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Instability at the uncovertebral C3/C4 joint causes "The bow hunter's stroke."

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Abstract
Spinal artery blockage or stenosis brought on by repeated head motions characterizes a bow hunter's stroke. Hypoplastic, stenotic, or blocked contralateral vertebral arteries and minimal input from the anterior circulation via the circle of Willis typically characterize the isolating conditions that lead to the limited blood flow. Vertebrobasilar insufficiency can cause a wide variety of symptoms, from mild vertigo and nystagmus to severe hemiparesis, sensory abnormalities, Horner's syndrome, difficulty swallowing, and even loss of consciousness. When blood flow is briefly blocked, symptoms may be short-lived, but when an embolus or thrombus causes an infarction in the afflicted vascular region, they can last much longer. When the head is rotated while the artery is anchored by surrounding bony or fibrous elements, occlusion of the extra-cranial vertebral artery occurs. Angiography is the gold standard, but other dynamic investigations measuring absolute or relative changes in cerebral blood flow due to head movement are also necessary for diagnosis. Surgery to restrict head rotation or decompress and release the compressed vertebral artery is one of many possible treatments. This syndrome carries a high chance of a cerebrovascular accident with long-lasting consequences, so it's important to take action to reduce that risk. When deciding between surgical procedures, it is important to take into account both the patient's current health and their goals.

Keywords: bow hunter’s stroke, vertebrobasilar, insufficiency

Introduction
Bow hunter syndrome, or rotational vertebral artery syndrome, is an extremely unusual form of vertebrobasilar insufficiency. Patients with Bow Hunter syndrome may experience compression of the vertebral artery at the atlantoaxial or sub-axial levels as a result of abnormal rotation of the head and neck. The term "bow hunter syndrome" was coined after a patient named Bow Hunter who suffered from Wallenberg syndrome (a lateral medullary infarction) while practicing archery due to poor circulation from keeping his head rotated for long durations.
The pathophysiology of Bow Hunter Syndrome relies heavily on knowledge of the anatomy of the vertebral artery. It is from the subclavian arteries that the arteries to the vertebrae branch off. As they travel upward via the cervical spine, they enter the transverse process of C6 (or C7 in 7.5% of instances) and exit through the transverse foramen. The vertebral arteries leave the brain via the foramen magnum, which is reached via an ascent through the transverse foramen of the atlas (C1).

Definition
Bow hunter's stroke has been described as "hemodynamic vertebral basilar insufficiency generated by forced or voluntary rotational head movements creating intermittent vertebral artery compression at the Atlanta-axial region."

Etiology and Pathogenesis
This syndrome, which is characterized by compression or stenosis of the VA, has been linked to a number of different causes in the literature. From its origin in the subclavian artery, the VA travels through the six foramina transvers aria of the cervical vertebrae, into the groove on the surface of the arch of the atlas, and finally into the dura mater. The vertebral arteries have a specific anatomical trajectory that makes them vulnerable to damage when the head is in motion. Some of the various causes and classifications of BHS are discussed.
As a first step, it's important to tell primary BHS apart from acquired BHS. Disc herniation, aberrant bony structures (such as osteophytes, idiopathic skeletal hyperostosis, and spondylotic alterations), ligaments, neck muscle hypertrophy, VA dissection, and other factors, like uncovertebral joint instability, are common contributors to the former. The most prevalent cause of BHS is osteophytes, often known as bone spurs. Injuries to the neck or head, including those sustained in sports-related accidents, car accidents, and operations to repair aneurysms, can lead to the development of cervical spondylosis and the subsequent development of acquired BHS.
According to the age of the patient at the time of the definitive diagnosis of BHS, the disorder can be further subdivided into pediatric BHS, juvenile BHS, and adult BHS. So far, this is absent from the scholarly record. A bony malformation at the atlantoaxial level or a sub-occipital bony protuberance impinging on the VA are two examples of congenital anomalies that can manifest in pediatric patients with BHS; in adolescents with BHS, an occipital condylar bone spur, an anomalous bypass of the VA, or an anomaly of the cervical spine may present instead. Similar to what was said about primary BHS, adult BHS also has a variety of potential causes. For the most part, adult patients are diagnosed with BHS, and the compressive reasons are place-specific. As a result, Cornelius *et al*. suggested categorizing BHS as either atlantoaxial, sub-axial, or a hybrid form.

Causes of atlantoaxial BHS include, but are not limited to, an ossified or thickened atlanto-occipital membrane, a dural fold in the foramen magnum, an assimilated posterior ring of the atlas, an accessory ossicle behind the atlanto-odontoid junction, erosive rheumatoid arthritis of C1-C2, C1-C2 facet hypertrophy Bony spurs of hypertrophied uncovertebral joints or lateral herniated discs frequently squeezed the ipsilateral VA in sub-axial BHS. Finally, a mixed form might be differentiated, as seen in the case reported by Kimura *et al*., which may involve bilateral VA stenosis (at C1-C2 and the subaxial level).

