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ORIGINAL ARTICLE

MRI of the vestibular nerve after selective vestibular neurectomy

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Abstract

Conclusion. In patients with Ménière's disease and persisting vertigo attacks after vestibular neurectomy (VNx) MRI of the vestibulocochlear nerve can identify residual vestibular nerve fibres that could be responsible for the vertigo attacks. *Objective*. To test if MRI of the vestibulocochlear nerve can corroborate the presence of residual vestibular nerve fibres in patients with persisting vertigo attacks and residual vestibular function after VNx. *Materials and methods*. Vestibulocochlear nerve bundles of seven post-VNx unilateral Ménière's patients were imaged using 1.5 Tesla MRI with steady state free precession (SSFP) sequences. Reformatted MR images orthogonal to the vestibulocochlear nerve axis in internal auditory canal were compared on the VNx and intact sides. Vestibular function was assessed with caloric tests, three-dimensional head impulse tests and vestibular evoked myogenic potentials. Of the seven patients only one was asymptomatic (totally free of vertigo); six had continued to experience vertigo attacks, albeit not as long or as severe as before VNx. *Results*. On the VNx side, MRI showed intact facial and cochlear nerves in all seven patients. In the six symptomatic patients, although superior and inferior vestibular nerve bulk and signal were reduced, residual bulk suggestive of inferior vestibular nerve was evident, correlating with evidence of residual posterior canal function on impulsive testing in all six symptomatic patients. In the asymptomatic patient, superior and inferior vestibular nerves were absent on MRI and impulsive testing revealed no residual posterior canal function.

Keywords: *Vestibular nerve, vestibular neurectomy, MRI, Ménière's disease*

Introduction

After selective vestibular neurectomy (VNx) – i.e. VNx in which hearing is successfully preserved [1] – for intractable vertigo from unilateral Ménière's disease [2] some patients continue to report vertigo attacks, albeit of lesser frequency and severity [3,4]. The question then arises: are these vertigo attacks due to the patient having developed Ménière's disease of the unoperated ear (this occurs in about 14% of Meniere's patients [5]), or could the attacks, despite the VNx, be arising from the operated ear because the VNx was incomplete [3,4]? Although caloric testing shows no residual lateral semicircular canal (SCC) function on the operated side in these patients [3], three-dimensional head impulses, a test not generally available, reveal residual vestibular function predominantly in the posterior SCC [4,6] probably due to incomplete neurectomy of the inferior vestibular nerve. Our aim was to determine whether high resolution MRI of the vestibulocochlear nerve, a test that is generally available, might also reveal residual vestibular nerve fibres, and indicate that the persisting vertigo attacks after VNx could be still arising from the operated ear due to residual vestibular nerve function, i.e. the VNx was incomplete [4].

Materials and methods

Patients

Seven patients with unilateral Ménière's disease, aged 39–72 years, who had undergone retrosigmoid VNx (three different surgeons) 2–17 years previously, underwent MRI of the vestibulo-cochlear nerve as part of their medical assessment. Six of the seven patients continued to have persistent vertigo

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attacks, albeit of lesser frequency and severity than before VNx. Hearing on the operated side was within 20 dB of the preoperative levels at all frequencies and none of the patients reported tinnitus or aural fullness and none had any low-frequency sensorineural hearing loss in the unoperated ear.

MRI

High resolution MRI was performed with a 1.5 Tesla MR Unit (GE Medical Systems, Milwaukee, USA) using the GE implementation of steady state free precession (SSFP), known as 3D-FIESTA (fast imaging employing steady-state acquisition). T2/T1-weighted axial images were generated in 0.8 mm slices, overlapped by 0.4 mm, using a small field of view (9.6 cm) and a high resolution matrix (512 × 512). No intravenous contrast was required. The SSFP sequence is a fully balanced steady-state coherent imaging pulse sequence designed to produce high signal-to-noise ratio and intrinsic spatial resolution images using relatively short acquisition times (~4.5 min). Curved reformatted images were generated along the course of the vestibulocochlear nerve in the internal auditory canal (IAC) (Figure 1A) using an Advantage Workstation 4.1 (GE Medical Systems) and a set of images orthogonal to the nerves at each point was also created. MR images orthogonal to the vestibulo-cochlear nerve axis in the IAC on the VNx side were compared to images on the intact side in the same patient (Figure 1B). For ease of comparison, the MR images on the intact side were reflected.

