ROTATIONAL VERTEBROBASILAR ISCHEMIA: HEMODYNAMIC ASSESSMENT AND SURGICAL TREATMENT

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OBJECTIVE: Rotational vertebrobasilar insufficiency is a severe and incapacitating condition. Proper investigation and management are essential to reestablish normal posterior circulation hemodynamics, improve symptoms, and prevent stroke. We present a series of 10 patients with rotational vertebrobasilar ischemia who were treated surgically and emphasize the importance of transcranial Doppler in the diagnosis and management of this condition.

METHODS: All patients presented with symptoms of vertebrobasilar insufficiency induced by head turning. Transcranial Doppler documented a significant decrease in the posterior cerebral artery velocities during head turning that correlated with the symptoms in all patients. A dynamic cerebral angiogram was performed to demonstrate the site and extent of vertebral artery compression.

RESULTS: The surgical technique performed was tailored to each individual patient on the basis of the anatomic location, pathogenesis, and mechanism of the vertebral artery compression. Five patients underwent removal of osteophytes at the level of the subaxial cervical spine, one patient had a discectomy, two patients had a decompression only at the level of C1–C2, and two patients had a decompression and fusion at the C1–C2 level.

CONCLUSION: The transcranial Doppler is extremely useful to document the altered hemodynamics preoperatively and verify the return of normal posterior circulation velocities after the surgical decompression in patients with rotational vertebrobasilar ischemia. Surgical treatment is very effective, and excellent long-term results can be expected in the vast majority of patients after decompression of the vertebral artery.

KEY WORDS: Bow hunter's stroke, Cervical osteophytes, Transcranial Doppler, Vertebral artery, Vertebrobasilar insufficiency

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It is known that manipulation and rotation of the neck can cause injury to the vertebral arteries and subsequent stroke in the vertebrobasilar system (18, 37). Ford (4) described the case of a patient with symptoms of syncope, vertigo, nystagmus, disturbances of vision, and dizziness that were attributed to intermittent compression of both vertebral arteries at the C1–C2 region because of excessive mobility associated with an os odontoideum. Tatlow and Bammer (34) later described the "syndrome of vertebral artery compression" and reported additional patients with symptoms of vertebrobasilar ischemia during head rotation. Extrinsic compression of the vertebral artery by osteophytes during head rotation was then demonstrated initially in cadaveric specimens (11, 34) and subsequently in patients (10, 29).

The most common symptoms of rotational vertebrobasilar insufficiency are syncope or near syncope (the feeling of almost "passing out"), dizziness, vertigo, visual blurriness, drop attacks, tinnitus, hypoacusis, and sensory or motor deficits induced by turning the head to a particular side (22, 34). The symptoms are characteristically alleviated once a neutral head position is resumed (22, 33, 34). Permanent neurological deficit after head turning has also been described in a syndrome known as "bow hunter's stroke" after Sorensen (31) described a patient who had a posterior fossa stroke after target practicing. Several different mechanical causes have been

reported to be responsible for rotational vertebrobasilar ischemia (3, 5–7, 10, 13, 16, 17, 19, 21, 22, 24, 25, 27, 29–31, 33, 36). Although the symptomatology represents a temporary ischemic event in the vertebrobasilar system, only in a few instances have hemodynamic studies been performed when evaluating patients with this condition (2, 6, 23, 24, 36). Our objective was to present the clinical data and results on a series of 10 patients who were treated surgically at the University of Washington and affiliated hospitals during the past 10 years. In addition, the ability of transcranial Doppler (TCD) to identify the altered posterior circulation hemodynamics preoperatively and also document the reestablishment of normal hemodynamics after an effective surgical treatment is demonstrated.

PATIENTS AND METHODS

During the past 10 years, more than 100 patients with a presumptive diagnosis of rotational vertebrobasilar ischemia were studied with TCD at the University of Washington according to a protocol described previously (33). One of the main advantages of the TCD is that the examiner can correlate the velocity changes with the reproduction of symptoms in real time at the bedside. In patients with rotational vertebrobasilar insufficiency, there is typically a decrease in the mean posterior cerebral artery (PCA) velocities of an average of 60% of baseline during head rotation. On resumption of the neutral position, there is a reactive hyperemic response, which is seen as an overshoot of the mean PCA velocities of at least 10% above baseline values (*Fig. 1*). This reactive hyperemic phase is followed by normalization of the mean PCA velocities. Using TCD, the head-turning maneuver can be repeated several times, and the decrease in the velocities and hyperemic response will consistently have the same pattern and reproducibility. In patients with symptoms that are not so characteristic and not reproducible with head turning, there is neither a significant decrease in the PCA velocities nor a reactive hyperemic phase. These patients are considered not to have rotational vertebrobasilar ischemia, and an angiogram is not indicated. Patients with a positive TCD test are then scheduled for a dynamic angiogram, in which the arterial injections are performed with the head in the neutral and rotated positions. The angiogram will demonstrate the exact site and degree of vertebral artery compression. Fine-cut cervical spine computed tomographic and magnetic resonance imaging scans at the level of the compression can help determine the pathogenesis of the arterial impingement. *Figure 2* is a flow chart summarizing the approach to a patient with presumed rotational vertebrobasilar ischemia.

