My Approach To: Bow Hunter's Syndrome

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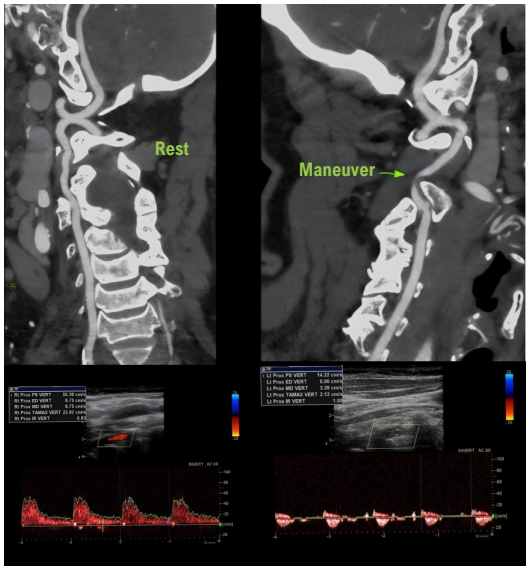
Central Illustration: My Approach To: Bow Hunter's Syndrome



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Top left: CT angiography of the vertebral artery at rest demonstrating normal opacification. Bottom left: Vertebral Doppler ultrasound at rest showing normal blood flow. Top right: CT angiography with contralateral neck rotation demonstrating arterial compression between C1 and C2. Bottom right: Doppler ultrasound with contralateral neck rotation demonstrating a staccato flow pattern.

Abstract

Bow hunter's syndrome (BHS) is a rare and underdiagnosed condition characterized by mechanical compression or temporary occlusion of the vertebral artery due to lateral rotation or hyperextension of the neck. This mechanical stress occurs predominantly in the V3 segment (C1–C2 level) because of its high degree of mobility.

The pathophysiology involves two main mechanisms – hemodynamic, in which extreme rotation immediately reduces local blood flow and causes paroxysmal symptoms (the triad of vertigo, dizziness, and imbalance) that resolve once the head returns to the neutral position; and thromboembolic, triggered by repeated microtrauma and chronic endothelial injury, which may lead to thrombus formation or dissections and result in cerebellar or brainstem infarctions.

Definitive diagnosis is based on dynamic digital subtraction angiography (DSA) and computed tomography angiography (CTA). However, dynamic vascular Doppler serves as an excellent non-invasive screening method, capable of recording critical real-time hemodynamic changes through positional maneuvers, such as a greater than 50% drop in peak systolic velocity (PSV) or disappearance of diastolic flow.

Therapeutic management varies according to severity. Mild cases are treated conservatively with a cervical collar, behavioral modifications, and antiplatelet therapy. Surgical interventions – such as decompression via osteophytectomy or cervical arthrodesis – and specific endovascular treatments are strictly reserved for patients with refractory symptoms or high risk of stroke.

Keywords

Vertebral Artery; Rare Diseases; Diagnosis

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Introduction

Bow hunter's syndrome (BHS), also known as rotational vertebral artery syndrome or rotational occlusion of the vertebral artery, is a rare and underdiagnosed condition in clinical practice, classified among the compressive syndromes of the vertebrobasilar system. It is characterized by compression or occlusion of the vertebral artery during lateral head rotation, resulting in transient or permanent neurological deficits.

The name derives from the posture adopted by archers when shooting arrows, during which they perform a vigorous rotation of the neck. An important variant of BHS occurs due to hyperextension of the head, known as the beauty parlor stroke syndrome, described in relation to the position assumed during hair washing in salon sinks.[1,2]

Incidence

The exact incidence of BHS remains unknown in the general population due to its dynamic nature and the high rate of underdiagnosis. It is more frequent among individuals aged 50 to 79 years, with a male predominance of approximately 2:1. However, it may occur at any age, including in children.[1]

Pathophysiology

To understand the pathophysiology of BHS, it is necessary to correlate the anatomical course of the vertebral artery (Figure 1) – traditionally divided into four segments – with its specific mechanical vulnerabilities during cervical kinematics (Table 1).

The V3 segment, due to its anatomical particularities, is the most frequently affected site. This occurs because the hypermobility of the atlantoaxial joint (C1–C2) accounts for approximately 50% of total cervical rotation,[3] making this region particularly susceptible to dynamic compression of the vertebral artery.

