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## Experimental Brain Research Effects of vestibular disorders on vestibular reflex and imagery

Bernard Cohen, Joëlle Provasi, P. Leboucher, I. Israël

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# Experimental Brain Research

## Effects of vestibular disorders on vestibular reflex and imagery

--Manuscript Draft--

<b>Manuscript Number:</b>	EXBR-D-16-00713R1
<b>Full Title:</b>	Effects of vestibular disorders on vestibular reflex and imagery
<b>Article Type:</b>	Research Article
<b>Funding Information:</b>	
<b>Abstract:</b>	<p>The aim of this study was to establish the effect of vestibular lesion on vestibular imagery. Subjects were required to estimate verbally their passively travelled rotation angles in complete darkness, i.e., to activate vestibular imagery. During motion the Vestibulo-Ocular Reflex (VOR) was measured. Thus we examined the coherence between the vestibulo-ocular reflex and self-rotation imagery, with vestibular-lesioned patients and healthy participants.</p> <p>Unilateral acute and chronicle patients, bilateral patients and healthy subjects were compared. The stimulus was a sequence of eight successive passive rotations, with four amplitudes (from 90° to 360°) in two directions.</p> <p>The VOR gain was lower in patients with unilateral lesions, for ipsilateral rotations. The healthy subjects had the highest gain and the bilateral group the lowest, on both rotation sides. Thanks to vestibular compensation after acute unilateral neuritis the VOR gain increased in lesion side and decreased in healthy side, resulting in a similar gain in both sides.</p> <p>A deficit of vestibular imagery was found exclusively in patients with bilateral hyporeflexia, on both sides. The performance in vestibular imagery was good in the control group and correct in the unilateral patients.</p> <p>Finally we found a significant correlation between the efficiency of the VOR and that of vestibular imagery, exclusively in the bilateral patients.</p> <p>The present study shows the complex relationship between vestibular imagery and the VOR. This imagery test contributes to another assessment of the spatial handicap of vestibular patients. It seems particularly interesting for patients with bilateral canal paresis and could be used to confirm this diagnosis.</p>
<b>Corresponding Author:</b>	Isabelle ISRAEL, PhD CNRS PARIS, FRANCE
<b>Corresponding Author Secondary Information:</b>	
<b>Corresponding Author's Institution:</b>	CNRS
<b>Corresponding Author's Secondary Institution:</b>	
<b>First Author:</b>	Bernard Cohen, Dr.
<b>First Author Secondary Information:</b>	
<b>Order of Authors:</b>	Bernard Cohen, Dr. Joëlle Provasi, PhD Pierre Leboucher, Dr.Ing. Isabelle Israël, PhD
<b>Order of Authors Secondary Information:</b>	
<b>Author Comments:</b>	No conflict of interest
<b>Response to Reviewers:</b>	<p>Response to the reviewer #2: of Manuscript EXBR-D-16-00713 entitled « Effects of vestibular disorders on vestibular reflex and imagery » by Cohen et al.</p> <p>1. Materials and Methods The Material and Methods section has been rearranged with all the sub-sections that</p>

you indicated.

The 1st paragraph page 5 has been corrected as you suggested, thank you !

The consent has been moved in the material/subjects sub-section.

The familiarisation paragraph is now in the paradigm description.

Information about the participants and patients recruitment has been added, and a new table completes this description.

The eye movements recording equipment is now described in the set-up section, as well as the head support, and the slow phase analysis of the nystagmus is indicated in the data analysis section.

We now used a repeated measures ANOVA for the VOR and the VIR gains, which is indeed more reliable to compare several groups with different variables !

It was added in the data analysis that the Spearman non-parametric correlation was used, at the end of a new statistical analysis section. The correlation paragraph in the results has been much reduced.

## 2.Results

The section on VOR gain has been shortened to one page and one Fig.

The VIR paragraph has also been shortened and the stimulus-response correlation chapter was removed.

The correlation between VOR and VIR has been greatly simplified and shortened. But a paragraph on coefficient of variation has been added...