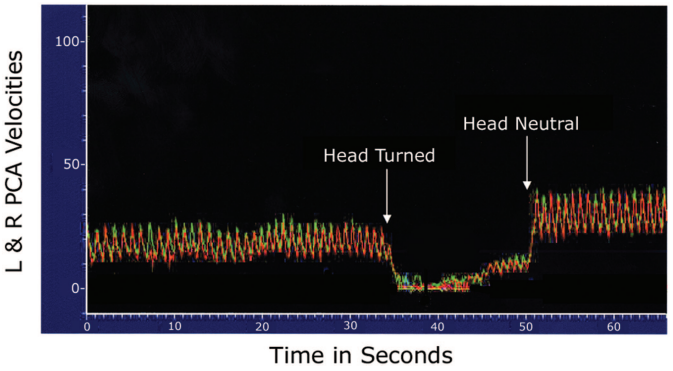

FIGURE 1. *Typical TCD waveform of a patient with rotational vertebrobasilar ischemia. There is an immediate decrease in the PCA velocities when the head is turned, which is followed by a hyperemic response on resuming a neutral head position.*

From the initial group of more than 100 patients studied, there were 16 patients with a positive TCD test. Of these 16 patients, 10 underwent surgical treatment, 2 are still undergoing preoperative workup, 3 were considered at high risk for surgery because of severe coronary artery disease, and 1 refused surgical treatment.

Table 1 contains a summary of the clinical history, hemodynamic and imaging findings, surgical procedure performed, and clinical results on all 10 patients who underwent surgical decompression of the vertebral artery. Three of the 10 patients are described in more detail as illustrative examples.

Illustrative Cases

Patient 2, a 52-year-old man, presented with a history of near syncope, blurred vision, dizziness, and leg weakness when turning his head to the right. His medical history was significant for hypertension, smoking, and high cholesterolemia. TCD during head turning to the right demonstrated a decrease in the PCA velocities to almost zero. A

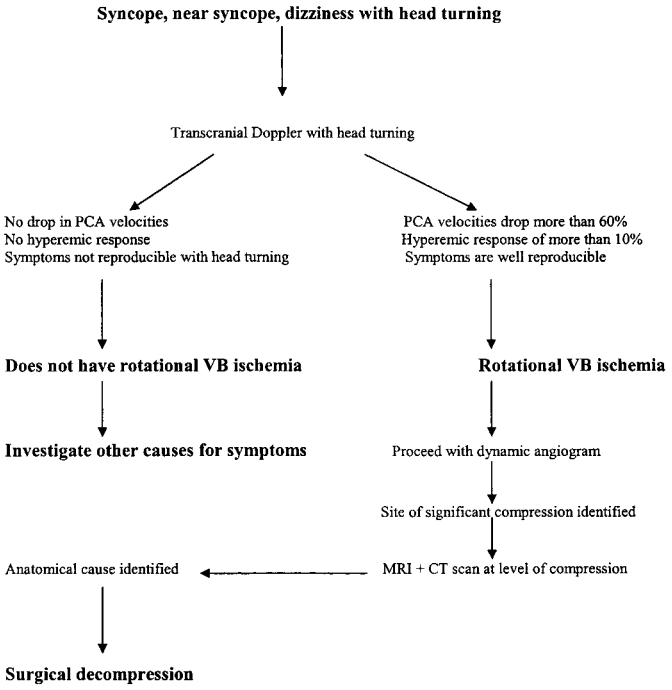

FIGURE 2. *Flow chart showing approach to a patient with presumed vertebrobasilar (VB) ischemia.*

TABLE 1. Summary of clinical data, imaging studies, surgical procedure performed, follow-up duration, and postoperative results[a]

Patient no.	Age (yr)	Clinical history	Preoperative TCD	Imaging studies	Surgical procedure	Postoperative TCD	Follow-up duration and postoperative results
1	62	Dizziness and syncope when turns head to the left	PCA velocities decreased 100%; hyperemia present (quantitative data N/A)	*MRI C-spine:* L VA compressed at C6–C7 *Angiogram*: R VA occluded; left VA occludes at C6–C7 during head turning	C6–C7 lateral discectomy *Intraoperative TCD* with passive head rotation demonstrated no decrease in PCA velocities	No decrease in PCA velocities	30 mo Mild dizziness when turning head to left; no syncope
2	52	Head turning to the right induces near syncope, blurred vision, dizziness, and leg weakness	PCA velocities decreased 91%; hyperemic response: 34% above baseline	*Angiogram*: L VA ended as PICA; absent PComA bilaterally during head turning R VA occludes at C3–C4	Anterior approach to C3–C4 with partial lateral discectomy and osteophyte removal	No decrease in PCA velocities	114 mo Asymptomatic
3	65	With head turning to the right has syncope, nausea and vomiting, dimming vision	PCA velocities decreased 75%; hyperemia 34% above baseline	*MRI C-spine:* occipitalization of C1; foraminal stenosis C2–C3 and C3–C4; Klippel-Feil malformation *Angiogram*: bilateral distal VA narrowing at the level of C1 during head turning	*First surgery:* posterior decompression of both vertebral arteries at C1–C2 by opening foramen transversarium *Second surgery:* C1–C2 fusion using transarticular screws	*TCD* after first surgery: PCA velocities decreased 75% with hyperemia to 28% above baseline *TCD* after second surgery: no decrease in PCA velocities	Still symptomatic after first surgery 3 mo after second surgery: mild dizziness; no syncope
4	76	Turning head to left causes tinnitus, near syncope, disequilibrium, and generalized weakness	PCA velocities dropped 96%; hyperemia 32% above baseline	*CT C-spine:* non-union of an old dens fracture with C1–C2 instability *Angiogram*: small L VA; both VAs nearly occlude with head rotation to the left	Posterior decompression of both VA and C1–C2 fusion using sublaminar wires	No decrease in PCA velocities	22 mo Mild neck pain
5	60	Syncope when turns head to left	PCA velocities decreased 93%; hyperemia 30% above baseline	*CTA C-spine:* occluded L VA *Angiogram*: L VA occluded; R VA occludes at C1–C2 with head turning	Partial C2 corpectomy with opening of the foramen transversarium *Intraoperative TCD* with passive head turning: no decrease in PCA velocities	No decrease in PCA velocities	12 mo Occasional dizziness with head turning; no syncope
6	54	Head rotation to right induces episodes of vertigo, nausea and vomiting, bilateral visual blurriness	PCA velocities decreased 68%; hyperemic response to 35% above baseline	*Angiogram*: hypoplastic R VA; L VA severely compressed at C5–C6 during head turning	Anterior approach to C5–C6 with osteophyte removal *Intraoperative TCD* with passive head turning demonstrated no decrease in PCA velocities	No decrease in PCA velocities	84 mo Asymptomatic
7	71	Near syncope and occipital neuralgia when turning head to left	PCA velocities decreased 60%; hyperemic response 44% above baseline	*Dynamic MRI and MRA C-spine:* R VA has decreased flow during head turning *Angiogram*: L VA hypoplastic; during head turning, there is marked stenosis of R VA at C1–C2	Decompression of R VA at C1–C2	No decrease in PCA velocities	5 mo Asymptomatic
8	68	Syncope, blackouts, dysarthria, and dimming vision induced by head turning to right	PCA velocities decreased 65%; hyperemic response to 17% above baseline	*Angiogram*: L ICA occlusion + L subclavian severe stenosis; R VA occludes at C6 during head turning	Anterior approach to C3–C4, C4–C5, C5–C6 with removal of osteophytes	No decrease in PCA velocities	3 mo Asymptomatic
9	54	Syncope when turning head to right	PCA velocities decreased 34%; hyperemic response to 41% above baseline	*CTA C-spine:* osteophytes at C3–C4 *Angiogram*: L VA occluded; severe stenosis of R VA at C3–C4 during head turning	Anterior approach to C3–C4 with removal of osteophytes	No decrease in PCA velocities	92 mo Asymptomatic
10	58	Dizziness and syncope when turning head to left	PCA velocities decreased 63%; hyperemic response to 15% above baseline	*Angiogram*: absent PComA bilaterally; compression of the L VA at C3–C4, C4–C5, and C5–C6 during head turning	Anterior approach to the cervical spine with removal of osteophytes from C3 through C6	N/A	88 mo Occasional dizziness when turns to left; no syncope

