Bow hunter's syndrome revisited: 2 new cases and literature review of 124 cases

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Rotational occlusion of the vertebral artery (VA), or bow hunter's syndrome, is a rare yet surgically treatable cause of vertebrobasilar insufficiency. The underlying pathology is dynamic stenosis of the VA by osteophytes, fibrous bands, or lateral disc herniation with neck rotation or extension. The authors present 2 previously unreported cases of bow hunter's syndrome and summarize 124 cases identified in a literature review.

Both patients in the new cases were treated by VA decompression and fusion of the subaxial spine. Each had > 50% occlusion of the left VA at the point of entry into the transverse foramen with a contralateral VA that ended in the posterior inferior cerebellar artery. Analyzing data from 126 cases (the 2 new cases in addition to the previously published 124), the authors report that stenosis was noted within V₁ in 4% of cases, in V₂ in 58%, in V₃ in 36%, and distal to C-1 in 2%. Patients presented in the 5th to 7th decade of life and were more often male than female. The stenotic area was decompressed in 85 (73%) of the 116 patients for whom the type of treatment was reported (V₁, 4 [80%] of 5; V₂, 52 [83%] of 63; V₃/V₄, 29 [60%] of 48). Less commonly, fusion or combined decompression and fusion was used (V₂, 7 [11%] of 63; V₃/V₄, 14 [29%] of 48). Most patients reported complete resolution of symptoms.

The authors conclude that patients with bow hunter's syndrome classically have an impaired collateral blood flow to the brainstem. This condition carries an excellent prognosis with decompression, fusion, or combined surgery, and individual patient characteristics should guide the choice of therapy.

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KEY WORDS bow hunter's syndrome; cervical spine; decompression; dynamic vertebrobasilar insufficiency; fusion; rotational vertebrobasilar insufficiency; vertebral artery; rotational occlusion

“Bow hunter's syndrome” is the descriptive synonym for rotational vertebrobasilar insufficiency. This condition is elicited by rotation or extension of the neck and caused by dynamic and reversible occlusion of the vertebral artery (VA) and lack of collateral blood supply to the brainstem. The literature on bow hunter's syndrome is limited to case reports and small series. Our review of 124 cases reported in the English literature from 1952 to 2011 along with 2 new cases from our own experience summarizes patient characteristics, symptoms, surgical strategies, and outcomes for patients with this rare syndrome of recurrent vertebrobasilar insufficiency that can be cured with spine surgery.

Case Reports

Case 1

This 55-year-old man had experienced 2 months of syncope attacks when he extended his neck and loss of vision when he turned his head to the left or up. MRI showed a left-sided foraminal disc herniation at C6–7 and a left dominant VA (Fig. 1). The right VA terminated in the posterior inferior cerebellar artery (PICA). On dynamic angiography, neck extension led to a 20% stenosis of the left VA and neck rotation to the left to a stenosis of 50% (Fig. 2). To decompress the left VA, the left transverse foramen of C-6 was unroofed via an anterior approach, and the left

ABBREVIATIONS CTA = CT angiography; DSA = digital subtraction angiography; MRA = MR angiography; PICA = posterior inferior cerebellar artery; SPECT = single-photon emission CT; VA = vertebral artery.

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FIG. 1. Case 1. MRI and CTA findings in a 56-year-old patient with bow hunter's syndrome. **Left:** A foraminal disc herniation is seen at the left C6–7 foramen (white arrow). **Right:** The left dominant VA (black arrow) has a larger caliber than the right VA.

discoligamentous herniation at C6–7 and uncarthrosis at C-6 were removed. C-6 and C-7 were then fused with a tricortical iliac crest allograft and anterior plate. At 6 months' follow-up, the patient could rotate his neck to the left without symptoms and reported minor symptoms with prolonged neck extension.

Case 2

This 47-year-old woman reported recurrent dizziness, disturbance of vision, and fainting spells triggered by rotating the neck to the left. She had experienced episodic neck and hand pain and upper-extremity clumsiness for several years, consistent with progressive myeloradiculopathy. The results of sensorimotor testing were normal except for brisk reflexes and a Hoffman sign on the right. MRI of the cervical spine revealed spondylosis with a midline disc protrusion at C3–4, a left foraminal disc herniation at C5–6 (Fig. 3 left), and a broad-based disc herniation at C6–7. Angiography showed dynamic stenosis of the left VA at C5–6, corresponding to the area of the herniated disc (Fig. 3 right). The right VA ended in the PICA. Since the patient had symptomatic cervical spondylosis, the decompression and fusion of C5–6 was extended to the degenerated segments C3–4 and C6–7 (not shown). Postoperatively, the fainting spells did not recur, although she continued to experience neck pain and stiffness.

Literature Review

We searched PubMed for the keywords “bow hunter's syndrome,” “bow hunter's stroke,” “rotational vertebrobasilar insufficiency,” and “dynamic vertebrobasilar insufficiency.” All papers written in English were obtained as full articles except for 1 historic report,¹⁰ and cross references were checked for additional reports, which were also obtained as full articles. An early report on 20 patients with rotational occlusion of the VA proximal to the C-6 transverse foramen and by tendons of the longus colli and scalenus anticus muscle was not included because data were only available in summarized form.¹⁷

Analysis

From 1952 to November 2011, 43 papers reported on 124 cases (Tables 1–3). The 2 largest series consisted of 20

cases in the subaxial cervical spine (C3–7)²⁸ and 17 cases at C1–2.²⁵ For purposes of analysis, we are including the 2 cases reported in the present paper, which brings the total number of cases analyzed to 126.

Patient Characteristics

Including our patients, there were 2 pediatric patients,⁶ 3 adolescents,^{6,10,22} and 121 adults (Fig. 4). Occlusion was located proximal to C-7 (V₁) in 5 (4%) cases, between C-3 and C-7 (V₂) in 73 (58%), at C1–2 (V₃) in 45 (36%), and distal to C-1 (V₄) in 3 (2%). Patients typically presented in the 5th to 7th decade of life; the mean age at presentation was 54 ± 7 years for patients with occlusion at V₁, 57 ± 12 years for occlusion at V₂, and 53 ± 18 years for occlusion at V₃. The mean age at diagnosis in all of the adults was 57 ± 11 years. The male-to-female distribution was 2:1 overall, with 80% of cases in the V₁ segment, 74% in the V₂, and 61% in the V₃ and V₄ segments occurring in male patients.

