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New Treatment of Vertigo Caused by Jugular Bulb Abnormalities

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Abstract

Objective. Jugular bulb abnormalities can induce tinnitus, hearing loss, or vertigo. Vertigo can be very disabling and may need surgical treatments with risk of hearing loss, major bleeding or facial palsy. Hence, we have developed a new treatment for vertigo caused by jugular bulb anomalies, using an endovascular technique. *Patients*. Three patients presented with severe vertigos mostly induced by high venous pressure. One patient showed downbeat vertical nystagmus during the Valsalva maneuver. The temporal-bone computed tomography scan showed a high rising jugular bulb or a jugular bulb diverticulum with dehiscence and compression of the vestibular aqueduct in all cases. *Intervention*. We plugged the upper part of the bulb with coils, and we used a stent to maintain the coils and preserving the venous permeability. *Results*. After 12- to 24-month follow-up, those patients experienced no more vertigo, allowing return to work. The 3-month arteriographs showed good permeability of the sigmoid sinus and jugular bulb through the stent, with complete obstruction of the upper part of the bulb in all cases. *Conclusion*. Disabling vertigo induced by jugular bulb abnormalities can be effectively treated by an endovascular technique. This technique is minimally invasive with a probable greater benefit/risk ratio compare with surgery.

Keywords

neurotology, vertigo, jugular bulb, downbeat nystagmus, endovascular, stent, coils

Introduction

Jugular bulb abnormalities (JBA) are frequent anatomic variations and are estimated to occur in 10% to 15% of the population.[1] Two types of abnormalities are classically distinguished—high-riding jugular bulb (HRJB), defined as a jugular bulb reaching the level of the internal auditory canal, and jugular bulb diverticulum (JBD), which is an irregular outpouching projecting from the bulb. JBA leads to inner-ear erosion in 1.5% to 2.8% of cases.[1] The erosions are related to HRJB in almost all cases and are also associated with JBD in more than 50% of cases.[2] The range of symptomatic forms of JBA is unclear, but it could be involved in ~50% of cases when it is associated with inner-ear erosion[2]: Symptoms include tinnitus and/or hearing loss, and/or vertigo.[3,4] Intensity of symptoms is variable, but vertigo can be very disabling and may need surgical treatments, for example, jugular bulb lowering or posterior semicircular canal plugging.[5-7] These surgical techniques are efficient at reducing tinnitus, hearing loss, and vertigo, but require careful surgery to avoid damaging the jugular bulb wall, the inner ear, or the facial nerve. Major complications have occurred, such as hearing loss or major bleeds.[6,8]

Hence, we have developed a new treatment for vertigo caused by JBA using an endovascular technique. This technique allows complete removal of symptoms and no complications were associated with this treatment in our limited series.

Patients

Case 1

A 24-year-old workman was suffering from severe vertigo and headaches, particularly when lifting heavy loads or motorcycling. He also described frequent pulsatile tinnitus without hearing loss. A physical

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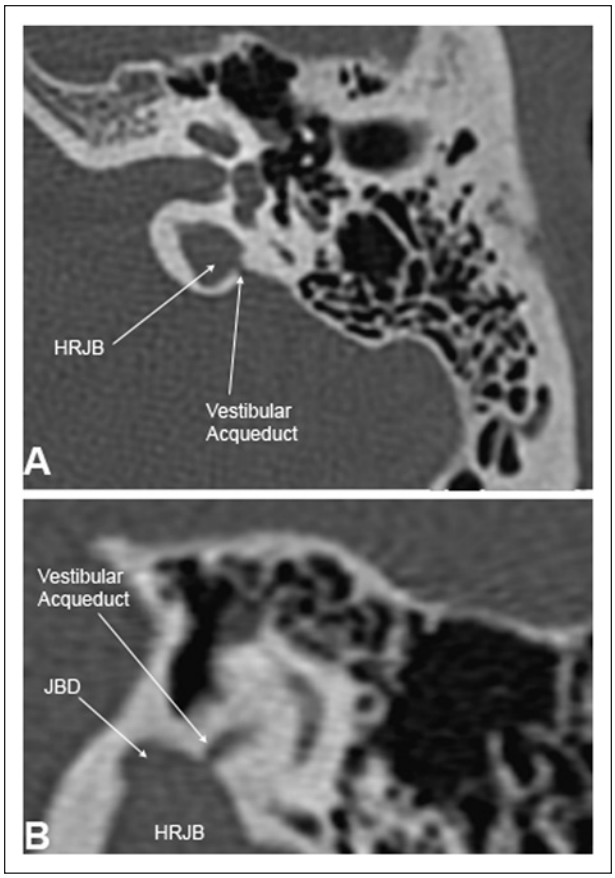