[a] TCD, transcranial Doppler; PCA, posterior cerebral artery; VA, vertebral artery; PComA, posterior communicating artery; R, right; L, left; PICA, posteroinferior cerebellar artery; ICA, internal carotid artery; CT, computed tomography; CTA, computed tomographic angiogram; MRI, magnetic resonance imaging; C-spine, cervical spine; N/A, not available.

cerebral angiogram showed a left vertebral artery ending as a posteroinferior cerebellar artery, and the right vertebral artery was severely compressed at the level of C3–C4 during head turning (*Fig. 3*). A standard approach to the anterior cervical spine with a partial lateral discectomy, removal of osteophytes and fibrous adhesions, and wide decompression of the vertebral artery was performed (*Fig. 4*). Postoperatively, there was no decrease in the PCA velocities. The patient has been asymptomatic since the surgical decompression.

Patient 5, a 59-year-old man, presented with a history of syncope when turning his head to the left. His medical history included hypertension, hyperlipidemia, pulmonary fibrosis, and coronary revascularization surgery. TCD examination showed a nearly 100% decrease of the left PCA velocities during head turning to the left. A dynamic angiogram showed an obstruction of the right vertebral artery at the C2 level during head turning and a fetal right posterior communicating artery with no cross-flow to the basilar artery. A right-sided anterior approach to the cervical spine using an incision along the medial border of the sternocleidomastoid muscle was used. The platysma was incised and the dissection continued medial to the carotid sheath. The inferior pharynx and larynx were retracted laterally, and the transverse process of C2 was identified. Doppler ultrasound was used to locate the vertebral artery course. Partial drilling of the lateral aspect of the C2 body, opening of the foramen transversarium, and release of fibrous adhesions with full decompression of the vertebral artery were performed (*Fig. 5*). Intraoperative TCD with passive head turning demonstrated no decrease in the PCA velocities. Postoperatively, TCD demonstrated no decrease in the left PCA velocities. The patient has had no syncope since surgery.

Patient 7, a 71-year-old woman, presented with near syncope and occipital neuralgia when turning her head to the left side. TCD demonstrated a 60% decrease in the PCA velocities. A magnetic resonance angiogram with the head turned suggested a compression of the right vertebral artery at the upper cervical spine. Dynamic angiograms showed marked stenosis of the right vertebral artery at the C1–C2 level (*Fig. 6*). A posterior cervical approach with opening of the C1 right foramen transversarium, lysis of adhesions, and full decompression of the vertebral artery was performed. A C2 rhizotomy, which can cause only some minor numbness in the occipital region, was also performed to relieve the occipital neuralgia. Postoperatively, TCD did not demonstrate a significant decrease in the PCA velocities during head turning. The patient has been asymptomatic since surgery.

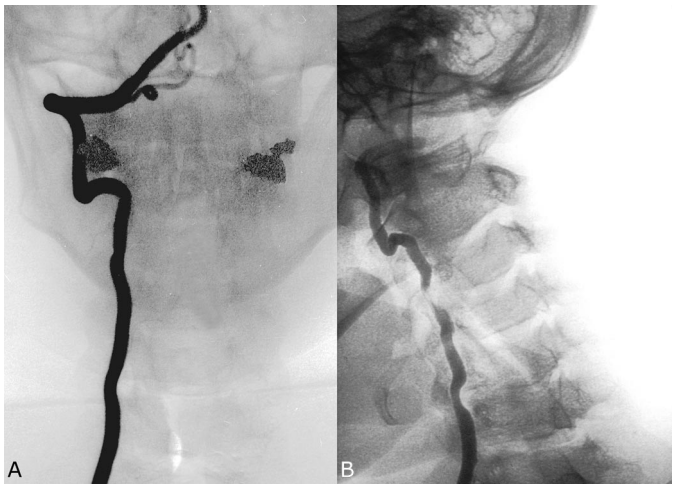

FIGURE 3. *Preoperative right vertebral artery angiograms with the head in neutral (*A*) and rotated (*B*) positions. Note the marked compression of the vertebral artery at the level of C3–C4 when the head is rotated.*