In a recent review of data from 126 individuals with BHS, Jost and Dailey found that the majority of occlusions (58%) occurred between C3 and C7, followed by C1-C2 (36%), and then only 6% occurred proximal to C7 and distal to C1. The occlusion of nondominant VAs also contributed to the development of BHS-typical symptoms. Except for one patient with an anomalous bypass at the VA who arrived with BHS and no changes in hemodynamic status, no case reports were found. Nonetheless, Rastogi *et al* data.'s reveals that the atlantoaxial (C1-C2) joint is significantly (p 0.0001) more frequently affected than any other level of VA blockage (99/142).

Repetitive shear stress causes endothelial damage, which contributes to the underlying pathophysiology of hemodynamic and/or thromboembolic dysfunction. Alternatively, the widely known mechanism behind hemodynamic BHS—blood flow stasis in the VA—may be responsible for thrombus development. Most cases of BHS are characterized by a compression of the dominant VA at the atlantoaxial or sub axial level, with the contralateral VA typically being hypo plastic, stenotic, or occlusive due to arteriosclerotic alterations. Since the left VA is dominant in half as many people as the right VA is, it stands to reason that the left VA would be engaged in a greater proportion of BHS patients. In 23 individuals, Husni and Storer found that the rotational blockage of a single VA brought on VBI. Twenty-two patients had non-existent or hypo plastic contralateral VAs, and a third had a constricted VA near its origin. However, the anterior cerebral circulation is unable to supply adequate collateral blood flow to the posterior cerebral circulation because the posterior communicating arteries are typically hypo plastic or aplastic. This artery, the posterior inferior cerebellar artery (PICA), may be the culprit in Bow Hunter's stroke because aplastic VAs frequently terminate in it. According to the findings of Darkhabani *et al*., 3 of 4 patients who were eventually treated with stenting had a dominant left VA and a hypo plastic or stenotic contralateral VA; in the other patient, VBI was caused by stenosis of the nondominant VA, which terminated in the PICA, causing vertiginous episodes with head movement related to ipsilateral PICA ischemia. Bilateral patent VAs have been observed to be mechanically occluded in some situations. All of these conditions can cause hemodynamic alterations in the posterior circulation system, which can exacerbate VBI symptoms and, in extreme cases, cause infarction of the posterior circulation.

The development of BHS into an ischemic stroke may be the result of thrombus formation or an artery-to-artery embolism related to VA dissection brought on by a bony spur or some other cause. Atherosclerotic embolism from artery to artery due to intimal and vascular damage from recurrent VA compression is a possible mechanism behind BHS, although the precise process is still unknown. The creation of a thrombus was a direct result of the activation of coagulation mediators and the promotion of platelet aggregation that resulted from repeated intimal injury and vascular damage. Some case reports corroborate this finding by describing individuals who experienced recurrent vertebrobasilar infarction and VA blockage on head rotation and also had a movable thrombus at the compression site.

Clinical Manifestation

- Adults can have a wide range of symptoms; however, some of the most prevalent are:
- Dizziness
- Fainting
- Impaired vision
- Tinnitus
- Nausea
- Horner’s syndrome
- Motor and sensory difficulties that occur with head rotation

Repeated mini-strokes (transient ischemic episodes) may also be reported or shown by affected individuals.

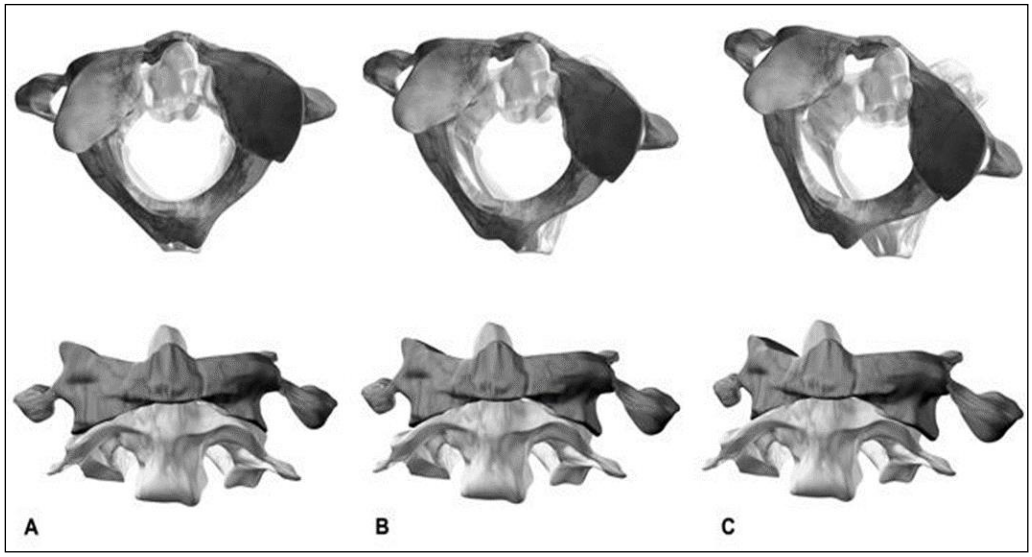

Fig 1: Superior (upper) and anterior (lower) views of motion at the atlantoaxial joint (A) in a neutral position, (B) with 10 degrees of rotation, and (C) with 20 degrees of rotation.