Figure 1. Patient 1: Left temporal computed tomography scan showing a high-riding jugular bulb (HRJB) with a diverticulum (JBD) and dehiscence of the vestibular aqueduct (A, axial view; B, coronal view).

examination distant from the vertigo crisis showed a blue retrotympanic mass rising from the left hypotympanum. There were no clinical vestibular symptoms and a video-nystagmography was normal, even after a Valsalva maneuver. The vestibular evocated myogenic potential thresholds were low in both sides (70 dB compared with normal superior to 90dB). A temporal-bone computed tomography (CT) scan showed an HRJB with JBD, and dehiscence of the left vestibular aqueduct (Figure 1). The patient was highly motivated to undergo treatment because of the severity of symptoms, which had resulted in him stopping work.

Case 2

A 31-year-old man was referred to our department after complaining of vertigo that occurred after strenuous effort or intensive laughter. Otoscopy and vestibular examinations were normal. Video-nystagmography, with caloric and rotational tests, was normal, but vertigo and downbeat vertical nystagmus appeared during the Valsalva maneuver (Figure 2, and supplemental video at http://sri.sagepub.com/). A CT scan showed HRJB with dehiscence of the right vestibular aqueduct (Figure 3). A magnetic resonance imaging (MRI) was considered normal.

Case 3

A 61-year-old man suffered from Menière-like syndrome with recurrent episodes of severe vertigo and nonpulsatile tinnitus. Otoscopy was normal. Audiometry testing revealed a right neurosensorineural hearing loss with average threshold at 70 dB. Video-nystagmography revealed a right hyporeflexia evaluated at 30% during caloric test.

Vestibular evocated myogenic potential thresholds were 75 dB on the right side compared with 95dB on the left. A CT scan showed JBD with dehiscence of the right vestibular aqueduct. An MRI confirmed the JBD with no associated anomaly.

Intervention

Our technique has evolved from the endovascular treatment of intracranial wide-necked aneurysms[9]: The vascular abnormality is plugged with coils and a stent avoids plugging the vessel's lumen (Figure 4F).

Prophylaxis with aspirin (250 mg daily) and clopidogrel (75 mg daily) were started 3 days before the embolization. In addition, heparin was given to achieve an activated clotting time of twice the baseline value recorded on the day before embolization.

The procedure was conducted under general anesthesia. Using a double venous femoral puncture, the left side is used to place coils and the right side for the stent.

Placement of Coils Catheter

A 6-F guiding catheter (Cordis, Miami Lakes, FL) was introduced into the left femoral vein and raised to the top of the jugular vein. A 1.4-F microcatheter (Tracker excel 14 microcatheter, Stryker Neurovascular, Kalamazoo, MI) was then placed into the diverticulum. This catheter was used later to plug the diverticulum with coils (Figure 4A).

Placement of the Stent

A 125-cm long 4-F catheter (Terumo Corporation, Tokyo, Japan) was placed in the right femoral vein and inserted to just above the jugular bulb or the diverticulum. This catheter was used as a guide to insert an 8-F guiding catheter (Cordis, Miami Lakes, FL, USA), which was more difficult to navigate inside the jugular bulb. For the same reason, a 300-cm long microguide