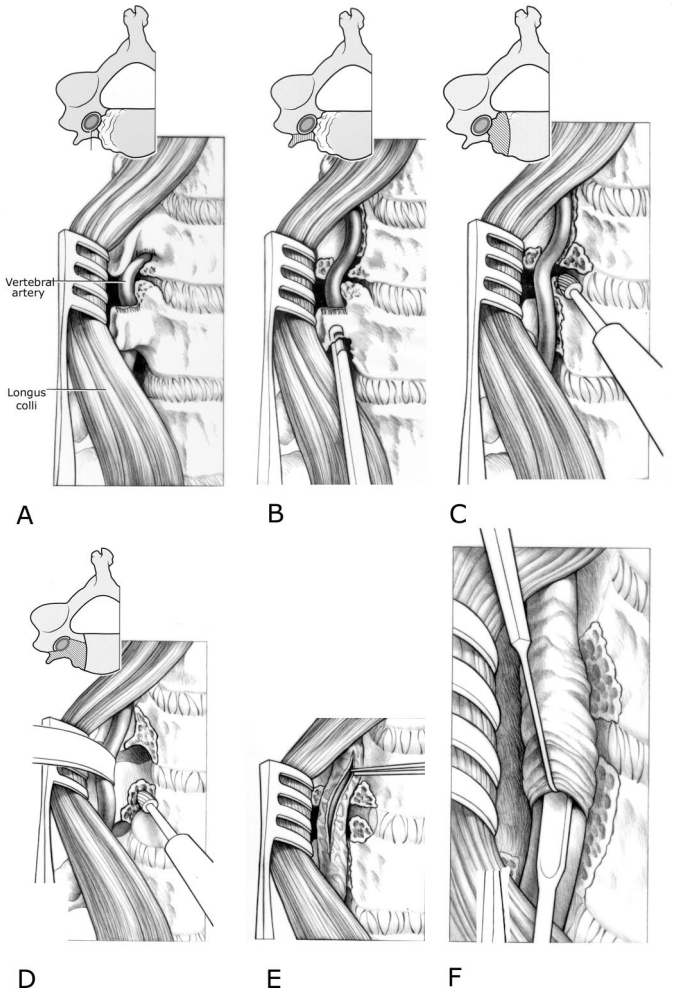

FIGURE 4. *Drawings showing surgical technique for the removal of osteophytes when performing a decompression of the vertebral artery at the subaxial spine.* A, *the longus colli is retracted laterally, and the transverse processes above and below the compression are exposed.* B, *the transverse process is removed with rongeurs.* C, *the uncinate process and osteophytes are removed with a high-speed drill.* D, *the artery is mobilized laterally, and additional drilling of medially located osteophytes is performed.* E, *the venous plexus around the vertebral artery is coagulated and incised.* F, *the fibrosed adventitia is excised to achieve full decompression of the artery.*

Surgical Treatment

The surgical approach chosen was based on the pathogenesis, mechanism, location of the vertebral artery compression, and angiographic findings. We performed 11 surgical procedures on 10 patients: one anterior decompression at C2, one posterior decompression at C1–C2, two C1–C2 fusions (including one patient who required a C1–C2 fusion as a salvage procedure), and seven decompressions of the vertebral artery

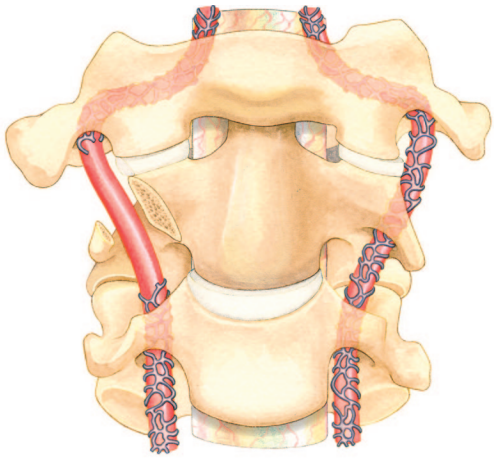

FIGURE 5. *Drawing showing extent of bone removal when using an anterior approach for a decompression of the vertebral artery at the level of C2. Also shown is the removal of adventitia and venous plexus around the vertebral artery.*

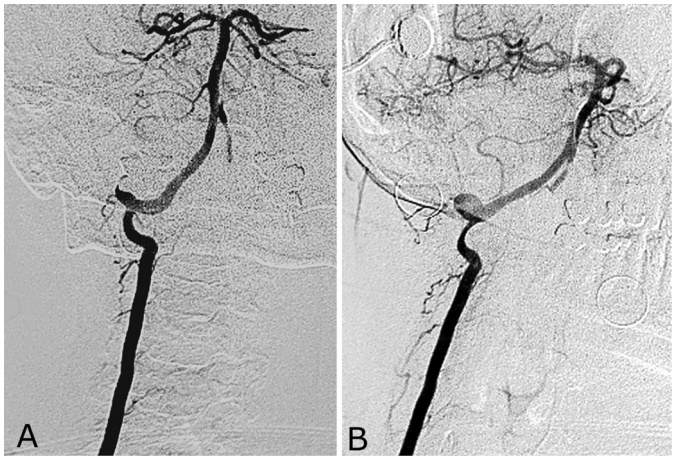

FIGURE 6. *Preoperative right vertebral artery angiograms with the head in the neutral (*A*) and rotated (*B*) positions. There is marked stenosis at the level of C1 when the head is rotated.*

at the subaxial spine, six being for removal of osteophytes and one for a disc herniation removal. We also used TCD intraoperatively in 5 patients to monitor the PCA velocities during and after the decompression. In 3 patients, we were able to obtain good and reliable signals throughout the procedure, including during a passive head turning maneuver. In another 2 patients, the signals were not reliable.

