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ORIGINAL ARTICLE

The Prevalence of Vascular Impingement of the Trigeminal, Facial, and Vestibulocochlear Nerves in Healthy Volunteers

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ABSTRACT

Aim: *To determine the prevalence of vascular impingement of the trigeminal, facial, and vestibulocochlear nerves in a healthy population.*

Subjects and Methods: *The trigeminal, facial, and vestibulocochlear nerves of 90 asymptomatic volunteers were assessed for the presence or absence of vessels abutting or displacing these nerves. The studies were performed on a 1.0-T magnet using a circular polarised head coil. Axial T2-weighted images, 3-dimensional constructive interference in steady state (TR/TE=12.3/5.9), through the brainstem were obtained at 0.7 mm intervals with a matrix of 192 x 512 and a 25 x 16 field of view.*

Results: *Forty two of 540 nerves (8%) showed evidence of impingement, 108 of 540 nerves (20%) showed evidence of abutment, and 390 of 540 nerves (72%) were normal. Among the 42 impinged nerves, 38 (90.5%) were facial/vestibulocochlear complex; 4 (9.5%) were trigeminal, and 1 (2.0%) had all 3 nerves impinged. No bilateral impingements were detected. Of the 108 volunteers with vascular abutment of the nerves, 101 nerves (93.5%) were facial/vestibulocochlear complex; 7 (6.5%) were trigeminal, and 3 (2.7%) had abutment of all 3 vessels.*

Conclusion: *Vascular impingement of the trigeminal, facial, and vestibulocochlear nerves is frequently seen in the healthy population.*

Key Words: *Cranial nerve diseases, Magnetic resonance imaging*

INTRODUCTION

Publications during the past decade suggest that a number of neurological syndromes characterised by paroxysms of motor or sensory activity may be caused by compression of the cranial nerves by adjacent blood vessels. This was followed by the demonstration that surgical microvascular decompression of these nerves is a safe and effective treatment for these syndromes when they fail to respond to conservative therapy.[1]

Vascular impingement of the trigeminal, facial, and vestibulocochlear nerves has been associated with several clinical conditions such as trigeminal neuralgia, hemifacial spasm (HFS), and benign paroxysmal vertigo, respectively. However, vessels related to these cranial nerves are frequently incidentally noted intra-operatively by skull base surgeons and in asymptomatic patients when high resolution magnetic resonance imaging (MRI) of the brainstem is performed for other reasons.[1,2]

The aim of this study was to determine the prevalence of vascular impingement of the trigeminal, facial, and vestibulocochlear nerves in the healthy population.

SUBJECTS AND METHODS

The study population consisted of 90 asymptomatic volunteers (58 men and 32 women). All volunteers had normal blood pressure and none had any neurological diseases or cranial neuropathy. The ages of the volunteers varied between 18 and 74 years (average, 43 years).

The trigeminal, facial, and vestibulocochlear nerves of all the volunteers (540 nerves) were assessed. The root exit zone (REZ), cisternal, and intracanalicular segments of these 3 nerves were evaluated for the presence or

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absence of vascular abutment or impingement. Identification of the name of the offending vessel and the site of vascular contact were not attempted.

The studies were performed on a 1.0-T magnet (Magnetome Expert, Siemens Medical Systems, Erlangen, Germany) using a circular polarised head coil. Axial T2-weighted images, 3-dimensional constructive interference in steady state (CISS; TR/TE=12.3/5.9), through the brainstem were obtained at 0.7 mm intervals with a matrix of 192 x 512 and a 25 x 16 field of view.

Nerves were considered to be impinged when a vascular loop was visualised to displace and distort the nerve course. However, if a vascular loop was noted to be touching but not displacing the nerve, then it would be classified as nerve abutment. Healthy subjects did not reveal any vessels in the immediate vicinity of the cranial nerves.

RESULTS

Among the 540 examined nerves, 42 (8%) showed evidence of impingement, 108 (20%) had evidence of abutment, and 390 (72%) were healthy. Among the impinged nerves, 38 nerves (90.5%) from 38 volunteers were facial/vestibulocochlear complex, 4 nerves (9.5%) from 4 volunteers were trigeminal, and 1 (2%) had all 3 nerves impinged. No bilateral impingements were detected.

Of the 108 nerves with vascular abutment, 101 nerves (93.5%) from 89 volunteers were facial/vestibulocochlear complex, 7 nerves (6.5%) from 5 volunteers were trigeminal, and 3 (2.7%) had abutment of all 3 nerves (Figures 1 and 2).