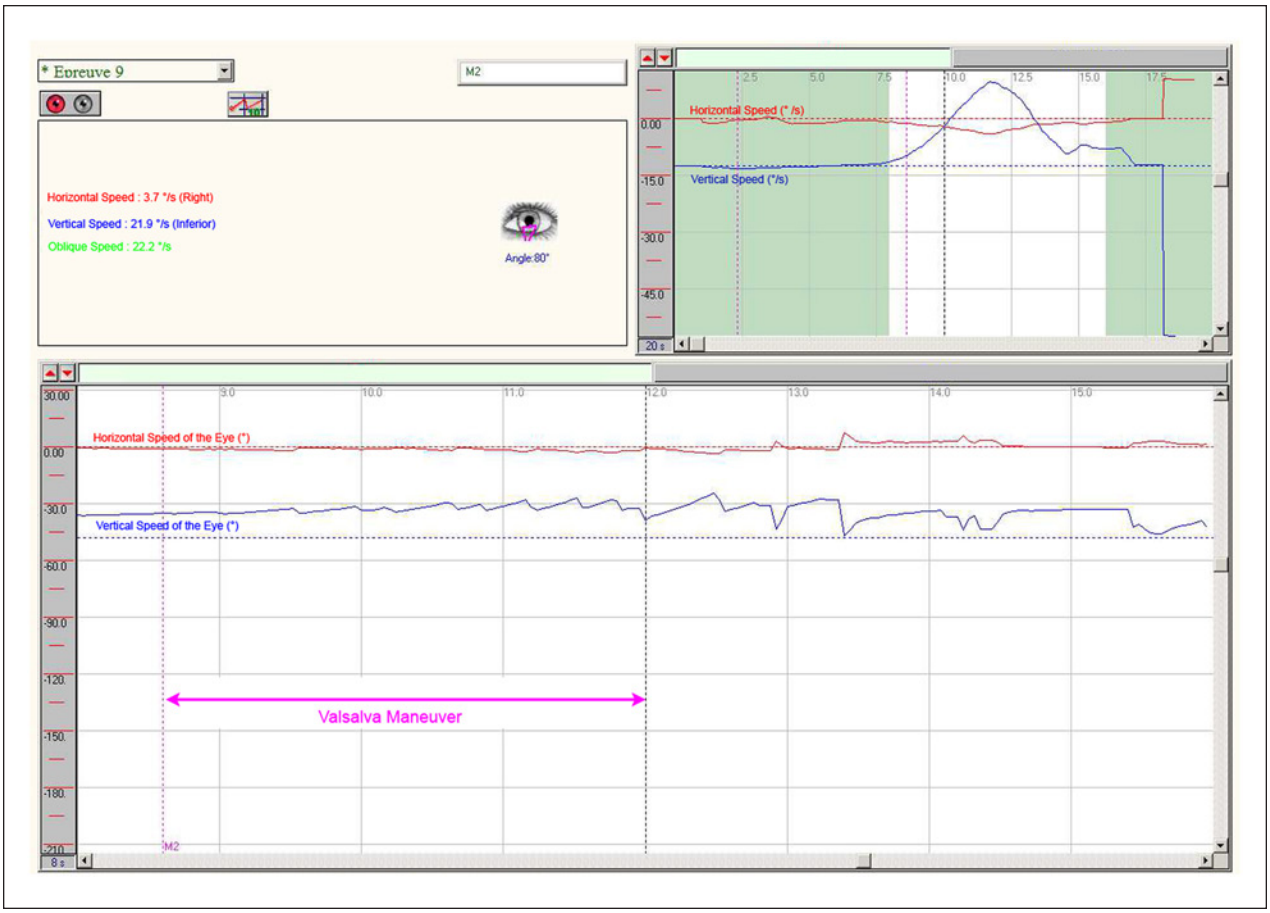

Figure 2. Patient 2: Video-nystagmography with the Valsalva maneuver to induce vertigo and downbeat nystagmus. (Supplemental video available online at http://sri.sagepub.com/)

(Transend guidewire, Stryker Neurovascular, Kalamazoo, MI) was placed inside the 8-F guide up to the sigmoid sinus (Figure 4B).

The microcatheter was used to deploy a stent in the jugular bulb when the 8-F was placed at the lower part of the dehiscence (Figure 4C). We used the same type of stent in both patients (Carotid Wallstent Monorail Endoprosthesis, Stryker Neurovascular, Kalamazoo, MI, 9 × 40 mm).

Coil Embolization

The 1.4-F microcatheter was held in place in the JBA by the stent (Figure 4D). It was then used to embolize the HRJB or JBD (Figures 4E and 5). We used 8 coils (GDC detachable coil, Stryker Neurovascular, Limerick, Ireland) in case 1 and 18 coils in case 2 (10 coils GDC detachable coil, Stryker Neurovascular and 8 coils Deltapaq 10 Stretch-Resistant, Micrus Endovascular, San Jose, CA; Figure 4F).

Anticoagulation, using heparin, was pursued for 24 hours after embolization, then switched to aspirin (250 mg/d) and clopidogrel (75 mg/d) for 3 months. The patients were discharged home on day 2.

Results

Case 1: Since treatment and after a 24-month follow-up, this patient experienced no more vertigo or headaches, allowing return to work. Tinnitus has become negligible and his hearing has remained normal. The retrotympanic blue mass had disappeared at a later otoscopy.