RESULTS

The TCD proved to be very useful and important in demonstrating hemodynamic changes in the posterior circulation during head-turning maneuvers in patients with rotational vertebrobasilar ischemia (*Table 1*). In this present series, there was a decrease in the PCA velocities during head turning in the range of −34 to −100% (mean, −74.5%) and a reactive hyperemic response in the range of +15 to +44% (mean, +31.3%) compared with baseline velocities (*Fig. 7*). All patients had a decrease in the mean PCA velocities of least −60%, the only exception being Patient 9, who had a mean decrease of −34%. This patient had reproducible symptoms (syncope) that correlated with the decrease in the PCA velocities and a consistent reactive hyperemic response, which justified a positive examination for rotational vertebrobasilar ischemia.

A dynamic angiogram was performed on all patients to demonstrate the exact site and extent of the vertebral artery compression and plan the operative strategy. There were four patients with compression of the vertebral artery at the level of C1–C2 and six patients with compression at the subaxial spine. Two patients (Patients 3 and 4) had bilateral compression of the vertebral artery during head turning. In nine patients, the contralateral vertebral artery was either occluded or hypoplastic or there was not a good collateral system through the posterior communicating arteries. Another patient had simultaneous bilateral occlusion of the vertebral arteries during head rotation.

The most common pathogenesis for the vertebral artery compression at the subaxial spine was an osteophyte arising from the uncinate processes. In one patient (Patient 1), the compression was caused by a C6–C7 lateral extruded disc fragment. The patients with compression of the vertebral artery at the subaxial spine had symptoms when turning their head toward the side of the affected vertebral artery. In all patients with vertebral artery impingement at the subaxial cervical spine, the symptoms resolved after removal of the mechanical cause of the compression.

The patients with compression at the C1–C2 level had symptoms when rotating their head to the contralateral side. Two patients (Patients 5 and 7) with vertebral artery compression at the C1–C2 level had a simple decompression only, and both obtained excellent results, with complete resolution of symptoms. Patient 3 had occipitalization of the C1 arch and a Klippel-Feil malformation and initially underwent only a simple decompression, which did not relieve the symptoms completely. A fusion was then performed as a salvage procedure

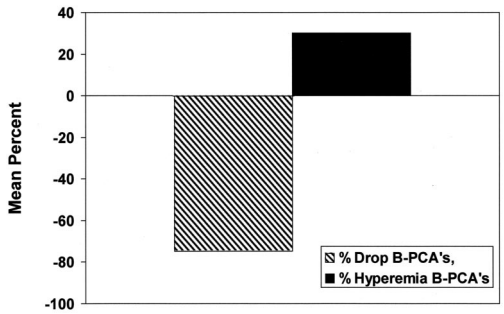

FIGURE 7. *Bar graph demonstrating the mean decrease of the PCA velocities during head turning and the mean elevation of the PCA velocities during the hyperemic response.* B, *bilateral.*

after a repeat TCD examination demonstrated a persistent decrease in the PCA velocities. The second procedure led to an improvement of the patient's symptoms. Patient 4 had evidence of C1–C2 instability preoperatively that was probably contributing to the vertebral artery compression, and a fusion was performed as a first choice. Intraoperative Doppler was very useful in three patients, documenting reestablishment of normal posterior circulation hemodynamics during a passive head turn after the surgical decompression was achieved.

All patients obtained remarkable clinical improvement and had a return to their previous level of function, and no patient experienced recurrent syncope after the surgical decompression. Nine patients had documented reestablishment of normal vertebrobasilar hemodynamics by TCD examination postoperatively. In one patient, the postoperative TCD result was not available. Follow-up ranged from 3 to 114 months, with an average follow-up of 45.3 months. There were no complications related to the surgical procedure in any patient.

DISCUSSION

Hemodynamics and Pathogenesis

Usually, when one rotates the head, there is compression of the contralateral vertebral artery at the atlantoaxial level, which is compensated by the presence of a normal ipsilateral vertebral artery that provides blood flow to the basilar system. In patients with rotational vertebrobasilar ischemia, one vertebral artery is usually hypoplastic or occluded (10, 33), and there is no significant collateral blood flow from the anterior circulation to the basilar system through the posterior communicating arteries. This explains the typical symptoms of rotational vertebrobasilar ischemia (syncope or near syncope, drop attacks, dizziness, blurred vision), all of which are caused by a temporary hemodynamically significant compression or complete occlusion of the dominant extracranial vertebral artery during head rotation, with resultant diminished blood flow to the basilar system.

Isolated occlusion of a nondominant vertebral artery and bilateral vertebral artery occlusion induced by head turning have also been reported (14, 20). Multiple episodes of stretching and compression of the vertebral artery can also lead to intimal injury and thrombus formation, with subsequent embolization and stroke (22, 23, 30, 35). The vertebral artery can be compressed or occluded at any point from its origin at the subclavian artery to the point of dural entrance into the posterior fossa. Fibrous bands at the neck (17), the anterior scalene muscle at the scalenovertebral angle (10, 15, 28), osteophytes at the foramen transversarium (21, 25, 26, 33), subluxation of the apophyseal joint and hyperrotation of the transverse process (13), subluxation of the subaxial spine, hypertrophy of the uncinate process or facets at the cervical spine, cervical disc herniation (24, 36), stretching of the artery where it courses from the C2 to the C1 foramen transversarium (5–7, 16, 19, 30, 33), or stretching at the point of dural penetration at the foramen magnum (1) have been described as mechanical causes. By far the most common cause of rotational vertebrobasilar insufficiency is an osteophyte arising from the uncinate process and compressing the vertebral artery at some point along its course in the subaxial spine (22, 26). A fibrous ring adherent to the adventitia is often found at the site of the compression, constricting the artery, and seems to play an important role in the pathogenesis of this syndrome (22, 26). In relation to the direction of the head rotation, the vertebral artery being compressed is usually the contralateral one in cases of compression at C1–C2 and the ipsilateral one when the compression is at the subaxial cervical spine (19).