DISCUSSION

Cranial neuropathy could be due to many causes such as mass effect by adjacent vessels or vascular aneurysms or tumours at the cerebellopontine angle. Inflammatory and demyelinating processes such as multiple sclerosis can also result in a similar clinical picture. Vascular impingement of cranial nerves has become a well recognised concept in the pathogenesis of cranial neuropathies, particularly in relation to the trigeminal, facial and vestibulocochlear nerves.[2-8]

Trigeminal neuralgia (tic douloureux) is characterised by short episodes of intense and occasionally disabling facial pain radiating down the jaw. These episodes can occur spontaneously or may be triggered

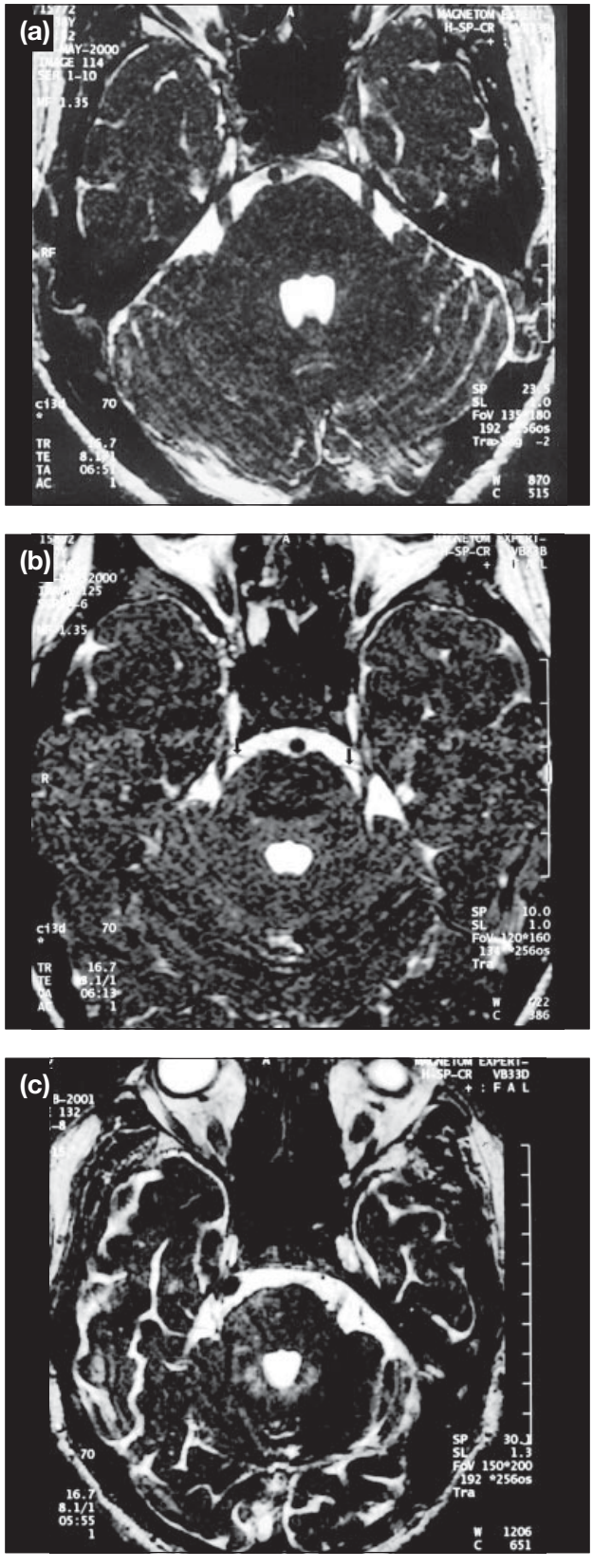
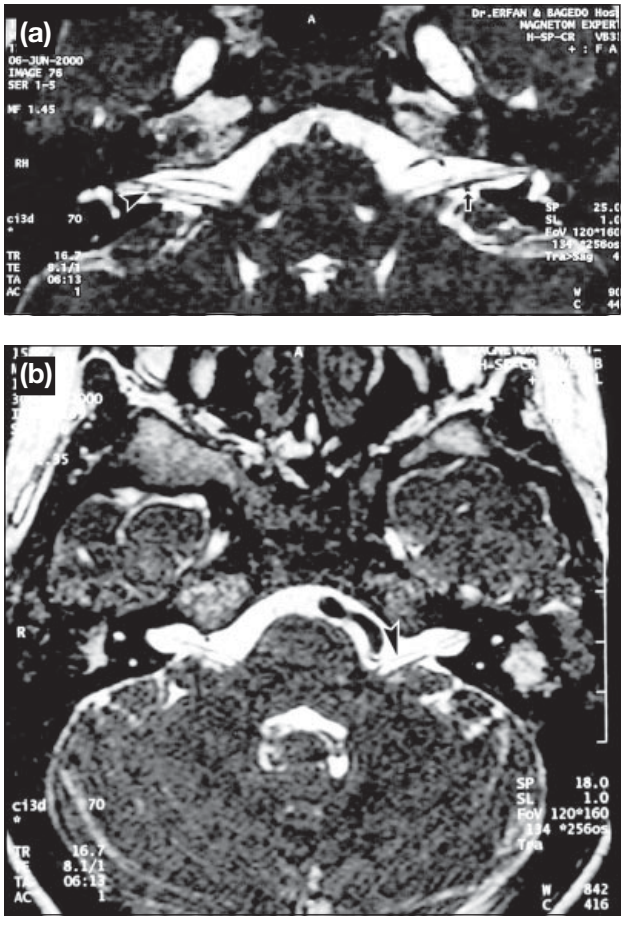