Case 2: After 14 months of follow-up, no recurrence of vertigo was observed, even during Valsalva maneuvers.

Case 3: No recurrence of vertigo was observed after 12 months of follow-up. Audiometry testing showed a 10 dB improvement and tinnitus remained stable.

The 3-month arteriographs showed good permeability of the sigmoid sinus and jugular bulb through the stent, with complete obstruction of the JBA in all cases.

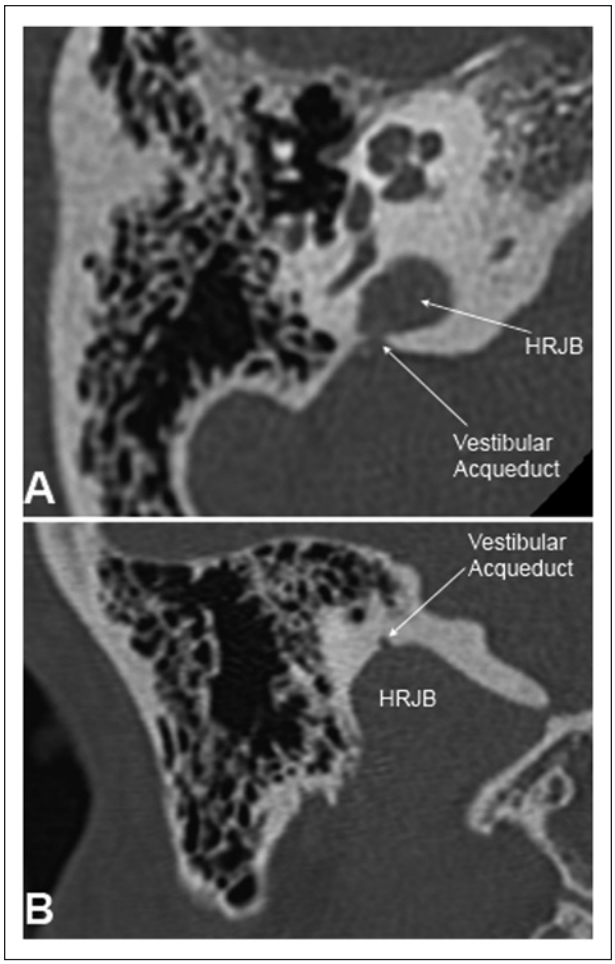

Figure 3. Patient 2: Right temporal computed tomography scan showing a high-riding jugular bulb (HRJB) and dehiscence of the vestibular aqueduct (A, axial view; B, coronal view).

Discussion

The jugular bulb only appears after the age of 2 years.[10,11] Its growth is probably induced by hemodynamic factors, such as a shift from a fetal to a postnatal circulation, along with the acquisition of an erect posture. Hemodynamic factors also explain why the jugular bulb is predominant on the right side in 75% of individuals because of the venous pressure that comes more directly from the right atrium.[3,10] JBA is associated with inner-ear dehiscence in 3% of cases. This dehiscence involves the vestibular aqueduct in 93% of cases, rather than the facial nerve (5%) or the posterior semicircular canal (2%).[1] In another study, Friedmann et al[2] found that dehiscence of the vestibular aqueduct during JBA was symptomatic in two thirds of cases, and more frequently resulted in hearing loss (16/24 cases) rather than vertigo (2/24 cases).

A diagnosis of vertigo caused by JBA should be suspected when vertigo is induced by high venous pressure when carrying out activities such as lifting a heavy load, coughing, or during the Valsalva maneuver. To our knowledge, no nystagmus induced by Valsalva maneuver has ever been described in case of JBA. But it has been described in superior canal dehiscence showing an upbeat vertical nystagmus corresponding to an inhibition of this canal.[12] The downbeat nystagmus observed in our patient 2 is probably due to an inhibition of the posterior canal as the nystagmus beat downward and the posterior canal is very close to the JBA, even if no dehiscence of this canal was found in the CT scan.[13] JBA may also present sound-induced vertigo (Tullio phenomenon) via a third window mechanism.[14] Associated signs, such as pulsatile tinnitus, or conductive or neurosensory hearing loss, can also indicate JBA.[3]

Nonpulsatile tinnitus presented by our patient 3 is a common symptom in Menière-like syndrome. The prognosis of those tinnitus is bad because tinnitus is probably generated by the brain rather than the inner ear itself.[15]

Vestibular tests in JBA should include a video-nystagmography with the Valsalva maneuver to assess induced nystagmus. Vestibular evocated myogenic potentials may also indicate lower threshold.[2,16]

Vertigo from JBA requires a temporal-bone CT scan to visualize the morphology of the JBA, inner ear dehiscence, and to rule out major differential diagnoses, such as superior semicircular canal dehiscence. An MRI is useful in ruling out other causes of vertigo but cannot pinpoint a diagnosis of JBA[2] (Table 1).

