The site of brainstem lesions causing semicircular canal paresis: an MRI study

D A Francis, A M Bronstein, P Rudge, EPGH du Boulay

Abstract

Ten patients with canal paresis of central origin and ten patients with peripheral canal paresis were studied using MRI of the brainstem to identify lesions within the central vestibular pathways. In the central group, the magnitude of the canal paresis was generally lower than in the peripheral group and removal of fixation had little effect on the nystagmic response. In the peripheral group, removal of fixation enhanced the nystagmus and lessened the discrepancy between the two ears. Statistical processing of the MRI showed that in the central group significant spatially coincident lesions occurred within the medial vestibular nucleus, lateral vestibular nucleus and proximal portion of the vestibular fascicle.

Caloric testing is an important part of neuro-otological assessment of the vestibular system, using a technique that has remained largely unchanged for the past 50 years. Abnormal responses take two basic forms: canal paresis and directional preponderance. In the former, with which we are primarily concerned here, the duration of the nystagmus evoked from the impaired ear by irrigation with water at 7°C above and below body temperature is reduced with respect to the normal side; normative data collected by Hallpike et al. have shown that interaural differences of as little as 15 seconds may be significant. A Paretic response indicates an ipsilateral lesion occurring at some point along the vestibular pathway, from the labyrinth to the vestibular nuclei situated in the floor of the fourth ventricle, and is thus of limited localising value. While it is most often due to a peripheral disturbance of vestibular function more central lesions can cause similar abnormalities. However, work in this area of “central” canal paresis has been hampered by lack of accurate anatomical localisation of brain-stem lesions. The development of MRI has improved both the definition of normal architecture within the brainstem and the detection of pathological lesions. This has allowed us to evaluate and correlate the occurrence of canal paresis with respect to lesions within the central vestibular pathways.

Materials and methods

Patients with central canal paresis were included in the study on the basis of the caloric finding, demonstrating a significant canal paresis (see below), and the presence of clinical evidence of brainstem disease. These patients had been referred to the MRC Hearing and Balance Clinic at Queen Square for the assessment of eye movement and/or balance disorders. For comparison an equal number of patients attending the same clinic with presumed peripheral vestibular dysfunction and observed canal paresis were assessed.

Ten patients were recruited to each group and their clinical features are shown in table 1. All patients within the “central” group had established multiple sclerosis; by definition, the peripheral patients had no central neurological signs and were diagnosed as having a unilateral peripheral vestibular disorder, either idiopathic or suspected viral labyrinthitis (“vestibular neuritis”) or endolymphatic hydrops. There was a wide range in the duration of the vertiginous or brainstem symptoms in both groups of patients (one month to 20 years in the central group and two weeks to three years in the peripheral group) without significant differences between the two (t = 1.63). All but one patient with central signs were less than 50 years of age; this lessened the risk of areas of age-related abnormal signal being misinterpreted on the MRI scan.

Caloric irrigation was performed by the authors according to Fitzgerald and Hallpike's method. The duration of the nystagmus was measured by direct observation, with and without visual fixation; the latter was achieved by donning Frenzel's glasses when the nystagmus was no longer apparent in the presence of fixation. Quantitative data were derived using the standard formula for canal paresis (CP):

\[
\text{CP} = \frac{(\text{left cold} + \text{left hot}) - (\text{right cold} + \text{right hot})}{(\text{right cold} + \text{right hot} + \text{left cold} + \text{left hot})} \times 100
\]

On the premise that an interaural difference...
of at least 30 seconds was pathologically relevant (that is, 2 SD from a large normal population*), a canal paresis of greater than 8-3%, using the formula above, was considered significant. For convenience we took 9% as our lower limit.