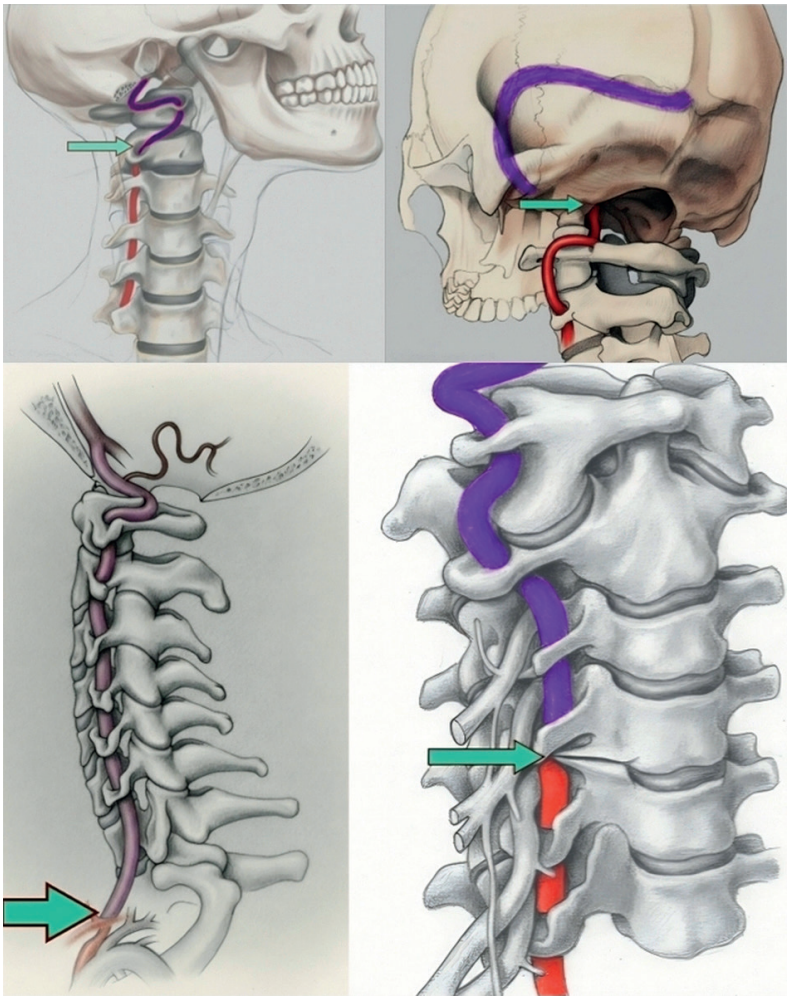

Figure 1 – *Segments of the vertebral artery; bottom left: V1 segment; bottom right: V2 segment; top left: V3 segment (the most frequent site); top right: V4 segment.*

Arterial dominance is another critical factor: compression of a dominant vertebral artery carries a greater potential for hemodynamic repercussions, especially when associated with hypoplasia of the contralateral vessel.

Two pathophysiological mechanisms may occur either independently or simultaneously. The hemodynamic mechanism manifests when extreme cervical rotation (generally greater than 60°) causes mechanical compression and an immediate reduction in blood flow. This drop in perfusion compromises the most vulnerable regions of the posterior circulation, resulting in transient neurological symptoms, which resolve as soon as the head returns to the neutral position and the vessel regains its patency. The severity of symptoms is directly related to the degree of dynamic stenosis, the presence of contralateral hypoplasia, and the efficiency of the circle of Willis in compensating for the deficit through the posterior communicating arteries.

The thromboembolic mechanism, in turn, is associated with chronic structural damage to the arterial endothelium. Mechanical stress and repeated compression of the vertebral artery produce endothelial microtrauma, culminating in subintimal dissections or mural thrombi. These may give rise to distal embolization, causing cerebellar or brainstem infarctions, even without sustained cervical rotation. This phenomenon is particularly observed in the pediatric population, in which cervical hypermobility predisposes to arterial dissections after minimal trauma or abrupt rotational movements.[4]

The clinical presentation of BHS is typically paroxysmal and strictly related to head position. The classic symptomatology consists of a triad of vertigo, dizziness, and imbalance triggered by lateral rotation of the neck, and may also include syncope, diplopia, auditory disturbances, or infarcts within the posterior circulation.

The differential diagnoses include benign paroxysmal positional vertigo, Ménière's disease, and vestibular migraine, which makes it imperative to investigate transient symptoms triggered exclusively by cervical rotation.

Parameter	Behavior in BHS (rotation)
PSV	Absence of flow or >50% reduction
EDV	Absence or marked reduction
Wave morphology	Staccato pattern – distal Tardus-parvus pattern (dampened) - proximal
RI	Reduction – proximal Increase – distal
AT	Increases in proximal obstructions

Table 1 – *Flow patterns observed on vascular Doppler in BHS. PSV: Peak Systolic Velocity; EDV: End-Diastolic Velocity; RI: Resistive Index; AT: Acceleration Time; BHS: Bow Hunter's syndrome.*

Diagnosis

The gold standard for diagnosis is dynamic digital subtraction angiography (DSA). Dynamic computed tomography angiography (CTA) also plays an important role, as it allows three-dimensional reconstruction, assessment of the stenotic segment, and identification of the anatomical structure responsible for the compression.