Dynamic stenosis of the VA was relatively evenly distributed between the left (49.5%) and right (36.7%) sides; it was bilateral in 13.7% of cases. Pediatric patients presented with congenital anomalies such as a bony malformation at C1–2 or a suboccipital bony protuberance impinging on the VA.^{6,22} In adults, the compressive cause depended on the location. At the craniocervical junction, the VA was fixed to an ossified or thickened atlanto-occipital membrane (Fig. 5)^{37,41} or compressed by a dural fold in the foramen magnum,¹ an assimilated posterior ring of C-1 with bilateral condylar and clival hypoplasia and platybasia,³⁴ an accessory ossicle behind the left atlanto-odontoid junction,⁵³ erosive rheumatoid arthritis of C1–2,³⁶ or C1–2 facet hypertrophy.⁵ In the subaxial cervical spine, the VA was most frequently compressed by a bone spur of a hypertrophied uncovertebral joint,^{4,13,15,28,32,42,50} rarely by a laterally herniated disc.^{30,49} In 1 case, the VA was impinged by scissoring between the anterior root of the upper transverse process and a subluxating superior articular process of the inferior vertebral body;¹⁹ in another case, there was a chronic VA dissection;¹⁸ and in a third case, an anomalous prevertebral course, with the VA entering the transverse foramen at C-4 only, led to a compression of the VA between the thyroid cartilage and the anterior aspect of the C-6 transverse process.⁷ Fibrous bands and a tight scalenovertebral angle resulted in dynamic flow obstruction in the first segment of the VA.^{12,21,23,39,45} Although most cases manifested spontaneously, a posttraumatic onset of symptoms has also been reported.^{41,53}

By definition, rotational vertebrobasilar insufficiency is characterized by reversible symptoms that are elicited upon neck rotation. The most commonly reported symptoms were syncope, near-syncope, and drop attacks; vertigo; dizziness; and impaired vision (Table 4). Other symptoms, including paresis, deficits, and pain, were also reported. Symptoms were present before diagnosis for a median of 12 months (range 1 week to 20 years).

Dynamic angiography and later digital subtraction angiography (DSA), CT angiography (CTA) and MR angiography (MRA) with and without 3D reconstructions, extracranial and transcranial Doppler sonography, and single-photon emission CT (SPECT) were all used in the evaluation of patients. Dynamic angiography and Doppler

FIG. 2. Case 1. Dynamic angiography of the left VA in a 56-year-old patient with bow hunter's syndrome. **A:** The left VA is dominant and fully patent in the neutral position. **B:** Neck extension causes a 20% stenosis at C6–7. **C:** Neck rotation to the left causes a 50% stenosis.

sonography were also applied intra- and postoperatively to document resolution of the stenosis.^{50,51,53}

At C1–2, the stenosis was on the right side in 30% of the patients for whom the affected side was specified (13 of 44) and on the left in 64% (28 of 44). Three patients (7%) had bilateral stenosis,^{5,6} and the affected side was not indicated in 1 case (Fig. 4).¹⁰ In the 3 patients with stenosis distal to C-1, it was bilateral in 1, on the left in 1, and on the right in 1. From C-3 to C-7, the stenosis was on the right side in 40% of the patients for whom the side was specified (23 of 57), on the left in 40% (23 of 57), bilateral in 19% (11 of 57). The stenotic side was not specified in 16 cases. Monosegmental stenosis was observed in 52 patients, multisegmental in 8 including 1 patient with stenosis at C5–6 and C1–2. In 13 patients the stenotic level within V₂ was not specified.

Where mentioned, symptoms were exclusively triggered by a contralateral neck rotation in patients with a stenosis of the VA at C1–2. In contrast, an ipsilateral neck rotation evoked symptoms in 94% of patients (34 of 36) with a subaxial stenosis of the VA (V₂). It was also reproducible by a contralateral rotation in 8% (3 of 36)^{3,13,40} and

exclusively by a contralateral rotation in 1 case.⁵¹ Three of 5 patients with a dynamic stenosis of the VA proximal to C-7 were affected on the right side and 2 on the left. Symptoms were caused by ipsilateral rotation in 3 and contralateral rotation in 2 patients. Threshold angles at which neck rotation triggered symptoms were 30°,^{4,30} 45°,^{5,20,34,37,49} 55°,⁹ 60°,^{45,52} and 90°.^{19,47} In 22 papers, the authors reported that the contralateral VA of their patients was hypoplastic, absent, or terminating in a PICA, and 6 noted a deficient circle of Willis with absent posterior communicating arteries (Tables 1–3).

Conservative Management

Management strategies were described for 116 of the cases (Table 5). In total, 9 cases (8%) were initially managed conservatively, including 4 cases in which patients chose conservative therapy (cervical collar, refraining from extreme neck rotations, aspirin) despite recommended surgery.^{16,34,36,37} One patient chose conservative therapy because symptoms were only triggered by extreme neck rotation during chiropractic maneuvers.⁷ In another pa-

FIG. 3. Case 2. MRI and angiographic findings in a 47-year-old patient with bow hunter's syndrome. **Left:** MR image shows a left foraminal disc herniation (*asterisk*) that abuts the left VA (*arrows*). **Right:** Angiogram revealing the corresponding stenosis of the left VA at C5–6.

tient, bow hunter's syndrome was brought on by a dissected contralateral VA. Symptoms disappeared with recanalization of the dissection.⁵² One patient was treated with halter traction.⁴⁰ Two patients were initially treated with a trial of bracing and subsequently underwent decompression surgery.^{11,13}

Interventional Management and Bypass Surgery

Two patients had stents placed.^{29,45} In 1 patient with a VA stenosis in the subaxial cervical spine and a concomitant ostial atherosclerotic stenosis of the same artery, symptoms disappeared after stenting of the ostial atherosclerotic stenosis, which increased perfusion pressure across the subaxial stenosis.²⁹ One patient underwent a PICA to PICA bypass surgery.¹⁸