Each patient had standard T1 and T2-weighted MRI scans performed on a 0.5 Tesla Picker superconducting system. An image matrix of 128 by 256 was routinely employed, giving pixel dimensions of approximately 2.4 mm by 1.2 mm. The most informative transverse brainstem image was at the level of the ponto-medullary junction. Here the superior (SVN), medial (MVN) and lateral (LVN) vestibular nuclei can be localised to the most lateral aspect of the floor of the fourth ventricle (figure 1). Lesions occurring in more caudal slices of the brainstem were not resolved well enough in relation to brain stem anatomy for statistical analysis. Most patients had TR/TE 200/80 and TR/TTI 1500/1000. MRI was performed using non-spectacles, enhanced the image brainstem nuclei can

Table 2 Caloric findings in 20 patients with canal paresis of peripheral and central origin

<table>
<thead>
<tr>
<th></th>
<th>Central n = 10</th>
<th>Periphera l n = 10</th>
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<tbody>
<tr>
<td>Canal Paresis</td>
<td></td>
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<tr>
<td>(Mean and Range)</td>
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<tr>
<td>VORS Index*</td>
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<tr>
<td>Left nystagmus</td>
<td>114% (100-128%)</td>
<td>206% (130-280%)</td>
</tr>
<tr>
<td>Right nystagmus</td>
<td>113% (100-126%)</td>
<td>186% (130-256%)</td>
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<tr>
<td>Canal paretic side</td>
<td>106% (100-136%)</td>
<td>196% (126-252%)</td>
</tr>
<tr>
<td>Non paretic side</td>
<td>128% (100-200%)</td>
<td>142% (131-154%)</td>
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*VORS index: vestibulo-ocular reflex suppression index, calculated as,

VORS Index = \[\frac{\text{duration of nystagmus without fixation}}{\text{duration of nystagmus with fixation}} \times 100\]

Figure 1 Schematic diagram showing the significant structures present at the level of the ponto-medullary junction. The vestibular nuclear complex is shown in solid black. The approximate trajectory of the VI, VII and VIII (vestibular) fascicles are indicated by dotted lines. SVN: superior vestibular nucleus; MVN: medial vestibular nucleus; LVN: lateral vestibular nucleus; MLF: medial longitudinal fasciculus; SCP: superior cerebellar peduncle, ML: medial lemniscus; CTT: central segmental tract.

cent transverse brain slices, were used to determine the presence of lesions. The chosen brain slice from each patient was enlarged and reported blindly, that is, the radiologist did not know the caloric result or whether the patient belonged to the central or peripheral group. Unequivocal areas of abnormal signal were subsequently traced on acetate sheets overlying an anatomical template outlining the brainstem contours. Identified lesions were coded and analysed for their relevance to the reported canal paresis by a statistical method described fully in previous studies. Briefly, this tests whether a given number of lesions overlapping a defined area of brainstem within a group of patients, linked by a common clinical finding, have occurred by chance.

Results
Caloric testing

The results are summarised in table 2. The canal paresis was generally of lower magnitude in patients with central lesions. Only three patients with central signs, compared with seven patients with peripheral vestibular dysfunction, had a canal paresis in which the responses from the affected ear were half the duration of those from the normal ear (that is, a canal paresis of 33%).

Removal of fixation, by the use of Frenzel's spectacles, enhanced the nystagmus in eight patients with peripheral lesions; the discrepancy between the two ears becoming less than 9% in six. By contrast, none of the patients with central lesions showed enhancement on removal of fixation, reducing the degree of canal paresis to below 9% in only one. Vestibulo-ocular suppression (VORS) was further quantified with the formula:

VORS index = \[\frac{\text{duration of nystagmus without optic fixation}}{\text{duration of nystagmus with optic fixation}} \times 100\]

Use of this formula showed values close to 1 for patients with central lesions (absent VORS) and close to 2 for peripheral patients (preserved VORS) (table 2, right and left nystagmus shown separately). Interestingly, in the peripheral group, VORS indices obtained from the paretic ear were greater than those from the non paretic ear (paretic side; lesions appearing as areas of increased signal (white) and low signal (black) respectively. Both sequences, and information from adjac
direction to the canal paresis in two of these.

**MRI findings**

All 10 patients with central canal paresis had an ipsilateral ponto-medullary lesion although in two cases the abnormal MRI signal did not intersect with the vestibular nuclei or fascicle (table 3). One patient, aged 53 years, with peripheral vestibular dysfunction had a ponto-medullary lesion on MRI scanning but this was contralateral to her canal paresis and probably an incidental finding considering her age. All other scans in this group were normal at this level.