Figure 1. Axial 3-dimensional constructive interference in steady state magnetic resonance study at the mid-pontine level shows (a) normal trigeminal nerves; (b) vessels abutting the trigeminal nerves bilaterally (arrows); and (c) impingement of the right trigeminal nerve by the basilar artery.



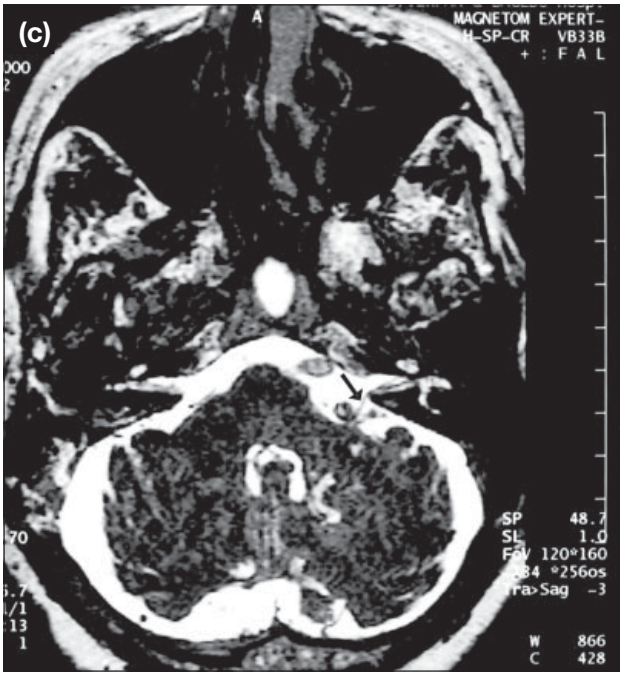


Figure 2. Axial 3-dimensional constructive interference in steady state magnetic resonance study at the internal auditory canal level shows (a) normal left facial nerve and a vessel abutting the left superior vestibular nerve posteriorly (arrow) and a vessel abutting both the right facial and superior vestibular nerves (arrowhead); (b) the basilar artery is seen abutting the root exit zone of the left facial nerve (arrowhead indicates left facial nerve); and (c) the left vertebral artery is impinging on the root exit zone of the left vestibulocochlear nerve (arrow indicates left vestibulocochlear nerve).

by light touch, chewing, or changes in temperature. Frequently, however, routine imaging of the brain does not reveal the underlying aetiology. In such cases, a small vessel is often found to be compressing the trigeminal nerve.[2-4]

HFS is clinically divided into 2 categories of typical and atypical. Typical HFS represents approximately 90% of all patients with HFS. Patients in this category present with spasmodic facial muscle contractions that start in the upper face and spread downward to involve the rest of the face. Patients with atypical HFS represent approximately 8% of patients with HFS and present with spasmodic contractions that start in the lower half of the face and progress upward. Bilateral HFS symptoms are seen in less than 2% of all HFS patients.[5,6]