Decompression

The stenotic area was treated with decompression in 85 (73%) of the 116 patients for whom management strategies were described (V_1 , 4 [80%] of 5; V_2 , 52 [83%] of 63; V_3 and V_4 , 29 [60%] of 48), and symptoms improved or disappeared in 87% of the patients with available follow-up (65 of 75) (Table 6). Six patients (8%) experienced residual or persistent symptoms: vertigo,⁶ hemiparesis,⁶ dizziness,⁵¹ and persistent syncope.⁵¹ Symptoms improved after secondary fusion of C1–2 in 1 patient. Symptoms recurred in 4 patients;^{19,25} 2 of these patients underwent a secondary fusion, 1 was treated with anticoagulants,²⁵ and 1 patient was only freed of symptoms after 2 decompressions at the same level and an additional decompression at another level.¹⁹ One patient underwent a secondary fusion procedure because of C1–2 instability.²² To decompress a narrowed VA in the subaxial cervical spine, most authors unroofed the transverse foramen by removing the costal process and, if present, any impinging discoligamentous tissue.²² Resecting the fibrous adventitia around the VA was also recommended.⁵¹ Stenoses at the craniocervical junction were approached from posterior^{14,41} or anterolateral^{6,11} directions.

Fusion

Thirteen (27%) of 48 patients with stenosis at C1–2 or

TABLE 1. Cases of bow hunter's syndrome caused by a stenosis in the V_1 segment of the vertebral artery

Authors & Year	Age (yrs), Sex	Side	Level	Trigger	Duration (mos)	Sx	Contributing Factors	Therapy	Outcome, Latest Follow-Up
Mapstone & Spetzler, 1982	54, M	Rt	V_1 , close to SA	Rt rot	—	Vertigo, syncope, lt leg paresis & hypesthesia	Lt VA hypoplastic	Decompr	No symptoms
Kojima et al., 1985	64, M	Rt	V_1 , 3 cm from origin	Rt rot	7	Vertigo		Decompr	No symptoms, 18 mos
Fujimoto et al., 1988	50, M	Lt	V_1 , 2 cm from origin C6–7	Rt rot	1	Dizziness, vertigo, nausea, emesis		Decompr, section of sympathetic chain; removal of ant TF at C–6	No symptoms, 6 mos; miosis
Sell et al., 1994	44, F	Lt	V_1 , 2 cm from origin	Lt rot	—	Amaurosis, lt arm pain & paresthesia, facial & neck pain		Decompr w/ resection of 1st rib & division of mid & ant scalene muscles	No symptoms
Sugiu et al., 2009	56, M	Rt	V_1	Lt rot	—	Vertigo, syncope	Lt VA stenotic origin	Stenting of origin of lt VA	No symptoms, 6 mos

Ant = anterior; decompr = decompression; mid = middle; rot = rotation; SA = subclavian artery; Sx = symptoms; TF = transverse foramen; VA = vertebral artery; — = not available.

TABLE 2. Cases of bow hunter's syndrome caused by a stenosis in the V₂ segment of the vertebral artery

Authors & Year	Age (yrs), Sex	Side	Level	Trigger	Duration (mos)	Sx	Contributing Factors	Therapy	Outcome, Latest Follow-Up
Sheehan et al., 1960	43, M	Lt	V ₂	Lt rot	60	Nystagmus, dizziness, lt trigeminal hypalgnesia, lt neck & arm pain, lt arm paresis	Spondylosis, bilat VA compression on ipsilat rot	Halter traction	—
	48, M	—	V ₂	Lt/rt rot, ext	2	Blurred vision, transient amaurosis, dizziness, drop attack, neck pain	Spondylosis, bilat VA compression	—	—
	68, F	Lt, rt	V ₂	Rot	19	Dizziness, drop attack, transient quadriplegia, bilat hearing loss, hand & feet paresthesia	Lt & rt VA compressed by osteophytes	—	—
	51, M	—	V ₂	Rot	132	Nystagmus, diplopia, drop attack, progressive paraparesis, paresthesias in fingers & toes	Cervical spondylosis	Laminectomy	—
	73, M	—	V ₂	Rot, ext	120	Nystagmus, dizziness, drop attack, syncope, right arm paresis, ataxia	Cervical spondylosis, compression of rt VA by bony spur	—	—
Bauer et al., 1961	32, M	Rt, lt	V ₂	Lt/rt rot	—	Vision, drop attacks, ataxic gait, lt hemiparesis, nystagmus, vertigo	Spondylotic compression of both VAs w/ ipsilat rot	Fusion	No Sx, 6 mos
Gortvai, 1964	56, M	—	V ₂	Lt/rt rot, ext	—	Dizziness, drop attacks, syncope, rt hemiparesis, neck pain	Rt VA absent, compression of lt VA at C5–6 by fibrocartilaginous mass, disc, bone spur	C-collar, then decompr of lt VA at foramen C5–6 & removal of ant TF at C-6	Immediate recovery & no Sx, 5 mos
Hardin, 1965	67, M	—	C-6	Rot	3	Dizziness, tinnitus, deafness	—	Decompr of TF at C-6	Persisting tinnitus & deafness, 31 mos
	50, M	—	C-6	Rot	36	Blurred vision, dizziness, dysarthria, tinnitus, ataxia	—	Decompr of TF at C-6	No Sx, 58 mos
	62, F	—	C-2	Rot	2	Blurred vision, diplopia, aphasia, hand & leg hypesthesia, headache	—	Decompr of TF at C-2	Hoarse, 12 mos
	64, M	—	C5–6	rRot	6	Diplopia, syncope, dysarthria, hand hypesthesia, aphasia, dysphagia	—	Decompr of TF at C-5, C-6	No Sx, 19 mos
	45, M	—	C-6	Rot	3	Blurred vision, dizziness, syncope, hand hypesthesia	—	Decompr of TF at C-6	No Sx, 12 mos
	60, M	—	V ₂	Rot	1	Blurred vision, diplopia, dizziness, syncope, ataxia, hand hypesthesia	—	Decompr of TF at C2–6	No Sx, 11 mos
	81, M	—	V ₂	Rot	3 days	Blurred vision, dysarthria, arm paresis, hand hypesthesia	—	Decompr of TF at C-6	Dizziness, arm paresis, 14 mos
	60, F	—	C-6	Rot	6	Dysarthria, limb paresis	—	Decompr of TF at C-6	No Sx, 16 mos
	75, F	—	C5–6	Rot	1	Blurred vision, diplopia, dizziness, syncope	—	Decompr of TF at C5–6	No Sx, 6 mos