Diagnosis

The value of TCD ultrasonographic studies in the evaluation and diagnosis of rotational vertebrobasilar symptoms has been shown previously (2, 33). It very useful as an initial diagnostic tool because of low costs, reproducibility, ease of use, and the possibility of performing a real-time correlation between the PCA velocities and symptoms (33). Typically, there is a significant decrease in the PCA velocities of at least 50% but more commonly to a mean of 20% of baseline when patients rotate their head (33). This is followed by a reactive hyperemic response of at least 10% above baseline velocities when the patient resumes the neutral position, which correlates with the disappearance of symptoms (33). This reactive hyperemia is a normal response to ischemia and reflects a vasodilatory phenomenon that occurs at the level of arterioles and capillary bed.

Only a few other authors have used a TCD assessment or other noninvasive cerebral hemodynamic study preoperatively to help establish the diagnosis and/or study the posterior circulation hemodynamics in patients with rotational vertebrobasilar ischemia. Garg and Edwards-Brown (6) used Doppler ultrasonography to study the vertebrobasilar system in a child with a post-traumatic thalamic stroke secondary to extrinsic compression of the vertebral artery during head turning. Jargiello et al. (12) demonstrated the usefulness of power color Doppler to image the compression of the vertebral artery in the subaxial spine in cases of vertebrobasilar insufficiency. Matsuyama et al. (19) reported five patients in their surgical series who had perfusion abnormalities in the posterior fossa on preoperative brain single-photon emission computed tomographic scans. Nakamura et al. (23), using duplex sonography, was able to document disappearance of end-diastolic flow in the right vertebral artery during head rotation that correlated with the patient's symptoms. Vates et al. (36) reported a 50% decrease in vertebral artery velocities at the level of the foramen magnum using TCD ultrasonography in a case of a herniated cervical disc causing vertebrobasilar insufficiency.

The ability to demonstrate that patients with symptoms of rotational vertebrobasilar ischemia have a measurable hemodynamic abnormality during head rotation contributes enormously to the diagnostic process. Many patients have symptoms that are not so characteristic and not easily reproducible

every time they turn their head. These patients will usually have normal hemodynamics on TCD and therefore are not considered as having rotational vertebrobasilar ischemia. In all of our patients, there was a significant decrease in the mean velocities of the PCAs during head turning, which was followed by a reactive hyperemic response upon resuming the neutral position.

We also used TCD measurements intraoperatively in five patients. In three patients (Patients 1, 5, and 6), we were able to confirm a complete decompression of the vertebral artery by detecting normal PCA velocities while performing a passive head turning. In all three patients, the PCA velocities remained normal throughout the postoperative period. In two patients, we could not detect reliable signals because of technical difficulties while performing a passive head turning maneuver. It would be most useful to monitor the PCA velocities when one is performing a posterior decompression at the C1–C2 level, the main problem being the difficulty in performing a passive head rotation maneuver in the prone position while maintaining a good Doppler signal.

The other advantage of the TCD could be clearly appreciated during the management of Patient 3, who did not have a substantial clinical improvement after an initial decompression of the vertebral arteries. Repeat TCD examination demonstrated a persistent decrease in the PCA velocities, which correlated with the symptoms. A fusion was then performed as a salvage procedure, with clinical improvement and normalization of the posterior circulation hemodynamics postoperatively.

Dynamic angiograms with progressive head rotation and multiple views are also invaluable as part of the workup for those patients who have a confirmed decrease in the PCA velocities on TCD. It demonstrates precisely the location of the vertebral artery compression and shows the vascular anatomy of the anterior and posterior circulation. It is important to visualize the entire course of both vertebral arteries from their origin all the way to the posterior fossa and interpret the results carefully so as to prevent misdiagnosis, a concept also shared by others (16).

Treatment and Surgical Options

Nonoperative treatment options include observation with limitation of head rotation. Surgery is indicated when symptoms are incapacitating and recurrent and diagnostic tests clearly demonstrate a hemodynamically significant compression of the vertebral artery as being responsible for the symptoms (22). Hardin et al. (8) reported the first case of a surgical decompression of the vertebral artery on a patient experiencing symptoms of rotational vertebrobasilar ischemia.

A simple decompression of the vertebral artery is known to be very effective when dealing with rotational vertebrobasilar ischemia resulting from various causes along the subaxial spine (15, 17, 21, 22, 24–26, 28, 33, 36). In cases of compression of the vertebral artery at the atlantoaxial level, three different approaches have been described: a posterior decompression of the vertebral artery at the level of the foramen transversarium of C1 (7, 30, 38), an anterior decompression at the level of the foramen transversarium of C1 (5), and an anterior decompression at the level of C2 (27). There is still some controversy as to whether decompression alone versus C1–C2 fusion should be performed for patients who have compression at the atlantoaxial level. Several authors have reported excellent outcomes after decompression alone (5, 7, 23, 27, 30), whereas others have performed a fusion as the initial procedure (30, 38).

In our series, we obtained excellent results in two patients with compression at the C1–C2 level by simply decompressing the vertebral artery. In another two patients, a fusion was performed: on Patient 3 because of persistent symptoms after an initial decompression and on Patient 4 because of a preexisting C1–C2 instability. It is possible that in Patient 3, the initial decompression failed because of a preexisting congenital spinal malformation, i.e., C1 occipitalization and Klippel-Feil abnormality. Unfortunately, there are no predictors of failure after a simple decompression at the upper cervical spine. Matsuyama et al. (19) performed a comparison of decompression of the vertebral arteries versus C1–C2 posterior fusion for rotational vertebrobasilar ischemia. All patients who underwent a C1–C2 fusion had complete relief of their preoperative symptoms. A 50 to 70% decreased range of motion was noticed, and five of nine patients reported a certain degree of limitation in daily activities. Six of nine patients who received decompression of the vertebral artery alone had complete relief of their symptoms, with no recurrence. Two of three patients who failed decompression alone and developed recurrent symptoms underwent a subsequent fusion, with good results. The authors did not make any comments on which factors could be responsible for the failed simple decompressions.