The results of the analysis to determine the anatomical significance of areas in which lesions overlapped, in patients with central canal paresis, is shown in figure 2. The shaded and hatched areas in the grid matrix superimposed on the contours of the ponto-medullary junction represent the squares where levels of significance < 0.05 were achieved. The anatomical structure contained within the shaded square (p < 0.01) corresponded to a large portion of the medial vestibular nucleus and in the hatched squares (p < 0.03), in addition to

<table>
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<tr>
<th>% CP</th>
<th>M VN</th>
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<th>Vest fasc</th>
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<td>1</td>
<td>9.0</td>
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<td>2</td>
<td>35.8</td>
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The + signs under a particular neural structure estimate their degree of intersection with an area of abnormal MRI signal (+ + + indicates 100%). M, L and SVN: medial, lateral and superior vestibular nuclei; vest fasc: vestibular fascicle.

**Discussion**

The vestibular nuclear complex consists of four separate nuclei each receiving afferent information from several different sources. The medial and superior vestibular nuclei receive information directly from the cristae of the semi-circular canals. In addition, the vestibular nuclei receive sensory input from the otolith organs, from proprioceptors, particularly from the neck, as well as visual information. The cerebellum is also a major source of afferent information, giving significant contributions to the superior, lateral and medial vestibular nuclei and overall has important inhibitory control upon vestibulo-oculomotor function.

This anatomical arrangement explains two important observations in our study of caloric stimulation. The first is that the degree of canal paresis was in general of a lower magnitude in patients with central lesions when compared with those with abnormalities in the peripheral vestibular system. This agrees with previous experimental studies on primates showing that the greatest degree of canal paresis is produced by lesions in the vestibular nerve up to the level of the nerve root entry zone, the site at which the maximum number of afferent nerve fibres from the labyrinth could be damaged. Lesions in the nuclei related to canal afferents would have to be massive to achieve an equivalent deafferentation.

The second important point concerns the effect of removal of fixation upon the induced nystagmus. The removal of visual fixation produced enhancement of caloric responses in most of our patients with peripheral lesions but little significant change in those with central lesions. This finding is consistent with established observations on the relatively greater inhibitory effect of optic fixation on caloric nystagmus in peripheral compared with central vestibular lesions. Although our study is the first to show that lesions in the region of the vestibular nuclei do indeed cause a canal paresis that is unaffected by fixation, the fact that our clinical material comprises patients with demyelination makes it difficult to establish whether the association of canal paresis and reduced vestibulo-ocular reflex suppression arises in a single lesion or whether they are due to two or more coexisting lesions.

An unexpected finding in this study was that in the peripheral group the magnitude of nystagmus suppression by optic fixation was greater during irrigation of the parietic side than that of the non parietic side. The data from the study by Hood and Korres, using a similar caloric technique, was re-analysed and showed comparable findings to ours although the authors did not comment upon this point. The likely explanation for this finding is that the
nye Esk cence a pipe side is e more pressed by vision simply because it is weaker, and therefore easier to suppress, than the one from the non paretic side. This is again supported by re-analysis of Hood and Korres data9 which shows that VORS indices from the paretic side (2:26) are higher than those from the normal population (1:85), giving the appearance of "hyper normal" visual suppression. In addition, the fact that VORS indices from the non paretic side were somewhat lower than normal, both in our study (1:42) and in Hood and Korres data (1:65), might be due to the intense central inhibition exerted upon the vestibular system when there is a marked asymmetry between the two sides, such that any additional inhibition from the visual system is not apparent.

An important aim of this study was to identify what structures are involved in cases with central canal paresis. The data show that lesions affecting predominately the MVN, and to a lesser extent the LVN and proximal portion of the vestibular fascicle, appear to be the most important causes of "central" canal paresis, whereas the SVN is relatively spared. One limitation of this study has been the inability to identify components of the vestibular nuclear complex below the ponto-medullary junction. There is, however, 

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*J Neurol Neurosurg Psychiatry* 1992 55: 446-449
doi: 10.1136/jnnp.55.6.446

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