(continued)

TABLE 2. Cases of bow hunter's syndrome caused by a stenosis in the V₂ segment of the vertebral artery (*continued*)

Authors & Year	Age (yrs), Sex	Side	Level	Trigger	Duration (mos)	Sx	Contributing Factors	Therapy	Outcome, Latest Follow-Up
Bakay & Leslie, 1965	47, M	Rt	C5–6	Ext, flex	48	Dizziness, syncope, hypesthesia in both hands	Lt VA hypoplastic	Decompr of bone spur, fusion C5–6	No Sx, 5 mos
	42, M	Rt	C5–6	Rt rot, ext	4	Dizziness, occipital headache	—	Fusion C5–6	No Sx, 8 mos
	—	—	V ₂	—	—	Dizziness	—	—	—
Nagashima, 1970	45, M	Rt	C6–7	Rt rot	276	Vertigo, bilat tinnitus, nausea, ataxia, rt CN V, VII, IX paresis	—	Decompr	No Sx
	63, M	Rt	C5–6	Rt rot	29	Dizziness, ataxia, rt CN IX & XII paresis, cervical radiculopathy	—	Decompr	Improved
	47, M	Rt	C6–7	Rt rot	72	Blurred vision, dysphagia, cervical radiculopathy, neck pain, headache	—	Decompr	Improved
	54, F	Rt, lt	C5–6	Rot	21	Blurred vision, dizziness, presyncope, nausea, neck pain	—	Decompr	No Sx
	66, M	Rt	C5–6	Rt rot	6	Oscillopsia, vertigo, hypesthesia in arms & legs	—	Decompr	No Sx
	44, F	Rt, lt	C6–7	Rt, lt rot	22	Transient amaurosis, vertigo, presyncope, headache	—	Decompr	Improved
	32, M	Lt	C4–5	Rot	48	Blurred vision, dizziness, diplopia, headache	—	Decompr	No Sx
	53, M	Lt	C5–6	Rot	60	Vertigo, presyncope, ataxia, rt CN V, VII, VIII, IX paresis	—	Decompr	Improved
	48, F	Lt, rt	C4–5; C6–7	Rot	120	Episodic amaurosis, dizziness, rt hemiparesis, lt CN V, VII, IX	—	Decompr	Improved
	58, F	Lt, rt	C5–6	Rot	96	Drop attack, rt/lt hemiparesis	—	Decompr	—
	41, F	Rt	C5–6	Rot	48	Blurred vision, vertigo, presyncope, lt hemiparesis, rt CN VII, IX, XII paresis	—	Decompr	No Sx
	50, M	Rt	C5–6	Rot	86	Blurred vision, oscillopsia, vertigo,	—	Decompr	No Sx
	56, M	Rt	C5–6	Rot	24	Blurred vision, headache, rt CN IX paresis	—	Decompr	No Sx
	52, M	Rt	C4–5	Rot	6	Oscillopsia, dizziness, ataxia, headache	—	Decompr	No Sx
	38, M	Lt	C4–5	Rot	36	Transient amaurosis, vertigo, syncope, lt/rt hemiparesis & hemiparesis, ataxia, rt CN IX paresis	—	Decompr	No Sx
	56, M	Rt	C5–6	Rot	24	Blurred vision, diplopia, vertigo, syncope, ataxia, dysphagia, rt CN V & IX paresis	—	Decompr	No Sx
	63, M	Lt, rt	C4–5	Rot	48	Blurred vision, vertigo, syncope, ataxia	—	Decompr	Died of MI
	71, F	Lt	C5–6	Rot	36	Dizziness, vertigo, neck pain	—	Decompr	No Sx
	35, F	Lt	C5–6	Rot	15	Blurred vision, dizziness, nausea, emesis, neck pain	—	Decompr	Improved
	58, F	Lt	C5–6	Rot	24	Dizziness, syncope, nausea, emesis, cervical radiculopathy, ataxia	—	Decompr	No Sx

(continued)

TABLE 2. Cases of bow hunter's syndrome caused by a stenosis in the V₂ segment of the vertebral artery (continued)