The treatment of JBA-induced vertigo has been rarely described until now. Couloigner et al[6] reported 13 cases of surgically treated JBA using mastoidectomy and decompression of the vestibular aqueduct by impacting JBA with bone wax.[5] However, this surgery remains risky because of the proximity of JBA with the facial nerve, the dura mater, and the inner ear. Moreover, the jugular bulb wall is very fragile. Any damage can lead to severe bleeding or wax embolism. Moreover, the maximal result from this surgery only occurs after several months as patients often initially present with dizziness and positional vertigo. In this series, vertigo disappeared in 7 patients (54%), decreased in intensity in 5 (38%), and remained disabling for 1 patient.[6] Hearing decreased in 3 of the 13 cases.[6]

In contrast, our endovascular technique is minimally invasive. Compared with classical surgical techniques, our procedure does not risk facial palsy or tearing of the dura mater.

The major theoretical risk of our technique is thrombosis of the stent: However, the prevalence of this is hard to estimate because venous stenting is a rare procedure. Intracranial venous stenting has been used in the transverse sinus as a treatment for idiopathic intracranial hypertension. Ninety-two cases of this latter indication

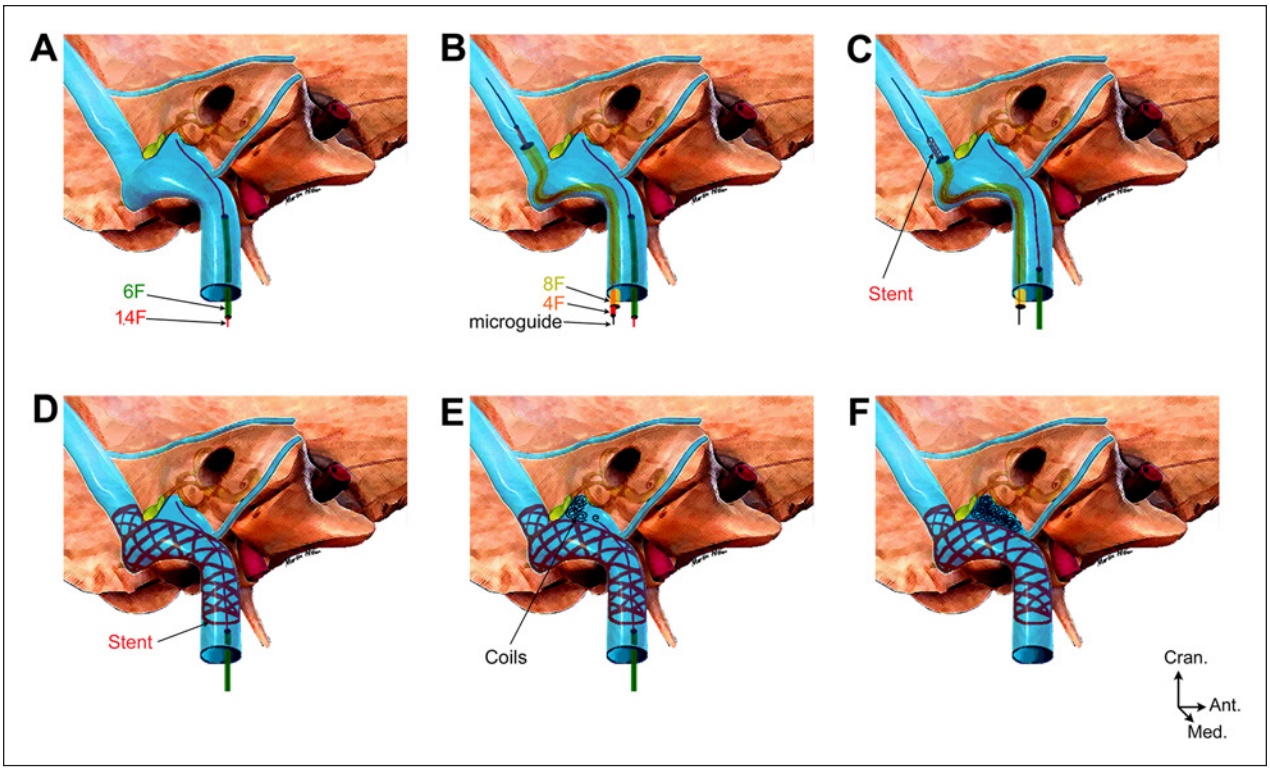

Figure 4. Endovascular treatment of symptomatic jugular bulb abnormalities (JBA): Medial view of left temporal bone. (A) The 6-F guiding catheter is raised to the top of the jugular vein with a 14-F microcatheter placed into the JBA. (B) The 4-F catheter is used as a guide to raise an 8-F guiding catheter above the jugular bulb, and a microguide is placed inside the 8-F guide up to the sigmoid sinus. (C) Deployment of the stent. (D) The 14-F microcatheter is kept in place by the stent in the JBA. (E) Embolization of the JBA by the coils (see also Figure 5). (F) Complete embolization of the JBA with the stent maintaining venous permeability.

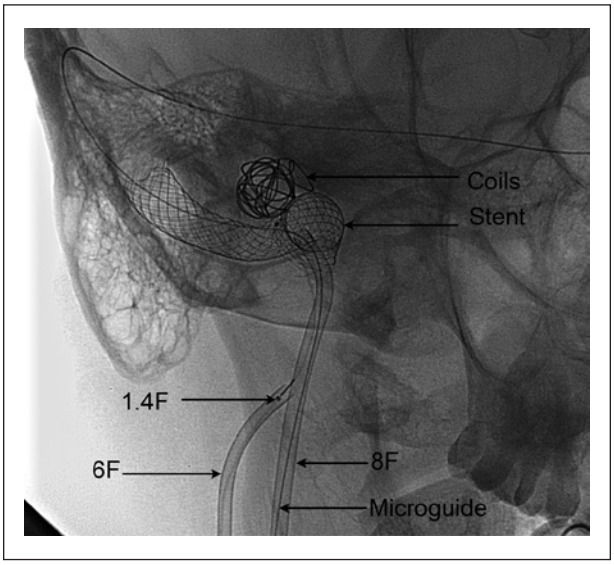