Vestibular function tests

All patients had standard bithermal caloric testing plus stimulation of the operated ear with 50 ml of water at 0°C. The head impulse test was used to determine individual SCC function by measuring head and (left) eye rotations in three dimensions with dual-search coils (Skalar, Delft, The Netherlands) in response to manually delivered head impulses in lateral, left anterior-right posterior, or right anterior-left posterior SCC canal planes [4,6]. Three-dimensional head and eye positions were computed from the search coil signals in rotation vectors. The coordinate reference frame was rotated by 45° to re-express those vectors as rotations about the (approximate) preferred axis of individual SCCs and to display as angular vestibulo-ocular reflexes (VORs) from lateral, anterior, posterior SCCs for VNx and intact sides [4,6]. For ease of comparison with head velocity, compensatory eye velocity was inverted. The VOR gain was determined for each trial as the ratio of the slope of a line fitted to the eye velocity compared with the slope of a line fitted to the head velocity during a 40 ms period starting 50 ms after head impulse onset [4]. Standard VEMP to 110 dB NHL

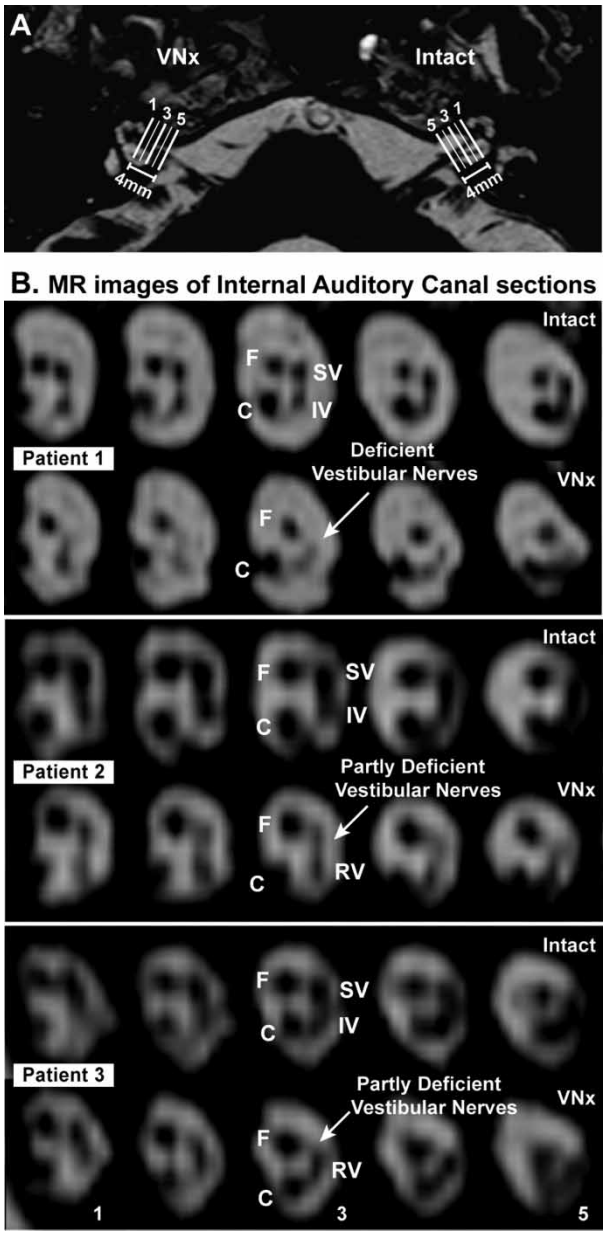

Figure 1. (A) Axial high resolution MR image curved reformatted along the vestibulocochlear nerve pathway in the IAC on the VNx and intact sides. (B) Orthogonal MR images reformatted along the vestibulocochlear nerve axis in the IAC in five 0.8 mm contiguous slices in Ménière's Patients 1, 2 and 3. Sections from the intact side have been reflected for ease of comparison between the VNx and intact sides. On the intact side, the facial (F), cochlear (C), superior vestibular (SV) and inferior vestibular (IV) nerves were observed. In Patient 1 who was asymptomatic post-VNx, only the facial and cochlear nerves are present on the VNx side with superior and inferior vestibular nerves completely absent. In symptomatic Patient 2, the superior and inferior vestibular nerve appeared to have deficient bulk and signal, with some residual vestibular (RV) nerve bulk still present. In symptomatic Patient 3, the superior vestibular nerve was absent, while the inferior vestibular nerve appeared almost intact with similar bulk and signal to the intact side.

air-conducted clicks to determine saccular function was recorded in all patients from the sternomastoid muscle as an averaged p13-n23 response waveform [7–9]. Individual SCC and saccular function of each patient was correlated with MRI of the vestibulocochlear nerves from the VNx and intact sides. We have previously reported the head impulse data from six of the seven patients [4].