Authors & Year	Age (yrs), Sex	Side	Level	Trigger	Duration (mos)	Sx	Contributing Factors	Therapy	Outcome, Latest Follow-Up
Smith et al., 1971	51, F	Lt	C5–6	Ext	12	Syncope, dysbalance, headache	—	Decompr C5–6 disc & bilat osteophytes	No Sx, 6 wks
	53, M	Lt, rt	C4–5; C5–6	Rot, ext	12	Vertigo, lt hemifacial hypesthesia, lt biceps paresis	—	Bilat decompr C4–5, C5–6	No Sx
Sturzenegger et al., 1994	37, F	Rt	C5–6; C1–2	Rt rot	—	Blurred vision, dizziness, vertigo, tinnitus, leg paresis, hemihypesthesia	Absent PCoAs	—	—
	52, M	Rt	C3–4	Rt rot	—	Blurred vision, dizziness, syncope vertigo, presyncope, leg paresis, leg hypesthesia	Lt VA hypoplastic & terminating in PICA, absent PCoAs	—	—
	76, F	Lt	C-2	Lt rot	—	Leg paresis, leg paresthesias, loss of balance, headache	Non-union odontoid Type II Fx; rt VA compressed at C-2 in neutral position, no PCoAs	—	—
	71, M	Rt	V ₂ , multilevel	Rt rot	—	Blurred vision, dizziness, vertigo	Lt VA absent, no PCoAs	—	—
DeMaria, 1995	70, F	Lt	V ₂	Lt rot	12	Dizziness	Rt VA absent	—	—
Kawaguchi et al., 1997	56, M	Rt	C-6; C4–5; C1–2	Rt rot	—	Amaurosis, tinnitus	Bilaterally hypoplastic PCoAs	1 & 2) decompr at rt C-6 TF; 3) decompr at rt C-4 TF	Recurrence 2 mos after 1st op, persistence after 2nd op, recovery after 3rd op
Ogino et al., 2001	66, M	Rt	C3–4	Rt rot	4	Nystagmus, vertigo, presyncope	—	Decompr at C-4 TF	No Sx
Vates et al., 2002	56, M	Lt	C4–5	Lt rot	6	Dizziness, syncope	Lt rot also caused extrinsic compression of rt VA at V ₃	Decompr at C-4 & C-5 TF	No Sx 6 wks
Nemecek et al., 2003	61, M	Lt	C6–7	Lt rot, ext	1	Vertigo, syncope, nausea	Rt VA absent, Lt VA stenotic	Decompr, removal of disc fragment entering TF	No Sx, 6 mos
Vilela et al., 2005	62, —	Lt	C6–7	Lt rot	—	Dizziness, syncope	Rt VA occluded	Decompr, C6–7 lat disc-ectomy	Mild dizziness, no syncope, 30 mos
	52, —	Rt	C3–4	Rt rot	—	Blurred vision, presyncope, leg paresis	Lt VA terminating in PICA, PCoAs absent	Decompr C3–4	No Sx, 114 mos
	54, —	Lt	C5–6	Rt rot	—	Blurred vision, vertigo, nausea, emesis	Rt VA hypoplastic	Decompr C5–6	No Sx, 84 mos
	68, —	Rt	C-6	Rt rot	—	Blurred vision, syncope, dysarthria	Lt ICA occluded	Decompr C3–4, C4–5, C5–6	No Sx, 3 mos
	54, —	Rt	C3–4	Rt rot	—	Syncope	Lt VA occluded	Decompr C3–4	No Sx 92 mos
	58, —	Lt	C3–4; C4–5; C5–6	Lt rot	—	Dizziness, syncope	Absent PCoAs	Decompr C3–6	Occasional dizziness, 88 mos
Velat et al., 2006	58, M	Lt	V ₂	Lt rot	12	Dizziness, vertigo, presyncope	—	Decompr at C-4, C-5, C-6 TF	No Sx, 4 wks

(continued)

TABLE 2. Cases of bow hunter's syndrome caused by a stenosis in the V₂ segment of the vertebral artery (*continued*)

Authors & Year	Age (yrs), Sex	Side	Level	Trigger	Duration (mos)	Sx	Contributing Factors	Therapy	Outcome, Latest Follow-Up
Bulsara et al., 2006	55, M	Rt	C5-6	Rt rot	240	Syncope	—	ACDF C5-6, decompr at TF C5-6	No Sx, 6 wks
Dabus et al., 2008	41, M	Rt	C-6, thy-roid	Rt rot	—	Dizziness	Rt dominant VA enters only at C-4	Conservative	No Sx, 9 mos
Kim et al., 2008	60, M	—	C2-3	Lt rot	—	Dizziness	Rt VA distally occluded	Decompr at C-2 TF	—
Tsutsumi et al., 2008	59, M	Rt, lt	C-6; C-5	Lt rot	12	Presyncope	—	Decompr at lt C-5 TF, rt C-6 TF	—
Miele et al., 2008	48, M	Lt	C4-5	Lt rot	12	Syncope	Rt VA hypoplastic	Decompr & fusion C4-5	No Sx
Petridis et al., 2008	66, M	Rt	C4-5	Rot/ext	—	Drop attacks	—	Pst decompr of C4-5 foramen & TF	No Sx
Natello et al., 2009	76, M	Rt	C5-6 C4-5	Rt rot/ext	6	Presyncope, ataxia, diplopia, blurred vision, dysarthria, bilat leg paresis, falls	Lt VA occluded, w/ dynamic rt VA stenoses by spondylosis & atherosclerotic ostial stenosis of rt VA	Stenting of proximal rt VA	No Sx, 14 mo
Ujifuku et al., 2009	78, M	Rt	C4-5	Rt rot	—	Blurred vision, syncope	Lt VA hypoplastic	—	—
Lu et al., 2010	74, M	Lt	C4-5	—	—	Syncope, vertigo	—	Decompr	No Sx, 100 mos
	55, M	Rt	C-4	—	—	Syncope, vertigo	—	Decompr	No Sx 84 mos
	67, M	Lt	C4-5	—	—	Syncope, vertigo	—	Decompr	No Sx 37 mos
	70, M	Lt	C5-6	—	—	Syncope, vertigo	—	Decompr	No Sx 6 mos
	73, M	Lt	C2-3	—	—	Vertigo	Far-lat decompr	Decompr	No Sx 60 mos
Kan et al., 2012	41, M	Rt	V ₂	Rt rot	—	Vertigo, nausea, emesis	Rt posttraumatic VA dissection, rt VA ending in PICA	PICA-to-PICA bypass	—
Present case	55, M	Lt	C6-7	Lt rot	2	Syncope, loss of vision	Rt VA ending in PICA	Decompr C6-7, fusion	No Sx; 6 mos
Present case	47, F	Lt	C5-6	Lt rot	—	Syncope, blurred vision, dizziness	Rt VA ending in PICA	Decompr, fusion C3-7	No Sx 3 mos

ACDF = anterior cervical discectomy and fusion; CN = cranial nerve; ext = extension; flex = flexion; Fx = fracture; MI = myocardial infarction; PCoA = posterior communicating artery; PICA = posterior inferior cerebellar artery; pst = posterior; — = not available.