Benign paroxysmal positional vertigo is an inner ear problem that results in short but severe room-spinning vertigo. This condition usually develops following trauma or a severe cold, but can also develop as part of the ageing process. The condition is sudden in onset and usually first noticed in bed, when waking from sleep. Any turn of the head seems to bring on violent but brief bursts of dizziness. For many patients, nausea and vomiting also accompany the vertigo. Patients often describe the occurrence of the vertigo with tilting of the head, looking up or down, or rolling over in bed. There is no new hearing loss or ringing noise associated with these attacks, which helps to distinguish it from other inner ear conditions.[9,10]

Although several theories have been proposed to explain the aetiology of these cranial neuropathies, most of the reported literature concentrates on the anatomic relation between the nerves and the juxtaposed vascular loops. More specifically whether the vascular loop is related to the REZ of the nerve. Others have described symptoms in patients with vascular loops related to the other segments of the nerves, away from the REZ.[6,11,12]

Vessels are often noted incidentally anywhere along the course of cranial nerves in asymptomatic people. Therefore, it is hypothesised that vascular impingement could be an incidental finding and that the degree, rather than the location, of impingement on the nerve probably plays a major role in the initiation of symptoms.

Most of the reported literature is concerned with the relationship between patients' symptoms and the identification of the offending vessels. In contrast, the aim of this study is to emphasise the prevalence of incidental vascular impingement and abutment in healthy volunteers. Although this topic is often discussed, however, it is not well documented in the English language literature.

A strong correlation has been observed between vascular impingement and nerve displacement, with vessels abutting and impinging the facial nerve noted in healthy volunteers (2 and 5 of 10 people respectively).

All the people in this study were healthy adult volunteers. All subjects underwent a detailed physical and neurological examination to rule out other clinical entities that may simulate these conditions such as blephrospasm, facial myokymia, chronic facial pain, migraine, and middle ear diseases. The data from this study indicate that vascular abutment and impingement on the cranial nerves is a frequent finding in the general asymptomatic population and that its relationship to cranial neuropathy should be cautiously interpreted. This is particularly important since arterial tortuosity may be related to the patient's age and arteriosclerosis.

Jannetta[9] and McLaughlin et al[11] reviewed the experience learned at the University of Pittsburgh Medical Center after 4400 microvascular decompression surgeries for various cranial nerves and documented the efficacy of this procedure. However, the article emphasised that the compression usually occurs at the REZ and did not evaluate the degree of nerve impingement or whether there was any nerve displacement. This series contained variable percentages of patients who did not benefit from the microvascular decompression surgery. This may raise some doubts about the absolute relationship between patients' symptomatology and the radiological observation of adjacent vessels.

Ryu et al reported 7 patients in whom both typical and atypical HFS did not occur from vascular compression at the REZ but from compression of the distal portion of the facial nerve.[12] Although Jannetta argued their findings, neither party evaluated the degree of nerve displacement, however.[13]

The use of axial and/or coronal thin section MRI techniques (CISS and 3-dimensional fast spin-echo) in the evaluation of HFS is not new and has been utilised before.[7,14] It is considered superior to conventional MRI techniques, since it allows visualisation of small cerebellopontine angle vessels that may offend the cranial nerves that are otherwise difficult to visualise. Similarly the thin sections obtained can be reconstructed in any of the 3 orthogonal planes to further illustrate the offending vessels. However, such reconstruction techniques were not used in this study.[14]

Both Jannetta[9] and Girard et al[7] documented that the vessels most commonly found to compress the facial nerve are the posterior inferior cerebellar artery followed by the anterior inferior cerebellar artery and the vertebral artery in descending order. Although it was not a primary concern in this study to identify the offending vessels, similar vascular distribution for the facial and vestibulocochlear nerves were observed. As far as the trigeminal nerve is concerned, compression is usually due to either the vertebral or the basilar artery. This study agrees with Girard et al in that the identification of neurovascular contact does not necessarily correlate with patients' symptoms.[7]

This study demonstrates that vascular impingement and abutment of the trigeminal, facial, and vestibulocochlear nerves are frequent incidental findings and that their significance has to be interpreted in light of the patients' symptomatology.

In conclusion, vascular impingement of the trigeminal, facial, and vestibulocochlear nerves is frequently seen in the healthy population. However, more specific prevalence of vascular impingement of cranial nerves in various age groups needs further study with a larger population in each age group.

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