A point that has to be remembered when considering a C1–C2 fusion is that one vertebral artery is usually either hypoplastic or occluded. When the use of transarticular screws is contemplated, it has to be very well planned and performed so as not to injure the other, dominant vertebral artery. Other options, which carry a smaller risk of injury to the vertebral artery, are sublaminar wires (19), Halifax clamps (19), or a C1 lateral mass screw plus C2 pedicle screw fixation (9).

CONCLUSIONS

Rotational vertebrobasilar ischemia can be very incapacitating because of temporary impairment of cerebral blood flow to the brainstem, thalamus, and occipital lobes and possible posterior circulation stroke. An accurate diagnosis depends not only on clinical symptoms but also on hemodynamic and angiographic studies. The recognition of its peculiar characteristics (vertebrobasilar insufficiency symptoms, short duration, and reproducibility with a specific head maneuver) and the use of TCD as an initial screening tool are important to proper selection of patients for a dynamic angiogram. The

angiogram should be performed to localize the site and extent of compression and choose the best surgical strategy. Excellent results can be expected in the great majority of patients with a simple decompression of the vertebral artery in the subaxial spine. When there is involvement of the vertebral artery at the level of C1–C2, a simple decompression is usually effective, but a fusion may be necessary as a salvage procedure with the expense of causing a significant restriction in head motion. The TCD is also very useful to document the reestablishment of normal posterior fossa hemodynamics postoperatively and can demonstrate a persistent decrease in the posterior cerebral artery velocities in an occasional patient who is still symptomatic after an initial decompression.

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COMMENTS

Vertebrobasilar ischemia with head motion underlies some colorful neurological syndromes, such as the "bow hunter's stroke" and the "beauty parlor stroke." In this report, a 10-year surgical experience with rotational vertebrobasilar ischemia is reviewed, along with its pathogenesis, presentation, and man-

agement. The authors argue compellingly for the use of transcranial Doppler (TCD) velocity measurements, which decrease (>10%) in the posterior cerebral artery with head turning, then increase (>10%) after a neutral head position is resumed in a reactive hyperemic response. When these TCD findings correlate reproducibly with neurological symptoms and signs, catheter angiography is used to clinch the diagnosis and plan surgical management. In patients with compressive abnormalities, positive TCD studies do not eliminate the need for angiography but do provide a useful tool, albeit somewhat unreliable, for assessing the adequacy of surgical decompression intraoperatively. However, in patients without compressive abnormalities, negative TCD studies can spare them an angiogram. The effective use of TCD studies as a diagnostic screening tool in 84 of 100 patients in this experience is significant.

The surgical management of this disorder is usually straightforward, aimed at removing the offending agent at the site of vertebral artery compression. This experience demonstrates the spectrum of pathological conditions and associated vascular anomalies, such as contralateral vertebral arteries that are occluded, are hypoplastic, or terminate at the posteroinferior cerebellar artery and diminutive posterior communicating arteries that poorly collateralize the posterior circulation. The surgical technique of osteophytectomy is beautifully illustrated. I favor a diamond bit, which makes drilling safer when working immediately adjacent to the vertebral artery.

Michael T. Lawton
San Francisco, California

The authors have provided an excellent review and management of patients with rotational vertebrobasilar ischemia. This group is well known for the application of TCD, and they clearly describe its applicability in this disease process. Over the past 10 years, this group has had experience with more than 100 patients carrying the presumptive diagnosis of rotational vertebrobasilar ischemia. Within that population, they were able to truly identify 16 patients, 10 of whom went on to have surgery. Most importantly, the authors delineate the parameters in diagnosing vertebrobasilar compression by use of TCD. They find that a reduction in posterior cerebral artery velocities of 60% and a postrotational hyperemic phase of greater than 10% are diagnostic for this disease process and seem to be good predictors for improvement with surgical decompression. As indicated by the authors, this subject has been addressed on multiple occasions. However, they thoroughly review the surgical approaches and also their experience with intraoperative applications using TCD.

The authors provide excellent descriptions of their surgical approaches with descriptive diagrams. *Figure 2* nicely delineates their algorithmic approach to this disease process. The authors suggest that preoperative angiography is essential. One may question whether computed tomographic angiography, which carries a lower risk, could be used. In addition, it would clearly demonstrate the level of occlusion and the relationship between the bony anatomy and vascular compression. The three-dimensional reconstructions that can be achieved are also excellent. In addition to demonstrating the vertebral compression, simultaneous computed tomographic perfusion studies would allow quantitative analysis of the effect of vascular compression on regional cerebral blood flow.

Philip E. Stieg
New York, New York

In this article, the authors present the results of surgical intervention in 16 patients with rotational vertebrobasilar ischemia. Although it represents a large series of patients with this uncommon disorder, I believe the primary contribution of the article is their description of the novel preoperative workup performed on more than 100 patients to identify those 16 who underwent surgical treatment.

The authors have presented a very strong rationale for the use of TCD as the initial diagnostic procedure in the preoperative workup. As pointed out by the authors, many patients who are considered for this diagnosis have somewhat vague symptoms that deter the treating physician from performing dynamic vertebral angiography. In all of the patients in this series undergoing surgical decompression, TCD demonstrated a significant decrease of the mean velocities of the posterior cerebral arteries during head turning, followed by a reactive hyperemia response upon resuming the neutral position. This characteristic finding on TCD seems to lend significant support to the diagnosis, and this justifies the use of angiography to provide further anatomic detail in localizing the site of dynamic vertebral compromise. However, one could argue that those patients who did not have this characteristic finding on TCD, and thus did not undergo angiography, may have rotational vertebrobasilar ischemia that was simply undiagnosed. I suspect that this is unlikely. Although rotational vertebrobasilar ischemia is an uncommon disorder, it is readily treatable by a well-planned surgical procedure that carries a very low risk and a high likelihood of success. This contribution by Vilela et al. should assist neurosurgeons in their efforts to identify patients who benefit from those surgical procedures.