Results

MRI of the vestibulocochlear nerve post-VNx

Reformatted MR images orthogonal to the vestibulocochlear nerve axis in the IAC from the Ménière's disease patients post-VNx show differences in the superior and inferior vestibular nerve bulk and signal between the VNx and intact sides (Figure 1). On the intact side in all patients, the facial and vestibulocochlear nerves were observed near the internal auditory meatus (Figure 1B, slice 5). The vestibulocochlear nerve divided into superior and inferior vestibular and cochlear nerves as it traversed the IAC to innervate the labyrinth and cochlea (Figure 1B, slices 1, 2, 3). The superior and inferior vestibular nerves can be two distinct nerves as in Patient 1, or can be close together to appear as dumbbell shaped as in Patients 2 and 3. On the VNx side in asymptomatic Patient 1, absence of the superior and inferior vestibular nerve bulk and signal was observed on the MR images, suggesting complete vestibular neurectomy (Figure 1B, slices 2, 3). In symptomatic Patient 2, reduced superior and inferior vestibular nerve bulk and signal were observed on the VNx side. In symptomatic Patient 3, MR images are suggestive of absence of superior vestibular nerve with residual preserved inferior vestibular nerve bulk and signal (Figure 1B, Patient 2, slices 2 and 3). There is also some displacement of the residual vestibular nerve bulk post-VNx (Figure 1B, Patient 3, slices 2 and 3). The other three symptomatic post-VNx Ménière's disease patients also showed similar residual vestibular nerve bulk and reduced signal on the VNx side.

Residual vestibular function post-vestibular neurectomy

We have previously reported the head impulse data from six of the seven patients [4]. Head impulse tests showed that in all post-VNx Ménière's disease patients SCC function on the intact side is normal, i.e. compensatory eye velocity of the angular VOR was almost equal to head velocity. However, if unilateral vestibular deafferentation is complete (e.g. following total vestibulocochlear neurectomy for vestibular schwannoma), because of the 'push-pull' behavior of the SCC the VOR gain on the intact side is 10–15% lower than normal due to absence of disinhibition from the deafferented side, illustrated in Patient 1 (Figure 2). In the six patients with Ménière's disease who were still symptomatic, the mean VOR gains on the intact side (Figure 2, e.g. Patients 2 and 3) were slightly higher: lateral SCC was 0.95 ± 0.03; anterior SCC was 0.88 ± 0.03; posterior SCC was 0.78 ± 0.02 (mean ± 1 SEM), probably because of disinhibition from the incomplete deafferentation of the operated side. On the VNx side when the vestibular deafferentation was complete, the VOR gains were 0.2 or

