebral artery obstruction. The average age of the patients was 49 years, and the most common presenting symptoms were vertigo and vision changes. The method of diagnosis was angiography in every case. However, some of the earlier published reports did not include the use of dynamic angiography. The surgical treatment depended on the cause of the extrinsic compression. The overall success rate for surgery based on relief of symptoms was 85%.

Of those treated conservatively, nearly 50% went on to infarct or had residual neurological deficits. For this reason, it is crucial to discuss potential risks with patients when seeking informed consent. Most patients prefer not to have surgery, but they must be aware of the potential complications of more conservative treatment. The three patients presented in this article elected to treat their rotational compression conservatively despite the risks and our recommendations for surgical intervention.

TABLE 1. Review of Cases in Literature^a

Series (Ref. No.)	No. of Patients	Average Age (yr)	Presenting Symptom	Method of Diagnosis	Imaging	Dynamic Angiography	Treatment	Cause	Outcome	Postoperative Angiography
Bakay and Leslie, 1965 (1)	2	44	Dizziness	Angiography	Myelogram	+	Decompression, ant fusion	C5-C6 osteophyte	Asymptomatic at 6 mo	Normal
Barton and Margolis, 1975 (2)	2	50	Dizziness, blackouts, vertigo	Angiography		+	None	C1-C2 vertebral occlusion	Medullary infarct in one patient	
Dadsetan and Skerhut, 1990 (3)	1	56	Vertigo, decreased vision	Angiography	CT	+	Ant scalenotomy, section of longus colli tendon	Vertebral occlusion near origin	Asymptomatic postoperatively	Normal
Grossman and Davis, 1982 (6)	1	19	Vertigo, diplopia, ataxia	Angiography	CT	+	Anticoagulation	C1-C2 vertebral occlusion	Cerebellar infarct	
Hanakita et al., 1988 (7)	3	58	Weakness, vertigo, nausea	Angiography	CT	+	Decompressive transverse foramenectomy	C1-C2 vertebral occlusion	Asymptomatic, 2-yr f/u in one case	Normal
Hardin and Poser, 1963 (8)	15	58	Dizziness, syncope, dysarthria	Angiography		+	Ant scalenotomy and fascial decompression, section of thyrocervical trunk, suture fixation of subclavian artery	Vertebral occlusion origin to C5	Asymptomatic, 13 of 15	
Hope et al., 1983 (9)	1	16	Headache, vision, ataxia	Angiography	CT	-	None	Hypoplastic left vertebral, absent left posterior communicating artery	Cerebral infarct	
Husni et al., 1966 (10)	20	40	Dizziness	Angiography		+	Ant scalenotomy, section of longus colli tendon, interposing fat pedicle	C5-C6 vertebral occlusion	Asymptomatic, 19 of 20; avg f/u, 2 yr	
Kojima et al., 1985 (11)	1	64	Vertigo with head turning	Angiography	CT	+	Decompression, C6 transverse process unroofed	Longus colli, ant scalene	Asymptomatic; f/u, 18 mo	Normal
Mapstone and Spetzler, 1982 (12)	1	54	Vertigo with head turning	Angiography	CT	+	Decompression	Longus colli	Postoperative resolution	Normal
Nagashima, 1970 (13)	20	52	Vertigo, dizziness, blurred vision	Angiography			Transverse foramenectomy	C4-C7 compression	72% asymptomatic	13 of 20 Normal
Nagler, 1973 (14)	3	34	Paralysis, ataxia	Angiography	Myelogram		Cervical laminectomy in one case	C1-C2 vertebral occlusion in one case	Residual weakness and ataxia	
Okawara and Nibbelink, 1974 (15)	1	43	Headache ataxia	Angiography	CT	-	Steroids	C1-C2 vertebral occlusion	Residual ataxia	
Powers et al., 1961 (16)	28		Vertigo, tinnitus, deafness, bruit	Angiography			Decompressive scalenotomy and section of thyrocervical trunk	17 of 28 ant scalene	Asymptomatic, 16 of 17	
Sheehan et al., 1960 (19)	26	53	Vertigo, dizziness, blurred vision	Angiography			Bed rest, limit head motion	Cervical spondylosis		
Shimizu et al., 1988 (20)	1	37	Dizziness, diplopia, nausea	Angiography	CT	+	Decompressive transverse foramenectomy	C1-C2 vertebral occlusion with embolism	Asymptomatic; f/u, 24 mo	Normal
Smith et al., 1971 (21)	2	52	Headache, syncope, vertigo	Angiography		-	Decompressive foraminotomy, ant fusion	C5-C6 osteophyte	Asymptomatic	Normal
Sullivan et al., 1975 (23)	1	37	Headache vision loss	Angiography	CT	-	Decompression, ant fusion	C5-C6 osteophyte with embolism	Residual visual field defect	Normal
Yang et al., 1985 (26)	2	58	Vision loss, ataxia, and dizziness with head turning	Angiography	CT	+	C1-C2 fusion/observation	C1-C2 vertebral occlusion	Asymptomatic; f/u, 6 and 18 mo	Normal

^a CT, computed tomography; +, performed; -, not performed; ant, anterior; f/u, follow-up.