Daniel L. Barrow
Atlanta, Georgia

This is an excellent and very well-thought-out article. The authors describe a noninvasive means to study patients with symptoms suggestive of rotational vertebrobasilar ischemia. Using TCD with provocation by head rotation, the authors were able to select patients for further invasive diagnostic studies and treatment who in fact did have clear evidence of vertebral artery compromise with appropriate cerebral hemodynamic responses after vessel occlusion (hyperemia). In addition to simplifying and in fact quantifying the diagnostic accuracy, the authors were able to very specifically select those patients who not only had the disease but were likely to benefit from vertebral artery decompression through a variety of techniques.

It is important for the reader to remember that during this 10-year experience, more than 100 patients presented with a clinical picture suggestive of rotational vertebral artery compromise. In fact, only 16

patients of this much larger group had objective evidence of arterial compromise. Thus, use of this important diagnostic test prevented the majority of patients from being subjected to diagnostic angiography and potentially decompressive procedures who in fact would not be expected to benefit from either. Using these very carefully performed noninvasive techniques, the authors were able to achieve excellent clinical and hemodynamic results with an extremely low complication rate.

Richard J. Parkinson
H. Hunt Batjer
Chicago, Illinois

Vilela et al. present 10 cases of vertebrobasilar ischemia associated with rotation of the cervical spine and resulting from transient vertebral artery compression by different spinal diseases. Although long recognized, this syndrome is probably underdiagnosed. The author's use of ultrasound both to make the diagnosis and to judge the surgical result builds nicely on the work of Nakamura and his colleagues. The authors have called our attention to this important entity, which is a readily treatable cause of stroke.

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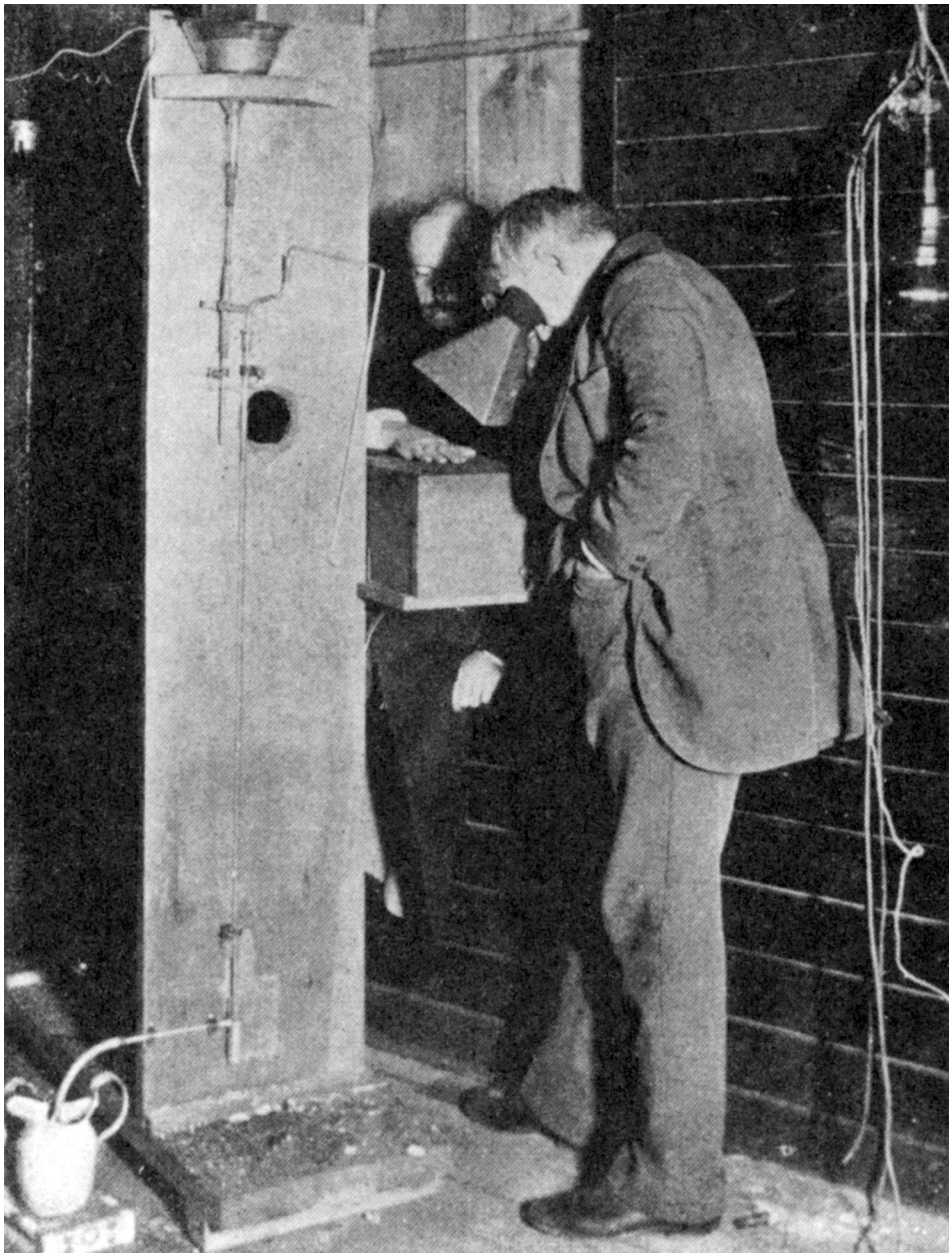

Thomas Edison examines a hand through a fluoroscope. He demonstrated the device for visitors at the 1896 Electrical Exhibition in New York. (Courtesy of the Burdy Library, Dibner Institute, Cambridge, Massachusetts.)