Dynamic vascular Doppler emerges, in this context, as a highly effective screening tool, as it is an accessible, low-cost, radiation-free method that does not require contrast administration. The dynamic nature of the examination allows real-time assessment of the vessel's hemodynamic behavior.

The examination should be performed with the patient in a neutral position and in maximum contralateral rotation, evaluating both vertebral arteries, preferably in the V1 and V2 segments and, when accessible, V3. Neck flexion and extension may be combined with rotation to enhance the maneuver.

It is essential to maintain the position for 30 to 60 seconds to increase the sensitivity of the test, since flow alterations may exhibit a latency period. Whenever possible, the patient should be asked to adopt the exact position that typically triggers the symptoms.

Although the greatest point of arterial stretching occurs at the C1–C2 level during contralateral rotation combined with extension, some authors suggest measuring flow in up to nine different positions, combining neutral position, flexion, extension, and ipsilateral and contralateral rotations.[5]

During the procedure, velocities and flow patterns are recorded in the neutral position for later comparison during the maneuvers (Figure 2). Flow variations with a reduction greater than 50% in peak systolic velocity (PSV) are indicative of significant compression. End-diastolic velocity (EDV) often shows a marked reduction; however, the most robust criterion is the complete disappearance of diastolic flow during rotation, which reflects increased downstream vascular resistance due to distal vessel occlusion (Central Illustration).

Vascular resistance rises considerably during distal compression maneuvers and decreases during proximal ones. In severe cases, complete absence of flow may be observed, consistent with total occlusion. Another detectable parameter is reactive hyperemia, characterized by an increase greater than 10% in flow velocities after returning to the neutral position. Contralateral evaluation is mandatory, given the risk of bilateral involvement.

The flow pattern obtained provides information not only about the presence of compression but also about the segment involved. In proximal compressions, a dampened (*tardus-parvus*) pattern is observed, with increased acceleration time and reduced peak velocity. At the exact point of compression, turbulence with elevated velocities may occur. In distal segments, there may be absence of flow, reduced systolic and diastolic velocities

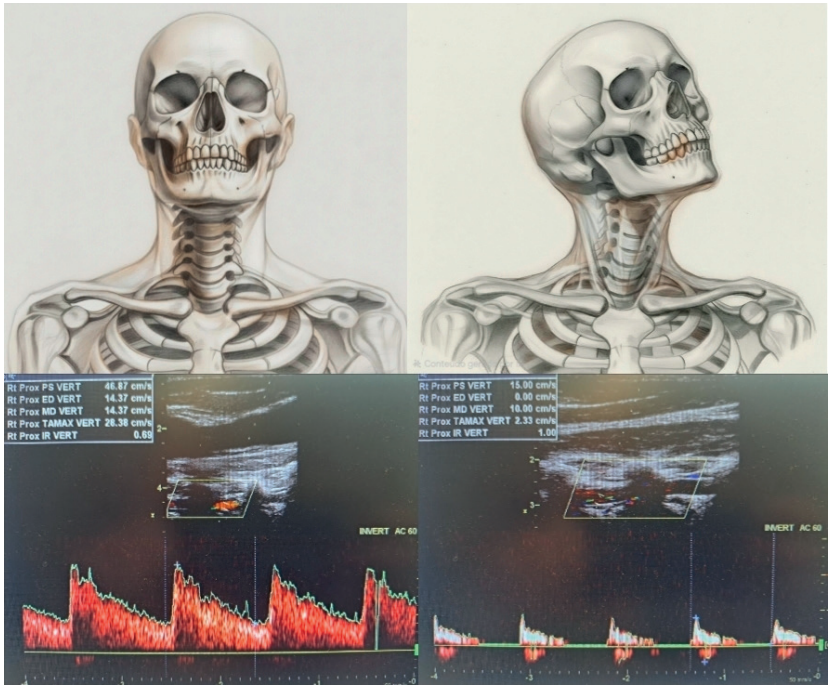

Figure 2 – *Vascular Doppler of the V2 segment performed in the neutral position (left), demonstrating normal flow; on the right, the same patient shows staccato-pattern flow after contralateral rotation maneuver.*

with increased resistance index (RI), or a staccato-type pattern (Table 2).

The reproduction of neurological symptoms during the maneuvers, with immediate reversal upon returning to the neutral position, supports the positivity of the test.

Treatment

The therapeutic management of BHS must be individualized based on the severity of symptoms, the risk of stroke, and the presence of cervical instability.