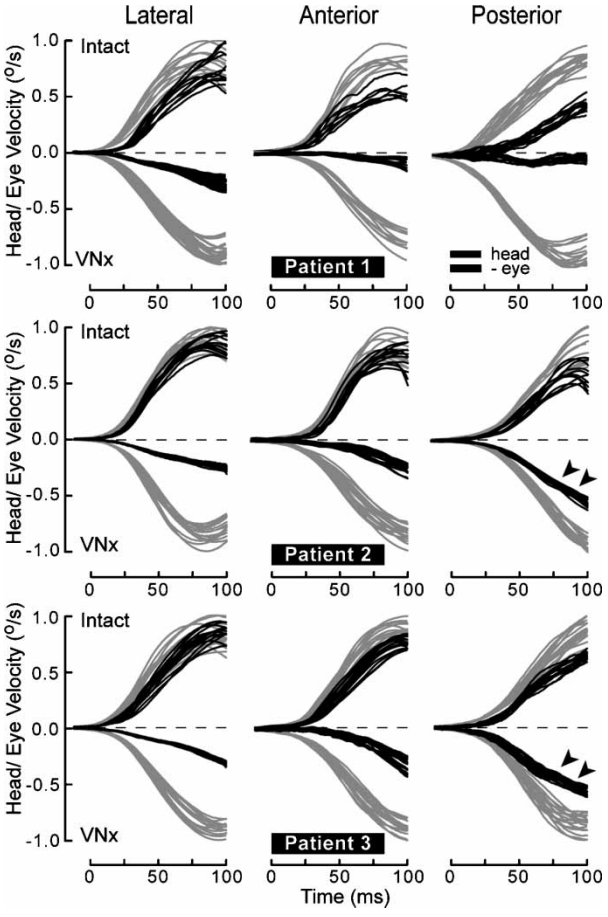

Figure 2. Comparisons of the angular VOR from lateral, anterior and posterior SCC canals in response to the three-dimensional head impulse test from three Ménière's disease patients post-VNx: Patient 1 is asymptomatic, Patients 2 and 3 still have persisting vertiginous attacks. Compensatory eye velocity has been inverted to facilitate comparison with head velocity. On the intact side in all patients, compensatory eye velocity is close to head velocity. On the VNx side, compensatory eye velocities were ~20% of peak head velocity for all three SCC canals for the asymptomatic Patient 1 and ~30% for lateral and anterior canals for symptomatic Patients 2 and 3. In Patients 2 and 3 on the VNx side, compensatory eye velocity was ~60% of peak head velocity (arrows) in response to posterior canal stimulation indicating residual posterior canal function. The mean VOR gains for lateral, anterior and posterior canals were: Patient 1 (0.21, 0.16, 0.11) on VNx side and (0.93, 0.54, 0.59) on intact side; Patient 2 (0.20, 0.29, 0.59) on VNx side and (0.97, 0.96, 0.82) on intact side; Patient 3 (0.19, 0.33, 0.58) on VNx side and (0.91, 0.82, 0.84) on intact side.

less, such as in asymptomatic Patient 1, showing complete loss of all SCC function. In contrast on the VNx side in the six patients with Ménière's disease who were still symptomatic (Figure 2, e.g. Patients 2 and 3), the VOR gains were: lateral SCC 0.24 ± 0.04; anterior SCC 0.29 ± 0.07; posterior SCC 0.52 ± 0.07 (mean ± 1 SEM). The higher mean VOR gain of the posterior canal of 0.52 on the VNx side in the six Ménière's patients who were still symptomatic indicated residual posterior canal function.

Standard VEMP test of saccular function on the intact side was normal in all Ménière's patients post-VNx. On the VNx side it was present and normal only in symptomatic Ménière's disease Patient 3, but absent in the other five symptomatic Ménière's patients and the asymptomatic Patient 1.

Discussion

The topography of the superior and inferior vestibular nerves varies from where they emerge into the IAC to where they merge to form the vestibulocochlear nerve bundle. The superior vestibular nerve innervates the lateral and anterior SCCs, the utricle and the antero-superior portion of the saccule, whereas the inferior vestibular nerve innervates the posterior SCC and the remaining portion of the saccule. The inferior vestibular nerve is usually adjacent to the cochlear nerve and some vestibular nerve fibres occasionally cross over to the cochlear nerve. The vestibular and cochlear nerves merge to form the vestibulocochlear nerve before entry into the brainstem, a point which shows inter-individual variations, posing a problem of identifying a cleavage plane between these nerves at operation [1,10]. Due to the proximity of the inferior vestibular nerve to the cochlear nerve, some inferior vestibular nerve fibres could be spared in an attempt to preserve the cochlear nerve.

MRI with a SSFP sequence in 0.8 mm overlapped slices enables visualization of the vestibular, cochlear and facial nerves within the IAC. Thin section (0.4–0.8 mm) axial reformatted MR images orthogonal to the vestibular nerve axis after curved reconstruction along the vestibulocochlear nerve in the IAC allow comparisons of the vestibular nerve bulk and signal between VNx and intact sides in the same patient. Curved reconstruction was used to display the vestibulocochlear nerve along its entire course because a simple sagittal reformatted image set was unable to display the nerves adequately due to its three-dimensionally curved course through the basal cisterns and the non-linear course of the IAC and hence does not provide accurate visualization of its components.

Using radiological analysis blinded to clinical findings, MRI was able to identify the intact side correctly in all seven patients. Residual inferior vestibular nerve was accurately identified in all six Ménière's patients who were still symptomatic, according to vestibular nerve bulk and signal, predicting the residual posterior SCC function. It was surprising that only one of these patients had residual saccular function, as measured by VEMP test [7,8], as about 50% of patients with Ménière's disease lose saccular function [9].

Here we have shown that high resolution MRI of the vestibulocochlear nerve using SSFP sequence, reformatted in a plane orthogonal to the vestibulocochlear nerve at several levels along the IAC, can identify residual vestibular nerve fibres that could be responsible for persisting vertigo attacks after selective VNx for Ménière's disease.

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