Figure 5. Arteriography showing the embolization of the jugular bulb abnormalities (JBA) by the coils (corresponding to Figure 4E): Patient 2.

have been reported: Complications were transient headaches, transient hearing loss in 3 cases, and stent thrombosis in 2 cases.[17,18] The thromboses were treated by thrombolysis. But no thrombosis was reported in 52 cases using prophilaxis with enteric-coated acetylsalicylic acid (150 mg daily) and clopidogrel (75 mg daily) for 1 week before a stent placement.[18]

The use of a stent in our technique allows the sinus to remain permeable and avoids the risk of idiopathic intracranial hypertension. This technique could therefore be used in cases of contralateral sinus or jugular hypoplasia.[4] Because this technique is new and because of local methodologies, we used angiography at 3 months. Nevertheless, it is also possible to use venous MRI angiography to check the permeability of the stent.[19]

Conclusion

Endovascular treatment of JBA-induced vertigo is minimally invasive and effective for vertigo and pulsatile tinnitus. Its validity to treat hearing loss needs further evaluation.

Table 1. Criteria of Diagnosis and Treatment of Vertigo Caused by Jugular Bulb Abnormalities (JBA).

		Indicators for JBA
Clinical symptoms	Vertigo Tinnitus Hearing loss: conductive or neurosensory Retrotympanic blue mass	Triggered by high venous pressure Pulsatile Tullio phenomenon : Vertigo induce by sound
Diagnosis confirmation	Video-nystagmograph with Valsalva maneuver Vestibular evocated myogenic potential CT scan MRI	Valsalva induced nystagmus Low threshold JBA with erosion of the vestibular aqueduct or posterior semi-circular canal No other cause of vertigo
Treatment	Stent and coil embolization, plus aspirin (250 mg daily) and clopidogrel (75 mg daily) for 3 months	
Follow-up	Angio MRI at 3 months and at 1 year with venous sequences.	

JBA, jugular-bulb anomalies.

Authors' Note

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Declaration of Conflicting Interests

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