Postoperative angiography was performed in 20% of the cases. In the majority of patients, angiography was performed in the immediate postoperative period. Only four patients were reported with angiographic follow-up 3 to 8 months after surgery. Regardless of timing, all postoperative angiography revealed complete resolution of the rotational compression. Nagashima (13) reported that in 10 of 13 patients, although the compression was relieved, there remained some evidence of a retained kink in the artery.

CONCLUSION

Rotational vertebral occlusion is an important cause of vertebrobasilar symptoms. As many prior case reports have demonstrated, this may lead to permanent neurological deficit if left undiagnosed. A thorough understanding of the anatomic course of the vertebral artery and the muscular and tendinous insertions that may result in rotational occlusion is imperative in the treatment of this disorder. These three cases are classic examples of vertebral compression with head rotation. Misdiagnosis must be avoided by the use of dynamic angiography if this entity is suspected. Dynamic angiography is not a new concept but can be misinterpreted if not performed correctly. The decision regarding the appropriate type of treatment must be based on the site of occlusion as well as the assessment of the patient as a surgical candidate. A review of the literature reveals that surgical treatment is effective and should be considered to avoid further morbidity.

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COMMENTS

The authors provide a very concise summary of a peculiar clinical syndrome: rotational vertebral artery occlusion with vertebrobasilar ischemic symptoms. The reader should pay particular attention to the illustrative case of Patient 1 and Figure 1 of the article. This case report clearly demonstrates a mechanism by which the cause of the patient's symptoms can be misdiagnosed and treatment directed at the wrong site. The use of dynamic angiography in these cases is critical, because transcranial doppler ultrasonography or angiography performed in the neutral position will routinely miss the diagnosis. The authors summarize the available data and provide insight into various surgical and medical treatment alternatives for patients with compression syndromes arising from the various anatomic sites.

H. Hunt Batjer
Chicago, Illinois

This article demonstrates the angiographic feature of rotational occlusion of the vertebral artery in the neck. Awareness

of this phenomena is important in clinical practice when evaluating transient ischemia of the posterior circulation. It would be helpful to know the potential effects of surgical treatment.

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This study presents three patients with posterior circulation transient ischemic attacks in whom dynamic angiography during rotational head movements confirmed vertebral artery compression. The authors emphasize that the use of dynamic angiography is critical to making the diagnosis and delineating the precise site of vertebral artery compression. The artery is susceptible to mechanical compression during head rotation, particularly because of compression from muscle, ligament, and fascial insertions along the ascent of the vertebral artery in the cervical spine.

Although the topic of mechanical vertebral artery compression and head rotation causing posterior circulation transient ischemic attacks has been reviewed previously (1), the strength of this article lies in the demonstration of the usefulness

of dynamic angiography for establishing the diagnosis and site of rotational vertebral artery occlusion. The authors provide a thorough literature review and conclude that the overall success rate for surgical treatment of this problem is high. We have also achieved gratifying clinical outcomes after surgical decompression of the cervical vertebral artery in similar patients with posterior circulation transient ischemic attacks and angiographic confirmation of vertebral artery compression with head turning. In all of our cases, the contralateral vertebral artery was absent, was hypoplastic, or ended in the posteroinferior cerebellar artery.

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ANNOUNCEMENT

Foundation for International Education in Neurosurgery Update and Request for Volunteers

The Foundation for International Education in Neurosurgery (FIENS) continues to develop activities that are primarily designed to foster education in developing areas of the world. These initiatives are coordinated with the activities of the World Federation of Neurosurgical Societies, which, through its Education Committee, has sponsored a number of regional educational programs for neurosurgeons in developing areas. The Foundation assists in providing faculty for these larger educational efforts. The major current activities of FIENS consist of programs in Africa. The Foundation has worked with neurosurgeons in Ghana in an attempt to help develop an indigenous neurosurgical training program there. This has involved sending senior level residents and faculty for varying periods of time, usually 3 to 6 months for residents and 2 to 4 weeks for faculty support. FIENS has supported a program in Zimbabwe to provide neurosurgical assistance to the program already in place in that country, and it has supported an African trainee, who currently is a resident at Yale, in the initial steps to develop a neuroscience initiative for Southern Africa. She has been a liaison with the Panafrican Neurosurgical Association, which strongly supports this concept, and we anticipate rapid development. Another new project involves a Senior Neurosurgical Interchange with Peru under the guidance of Dr. Anselmo Pineda and the Peruvian-American Neurosurgical Society. Opportunities exist for volunteer neurosurgeons to go to Peru and visit and work in a number of different medical centers around the country. FIENS maintains a roster of neurosurgeons interested in serving as volunteers. For volunteer experiences of at least 4 to 6 weeks, the Foundation will support the volunteer's air travel expenses. The host country is ordinarily able to underwrite most of the volunteer's expenses incurred at work. Further information and volunteer request forms can be obtained from the Office of the Secretary of the Foundation, Dr. David Fairholm: Division of Neurosurgery, Department of Surgery, Room 3100, 910 W. 10th Avenue, Vancouver, BC, Canada V5Z 4E3.