TABLE 3. Cases of bow hunter's syndrome caused by a stenosis in the V₃ and V₄ segments of the vertebral artery

Authors & Year	Age (yrs)			Duration (mos)		Trigger	Side	Level	Sx	Contributing Factors	Therapy	Outcome Latest Follow-Up
	Sex	Level	Side	Level	Side							
Ford, 1952	17, M	—	—	—	—	—	—	C1-2	Syncope, nystagmus, diplopia, vertigo, ataxia	Os odontoidum	C1-2 fusion	No Sx
Yang et al., 1985	55, M	Lt	C1-2	Rt rot	12	Rt rot	—	C1-2	Amaurosis	Rt VA hypoplastic, absent lt PCoA, rt PCA hypoplastic	C1-2 fusion	No Sx, 6 mos
Robinson et al., 1986	45, M	Lt, rt	C1-2	Lt rot; rt rot	3	—	—	C1-2	Syncope	Juvenile RA, degenerated C1-2 joints	C1-2 fusion offered, pt chose collar, aspirin	Persisting Sx when collar off, 10 mos
Shimizu et al., 1988	37, M	Lt	C1-2	Rt rot	1	Rt rot	—	C1-2	Diplopia, lt Horner's, nystagmus, dizziness, unsteadiness, lt hemihypesthesia	Prior trauma w/ C-1 fracture	Decompr	No Sx at 2 yrs
Hanakita et al., 1988	74, F	Lt	C1-2	Rt rot	1	Rt rot	—	C1-2	Vertigo, drop attacks, nausea, lt hemiparesis	Lt PCoA hypoplastic; rt VA ended in rt PICA	Decompr, dorsal TF removed	No Sx at 2 yrs
	53, F	Rt	C1-2	Lt rot	0.25	Lt rot	—	C1-2	Vertigo	Lt PCoA hypoplastic	Decompr dorsal TF removed	—
	48, F	Rt	C1-2	Lt rot	11	Lt rot	—	C1-2	Dizziness, vertigo, nausea	Lt PCoA hypoplastic; lt PCoA ended in lt PICA	Decompr, dorsal TF removed	No Sx
Fox et al., 1995	53, F	Lt	C1-2	Rt rot	3	Rt rot	—	C1-2	Blurred vision, nausea, presyncope	Rt VA was occluded, probably secondary to traumatic dissection at C-1	Conservative for 6 mos, then decompression of lt VA by arteriolat removal of TF at C-1	No Sx except lt CN IX palsy
Morimoto et al., 1996	70, M	Lt	C1-2	Rt rot, ext	36	Rt rot, ext	—	C1-2	Vertigo, nausea, syncope	Lt VA terminated in lt PICA	C1-2 fusion	No Sx
Matsuyama et al., 1997 ²⁵	57, M	Rt	C1-2	Rot	—	Rot	—	C1-2	Vertigo	—	C1-2 fusion	No Sx
	63, F	Lt	C1-2	Rot	—	Rot	—	C1-2	Dizziness	—	C1-2 fusion	No Sx
	65, M	Lt	C1-2	Rot	—	Rot	—	C1-2	Syncope	—	C1-2 fusion	No Sx
	59, M	Lt	C1-2	Rot	—	Rot	—	C1-2	Facial hypesthesia	—	C1-2 fusion	No Sx
	61, M	Lt	C1-2	Rot	—	Rot	—	C1-2	Vertigo	—	C1-2 fusion	No Sx
	53, F	Rt	C1-2	Rot	—	Rot	—	C1-2	Vertigo	—	C1-2 fusion	No Sx
	73, M	Lt	C1-2	Rot	—	Rot	—	C1-2	Syncope	—	C1-2 fusion	No Sx
	75, F	Lt	C1-2	Rot	—	Rot	—	C1-2	Vertigo	—	C1-2 fusion	No Sx
	56, F	Lt	C1-2	Rot	—	Rot	—	C1-2	Syncope	—	C-1 decompr	Reocclusion of VA, treated w/ C1-2 fusion
	53, M	Rt	C1-2	Rot	—	Rot	—	C1-2	Dizziness	—	C1-2 decompr	No Sx
	55, F	Lt	C1-2	Rot	—	Rot	—	C1-2	Syncope	—	C-1 decompr	No Sx
	64, M	Rt	C1-2	Rot	—	Rot	—	C1-2	Dizziness	—	C1-2 decompr	No Sx
	51, M	Rt	C1-2	Rot	—	Rot	—	C1-2	Dizziness	—	C-1 decompr	Reocclusion of VA, treated w/ anticoagulant
	58, M	Lt	C1-2	Rot	—	Rot	—	C1-2	Vertigo	—	C-1 decompr	No Sx
	57, F	Lt	C1-2	Rot	—	Rot	—	C1-2	Vertigo	—	C1-2 decompr	Reocclusion of VA, treated w/ C1-2 fusion
	71, F	Lt	C1-2	Rot	—	Rot	—	C1-2	Vertigo	—	C1-2 decompr	No Sx
	59, M	Lt	C1-2	Rot	—	Rot	—	C1-2	Syncope	—	C-1 decompr	No Sx

(continued)

TABLE 3. Cases of bow hunter's syndrome caused by a stenosis in the V₃ and V₄ segments of the vertebral artery (*continued*)