In patients with mild, infrequent symptoms and no structural neurological lesions, conservative management is the first-line approach. This strategy is based on behavioral modifications (avoiding the triggering position) or the use of a cervical collar. Antithrombotic therapy with antiplatelet agents (such as aspirin or clopidogrel) is indicated to prevent thromboembolic events resulting from endothelial injury. Although safe, conservative treatment shows effectiveness in controlling limiting symptoms or reducing stroke risk in only about 50% of cases.

Endovascular treatment remains controversial, as BHS results from an extrinsic compressive force that may flatten or fracture the stent structure, especially when the V3 segment is involved. Therefore, this approach should be reserved for highly selected cases.[6]

Surgical intervention is indicated for severe or refractory cases. It consists of decompression through osteophytectomy

Segment of the vertebral artery:	Anatomical description	Bow hunter's relevance
V1 (pre-apophyseal)	Origin from the subclavian artery to the transverse foramen of C6	Less common site; compression by fibrous bands or muscles
V2 (inter-apophyseal)	Course through the transverse foramina from C6 to C2	Frequently affected by osteophytes and disc herniations
V3 (atlanto-axial)	Exit at C2, looping over C1 to the foramen magnum	Most common site (C1-C2) due to the high amplitude of rotation
V4 (intracranial)	From the foramen magnum to the basilar artery junction	Rarely the primary site of extrinsic mechanical compression

Table 2 – *natomical segments of the vertebral artery and main causes of obstruction in BHS.*

(removal of osteophytes), a procedure that generally preserves cervical range of motion, although it carries a risk of recurrence due to postoperative fibrosis.

An alternative is cervical arthrodesis, which shows the highest success rate in preventing permanent neurological events; however, it imposes a definitive limitation on neck mobility.[1]

Discussion

BHS is a neglected and frequently underdiagnosed condition capable of causing severe limitations and permanent neurological sequelae, making early diagnosis crucial. Vascular Doppler, due to its wide availability and reproducibility, has become an indispensable tool for screening suspected patients, especially those with neurological symptoms closely related to changes in cervical position.

Clinical suspicion in cases of syncope without an apparent cause is essential, and routine dynamic positional maneuvers should be incorporated into cervical vascular examination protocols. Although methods such as dynamic CTA and DSA remain crucial for anatomical mapping and final therapeutic planning, Doppler plays a decisive role in optimizing and guiding the indication for these invasive examinations.

Author Contributions

Conception and design of the research, acquisition of data, analysis and interpretation of the data, statistical analysis, obtaining financing , writing of the manuscript and critical revision of the manuscript for intellectual content: Visconti RB.

Potential Conflict of Interest

No potential conflict of interest relevant to this article was reported.

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Study Association

This study is not associated with any thesis or dissertation work.

Ethics Approval and Consent to Participate

This article does not contain any studies with human participants or animals performed by any of the authors.

Use of Artificial Intelligence

The authors did not use any artificial intelligence tools in the development of this work.

Availability of Research Data

The underlying content of the research text is contained within the manuscript.

References

1. Duan G, Xu J, Shi J, Cao Y. Advances in the Pathogenesis, Diagnosis and Treatment of Bow Hunter's Syndrome: A Comprehensive Review of the Literature. Interv Neurol. 2016;5(1-2):29-38. doi: 10.1159/000444306.
2. Mavrovounis G, Drivas K, Syrakouli A, Syrakouli E, Mermiri M, Papanagiotou P, et al. Stroke at the Hairdresser's chair: A Scoping Review of the Beauty Parlor Stroke Syndrome. Am J Emerg Med. 2025;91:29-36. doi: 10.1016/j.ajem.2025.02.016.
3. Choi KD, Choi JH, Kim JS, Kim HJ, Kim MJ, Lee TH, et al. Rotational Vertebral Artery Occlusion: Mechanisms and Long-Term Outcome. Stroke. 2013;44(7):1817-24. doi: 10.1161/STROKEAHA.113.001219.
4. Wang X, Liu X, Yu L, Xue Y, Hu K, Song Y, et al. Endothelial Senescence of the Vertebral Artery in the Context of Cervical Degeneration: Biomechanically Driven Remodeling of the Hemodynamic Environment. Front Cell Dev Biol. 2026;14:1839226. doi: 10.3389/fcell.2026.1839226.
5. Kimihira L, Yoshimoto T, Ihara M. New Diagnostic Algorithm for Detection of Covert Bow Hunter's Syndrome. Int J Med Sci. 2021;18(10):2162-5. doi: 10.7150/ijms.56442.
6. Chen H, Colasurdo M, Costa ML, Kan P. Endovascular Management of Extracranial Vertebral Artery Stenosis. J Neurointerv Surg. 2025;18(1):3-7. doi: 10.1136/jnis-2024-022618.