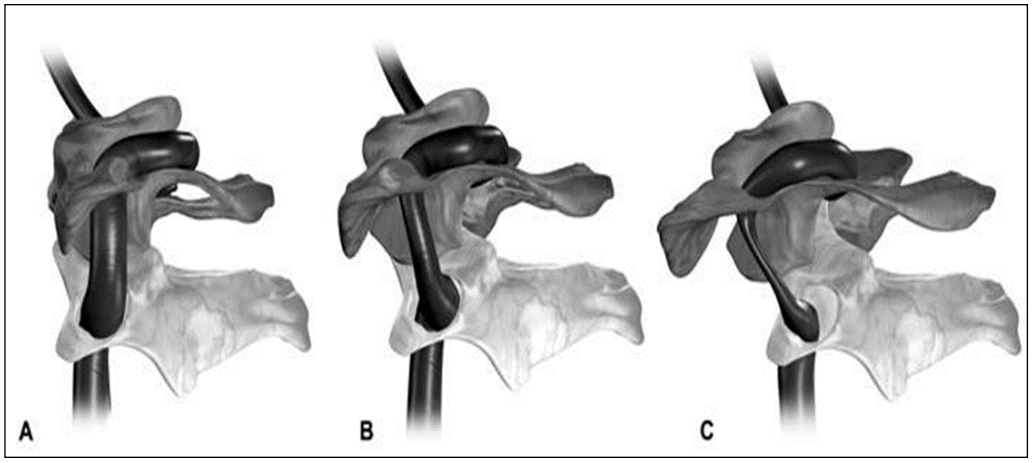

Fig 2: Lateral view of motion at the atlantoaxial joint (A) in a neutral position, (B) with 10 degrees of rotation, and (C) with 20 degrees of rotation. The vertebral artery narrows with progressive rotation.

Diagnosis

Imaging modalities such as CT/CTA, MRI/MRA, and neurosonography are frequently used in the evaluation and diagnosis of BHS because of their ability to detect aberrant bone structures, infarction lesions, and stenotic arteries, respectively. Digital subtraction angiography (DSA) is the gold standard for diagnosis because it clearly differentiates between patent and stenotic arteries. Figure 6. C1-C2 posterior cervical fusion. The trans articular screw and wiring technique is one of many that can be used to immobilize the C1-C2 joint.

Treatment and Outcomes

Anticoagulation therapy has been used for patients suffering from bow hunter's stroke, which is considered a conservative treatment option. The use of a cervical collar as a tangible reminder is another non-invasive method of preventing the patient from turning their head to the affected side. Many people refuse this treatment out of concern that their symptoms may recur or that they will lose consciousness.

Fusion and fixation at the C1-C2 level is the obvious surgical choice when rotation at that level is the root cause of the patient's problems (Fig. 6). Fusion of the C1 and C2 vertebrae has been linked to improvements in symptoms. However, up to 70% of neck rotation may be impaired by this method. Patients whose range of motion was severely restricted along one axis reported high levels of discomfort. The majority of patients who underwent surgery for Bow Hunter's stroke reported positive outcomes.

Surgery to relieve pressure on the vertebral artery is an alternative method of treatment. Using a posterior approach to the arch of C1, Shimizu *et al*. performed a hemi laminectomy to free the vertebral artery from its anchors in the transverse foramen and sulcus arteriosus. Following surgical intervention, patients showed no more complaints, and postoperative angiography revealed no rotational blockage at that level. Long-term symptom reduction may be achieved through surgical decompression of fibrous bands or osteophytic spurs, although there are no significant studies to back this up. Decompression of the atlantoaxial vertebral artery can also be achieved via the anterolateral approach.

Complication

Reduced vestibular function, syncope, dizziness, and permanent neurological impairments can arise from repeated transient ischemic episodes and strokes. Another danger is the possibility of injury from a fall that occurs as a result of one of these assaults. Since the atlantoaxial joint controls 55% of rotation and 15% of flexion and extension, people who need cervical fusion lose a lot of neck movement.

Conclusion

The long-term outcome of patients with this disease is not completely recognized due to the overall rarity of the disorder, which is reflected in epidemiological statistics about Bow Hunter's stroke. However, because the vertebral artery is mechanically obstructed during neck rotation in bow hunters, any surgery that relieves the compression and restores blood flow should greatly improve outcomes.

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