Authors & Year	Age (yrs), Sex	Side	Level	Trigger	Duration (mos)	Sx	Contributing Factors	Therapy	Outcome Latest Follow-Up
Sakai & Tsutsui, 1999	39, M	Rt	C1-2	Lt rot	—	Syncope, dizziness, nausea	Lt VA hypoplastic, terminated in PICA	Refused surgery, aspirin	—
Seki et al., 2001	47, M	Lt	C1-2	Rt rot	—	Syncope	Rt VA occluded	Ant decompr	—
Horowitz et al., 2002	55, M	Rt	C1-2	Lt rot	—	Vertigo, dysbalance, presyncope	Lt VA hypoplastic, rt PCA fetal	C1-2 fusion recommended, pt refused	—
Akar et al., 2000	30, F	Lt	C0-1	Rt rot	18	Dizziness, vertigo, drop attacks	Rt VA hypoplastic, terminating in 2nd metameric muscular branch, PCoAs absent	Decompr at dural penetration	No Sx
Puca et al., 2000	54, M	Rt	C1-2	Lt rot	—	Vertigo, dizziness, blurred vision, syncope,	Craniocervical malformation	Conservative	—
Netuka et al., 2005	54, M	Lt	C1-2	Rt rot/ext	240	Vertigo, syncope, nausea, rt hemiparesis, rt hemianopia	Rt VA absent	Decompr	—
Vilela et al., 2005	65, —	Rt, lt	C1-2	Rt rot	—	Blurred vision, syncope, nausea, emesis	Occipitalization of C-1, Klippel-Feil C2-3, C3-4	1) decompr VA C1-2; 2) C1-2 fusion	1) persistent Sx; 2) mild dizziness
	76, —	Rt	C1-2	Lt rot	—	Presyncope, tinnitus, dysbalance, weakness	C1-2 instability caused by non-union of odontoid Fx; lt VA small	Decompr & fusion C1-2	No Sx, 22 mos
	60, —	Rt	C1-2	Lt rot	—	Syncope	Lt VA occluded	Partial C-2 corpectomy, decompr of TF at C-2	Occasional dizziness on rot, 12 mos
	71, —	Rt	C1-2	Lt rot	—	Near syncope	Lt VA hypoplastic	Decompr C1-2	No Sx, 5 mos
Wakayama et al., 2005	45, F	Lt	C1-2	Rt rot	1	Dizziness, neck pain, blurred vision, syncope	Rt VA dissection, absent PCoAs	Conservative	No Sx, 6 mos, recanalized VA
Whitmore et al., 2007	57, M	Lt	C1-2	Rt rot	—	Syncope	Accessory ossicle impinging on lt VA	Decompr	No Sx, 6 mos
Chough et al., 2010	71, F	Lt	C1-2	Rt rot	36	Vertigo, hypesthesia, memory loss	—	C1-2 fusion	No Sx
Lu et al., 2010	77, F	Lt	C1-2	—	—	Amaurosis, tinnitus	—	Far-lat decompr	No Sx
	58, M	Lt	C1-2	—	—	Syncope	—	Far-lat decompr	No Sx
	65, F	Lt	C1-2	—	—	Syncope	—	1) far-lat decompr; 2) fusion	No Sx
	12, M	Rt	C0-1	—	—	Syncope, ataxia, headache	—	Decompr	No Sx
Cornelius et al., 2012	42, M	Lt	C1-2	Rt rot, ext	6	Syncope, vertigo	—	Anterolateral decompr	No syncope, less vertigo
	46, F	Lt	C1-2	—	>12	Amaurosis, dysphasia, dysphagia, dizziness, neck pain	—	Anterolateral decompr	No syncope
	16, M	Rt, lt	C1-2	Rt rot, lt rot	12	Syncope, neck pain	—	Anterolateral decompr	No Sx
	9, M	Rt, lt	C0-1	Lt rot	12	Vertigo, nausea, nystagmus, torticollis	—	Fusion	No Sx, 74 mos
	8, M	Lt	C1-2	Rt rot	6	Vertigo, ataxia, nausea, hemiparesis	—	Anterolateral decompr	Residual hemiparesis

C0 = occiput; PCA = posterior cerebellar artery; pt = patient; RA = rheumatoid arthritis; — = not available.

FIG. 4. Location of dynamic occlusion of the VA. Among patients categorized as having V₂ occlusions, 8 were described as having multisegmental occlusions. In 52 cases, the occlusions were specifically described as monosegmental, while in the remaining 13, information about the number of segments affected was not provided.

distal to C-1 underwent atlantoaxial or occipitocervical fusion,^{10,24,27,54} and 2 (3%) of 63 patients with compression at V₂ had fusion in the subaxial cervical spine^{2,3} without exposure and decompression of the VA. All were reported to have symptom-free outcomes.

Decompression and Fusion

Five (8%) of 63 patients with compression at V₂, including our 2 patients, had decompression and fusion during the same surgery for a stenosis of the VA in the subaxial cervical spine,^{2,4} and 1 patient with a stenosis involving C1-2 was treated with decompression and occipitocervical fusion.⁵¹ No recurrences were noted.

Discussion

Sorensen⁴³ coined the term “bow hunter’s syndrome”

after a patient who developed Wallenberg stroke during archery practice. This is in the strict sense different from the reversible symptoms of rotational vertebrobasilar insufficiency that are usually described as bow hunter’s syndrome. Therefore, we have not included Sorensen’s case, which remains the only report of the syndrome in a bow hunter. Although a 3-tiered classification has been proposed to distinguish different causes,³⁷ most authors have adopted the term to refer to rotational vertebrobasilar insufficiency.

The summarized data enable us to characterize this rare syndrome: typically, a patient presents in the 6th decade, is male, and experiences reversible symptoms of vertebrobasilar insufficiency upon neck rotation. Symptoms are most likely syncopal or near-syncopal events, drop attacks, vertigo and dizziness, and impaired vision. Imag-

FIG. 5. Illustrations showing a potential cause of bow hunter’s syndrome involving the V₃ segment of the vertebral artery. Although a patient may experience no symptoms when the cervical vertebrae are in a neutral position (A), contralateral rotation (B) may elongate and squeeze the VA under a thickened atlanto-occipital membrane in the vascular groove and transverse foramen of the atlas. Resection of the atlanto-occipital membrane and opening the transverse foramen (C) frees the artery and eliminates the compression. Copyright Department of Neurosurgery, University of Utah. Published with permission.

TABLE 4. Symptoms reported in 126 patients with bow hunter's syndrome

Symptom	No. of Patients (%)
Syncope, near syncope, drop attacks	69 (54)
Vertigo	49 (39)
Dizziness	45 (35)
Impaired vision	43 (34)
Limb paresis	21 (17)
Ataxia	20 (16)
Nausea, emesis	19 (15)
Cranial nerve deficits	16 (13)
Hypesthesia	12 (9)
Diplopia	10 (8)
Paresthesia	9 (7)
Headache	9 (7)
Neck pain	7 (6)
Tinnitus	7 (6)
Nystagmus	7 (6)
Dysarthria, aphasia	7 (6)
Arm pain	5 (4)
Dysphagia	4 (3)
Hypoacusis	2 (2)

ing is likely to reveal dynamic stenosis of the VA in the subaxial cervical spine in two-thirds of patients and at the craniocervical junction in one-third. This may be associated with a poor collateralization: either a hypoplastic or an absent contralateral VA or a deficient circle of Willis. Symptoms are most commonly elicited by ipsilateral rotation if the occlusion is in the subaxial cervical spine and by contralateral rotation if the occlusion is at the craniocervical junction. The condition is usually caused by degenerative changes in adults and congenital anomalies of the craniocervical junction in pediatric patients. Surgical therapy carries an excellent prognosis.

Conservative therapy is limited to wearing a cervical collar and receiving verbal warnings to avoid extreme neck rotations or to antiplatelet therapy intended to im-

TABLE 5. Treatment stratified by segment

Treatment	V ₁	V ₂	V ₃	V ₄
Not reported	10/73 (14%)			
Conservative*	2/63 (3%)		5/45 (11%)	
Decompr	4/5 (80%)	52/63 (83%)	27/45 (60%)	2/3 (67%)
Fusion	0/5	2/63 (3%)	12/45 (27%)	1/3 (33%)
Decompr & fusion	0/5	5/63 (8%)	1/45 (2%)	
Secondary fusion†			4/27 (15%)	
VA stent	1/5 (20%)	1/63 (2%)		
PICA-to-PICA bypass		1/63 (2%)		

* Count does not include patients who underwent a surgery after failed conservative treatment.

† Of the 27 patients who underwent VA decompression, 4 underwent a secondary fusion procedure because of persistent or recurrent symptoms.

TABLE 6. Outcome of decompression and/or fusion*

Treatment	Asymptomatic	Sx Persistence	Sx Recurrence
Decompr	65/75 (87%)	6/75 (8%)	4/75 (5%)
Fusion	14/14 (100%)	0	0
Decompr & fusion	6/6 (100%)	0	0

* Based on reports with available outcome data.

prove perfusion across the stenosis. In a minority of the reviewed cases, the patients did not have surgery, and follow-up was only reported in 3 of these cases: one patient did fine with a cervical collar and aspirin but experienced recurrent symptoms whenever the collar was off;³⁶ in the 2 other cases, the patients were referred for surgical treatment after failure of bracing.^{11,13} We therefore conclude that conservative therapy plays a marginal role and may not suit the lifestyle of the typical patient.

The options for surgery include decompression, fusion, or a combination. The literature suggests an excellent outcome regardless of the strategy. Most authors performed decompression without fusion, and this resolved the symptoms in 87% of patients. None of the patients who had fusion or decompression and fusion had recurrence of symptoms. Fusion was also a successful salvage procedure for recurrences or persistent symptoms after decompression.^{19,51} Thus, anecdotal evidence suggests that fewer recurrences of bow hunter's syndrome occur with fusion than with decompression alone. This must be weighed, however, against the drawbacks of fusion procedures, particularly at the occipitocervical junction, where neck rotation will be noticeably reduced. Lastly, the comfort level of the surgeon is likely to influence the choice of treatment: an anterior fusion procedure in the subaxial cervical spine or a posterior fusion at the occipitocervical junction may appear more straightforward to the spine surgeon who has limited experience with decompression of the VA.

Workup and Management Algorithm

In Fig. 6, we suggest a treatment and management algorithm. Given the natural history of rotational vertebrobasilar insufficiency, transcranial Doppler confirms the diagnosis if velocities of the posterior cerebral arteries drop by $\geq 50\%$ upon neck rotation and surpass baseline values by $\geq 10\%$ when the patient resumes a neutral neck posture.^{44,51} Hemodynamic compromise with head rotation can also be evaluated with SPECT.²⁴ Dynamic imaging studies such as DSA, CTA, or MRA reveal the side and localization of the reversible stenosis and the status of the contralateral VA and circle of Willis.¹⁶ A decompression without fusion may be appropriate if degenerative changes are limited. Conversely, anecdotal evidence suggests that a fusion procedure decreases the likelihood of postoperative recurrence, particularly at C1–2, but limits neck rotation by 50%–70%.²⁴ Intraoperative or postoperative Doppler ultrasound,^{51,53} dynamic DSA,⁵⁰ CTA, or MRA can be used to monitor successful decompression of the VA. Recurrent symptoms in a patient who has not had a fusion procedure are best managed by fusion of the causative segments.²⁴

FIG. 6. Diagram showing proposed management algorithm for rotational vertebrobasilar insufficiency.

Impaired Collateralization at Baseline and Physiological Versus Pathological Dynamic Stenosis

Impaired collateral blood supply to the posterior circulation is common: Hypoplasia of one VA occurs in 10% to 20% of patients,⁴⁴ and termination of the VA in the PICA is the second most frequent anomaly. Moreover, the circle of Willis often reveals impaired collateralization, as hypoplasia involving the posterior communicating arteries was present in 68% of specimens obtained from adults presenting with neurological dysfunction.³⁵ These configurations may predispose a patient to vertebrobasilar insufficiency during dynamic occlusion of the VA, which can be physiological or pathological.^{3,8,40,46} Stretching of the contralateral VA in its C1–2 course as well as forward and lateral pressure by the lateral mass of C-1 causes physiological rotational stenosis. Conversely, spondylotic changes in the vicinity of the course of the artery through the transverse foramen can impinge on the VA and decrease flow during ipsilateral head rotation.⁴⁰

Limitations

Our data were retrieved from case reports and clinical series, all of which were retrospective and differed in the extent of available information for each case. For instance, some authors probably confined symptoms to the most rel-

evant ones, omitting others, or did not report the detailed findings of cerebral arteriography. Moreover, other important data, such as long-term follow-up, were often missing. Still, this compilation of 126 cases represents the largest overview of bow hunter's syndrome to date, and the descriptive analysis of the retrieved data allowed us to create a characteristic patient scenario and develop a treatment and management algorithm for workup and therapy.

Conclusions

Bow hunter's syndrome, or rotational vertebrobasilar insufficiency, most often involves syncopal or presyncopal attacks, vertigo, and/or impaired vision, although the symptoms may vary. It usually develops in the 6th decade. It is caused by dynamic and reversible occlusion of the VA with neck rotation, more frequently in the subaxial spine than at the occipitocervical junction. Patients classically have an impaired collateral blood flow to the brainstem. This condition carries an excellent prognosis with decompression, fusion, or a combined surgery.

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