## 3.Discussion

In the first paragraph of the discussion, there is now a summary of the results. Then the VIR is discussed, followed by the correlation and differences between VIR and VOR, and the VOR is analysed only at the end, as you recommended.

The dissociated mechanisms in imagery and vestibular reflex are discussed together with recent literature on vestibule and cognition.

[Click here to view linked References](#)

B.S. Cohen<sup>1</sup>, J. Provasi<sup>2</sup>, P. Leboucher<sup>3</sup>, I. Israël<sup>4</sup>

Effects of vestibular disorders on vestibular reflex and imagery

1 Service ORL, Hôpital Saint-Antoine-UPMC, 46 Bld Raspail, 75007 Paris

2 Laboratoire CHArt, EPHE-PSL, 4-14 rue Ferrus, 75014 Paris

3 PRISME, ICM, CNRS UMR 7225, Hôpital Pitié-Salpêtrière, 75013 Paris

4 Laboratoire Cognac G, CNRS UMR 8257, 45 rue des Saints Pères, 75006 Paris

*Corresponding author :*

*Isabelle Israël*

*Laboratoire Cognac-G, CNRS*

*45 rue des Saints Pères*

*75006 Paris*

*isabelle.israel@bbox.fr*

*Tel : 06 34 39 35 58*

## Abstract

The aim of this study was to establish the effect of vestibular lesion on vestibular imagery. Subjects were required to estimate verbally their passively travelled rotation angles in complete darkness, i.e., to activate vestibular imagery. During motion the Vestibulo-Ocular Reflex (VOR) was measured. Thus we examined the coherence between the vestibulo-ocular reflex and self-rotation imagery, with vestibular-lesioned patients and healthy participants.

Unilateral acute and chronicle patients, bilateral patients and healthy subjects were compared. The stimulus was a sequence of eight successive passive rotations, with four amplitudes (from 90° to 360°) in two directions.

The VOR gain was lower in patients with unilateral lesions, for ipsilateral rotations. The healthy subjects had the highest gain and the bilateral group the lowest, on both rotation sides. Thanks to vestibular compensation after acute unilateral neuritis the VOR gain increased in lesion side and decreased in healthy side, resulting in a similar gain in both sides.

A deficit of vestibular imagery was found exclusively in patients with bilateral hyporeflexia, on both sides. The performance in vestibular imagery was good in the control group and correct in the unilateral patients.

Finally we found a significant correlation between the efficiency of the VOR and that of vestibular imagery, exclusively in the bilateral patients.

The present study shows the complex relationship between vestibular imagery and the VOR. This imagery test contributes to another assessment of the spatial handicap of vestibular patients. It seems particularly interesting for patients with bilateral canal paresis and could be used to confirm this diagnosis.

Keywords : vestibular disorders; self-motion perception; cognitive task; spatial imagery;

## Introduction

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6 The vestibulo-ocular reflex (VOR) keeps gaze stabilized in space during head  
7 movements. This apparently simple reflex (Collewijn 1989) has been extensively  
8 investigated, for example with patients suffering and recovering from vestibular disorders  
9 (Allum and Ledin 1999, Fetter and Dichgans 1990, Maire and Van Melle 2000). Vestibular  
10 input contributes directly to the VOR but also to non-reflexive perceptual responses including  
11 body orientation (Guedry 1974, Young 1981). In comparison, less is known about the  
12 vestibulo-perceptual system, probably because the vestibular organs (semi-circular canals and  
13 otoliths) are never stimulated alone, but always together with the tactile and proprioceptive  
14 system.  
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23 Moreover the relation between the VOR state and the complaints of vestibular patients  
24 is weak, according to Okada et al. (1999). We then wanted to find another test of vestibular  
25 function, closer to the patients problems. The vestibular system is actually the main idiothetic  
26 organ involved in path integration, i.e. keeping track of our own changes in orientation and  
27 position using self-motion cues (Mittelstaedt and Mittelstaedt 1980; Etienne and Jeffery  
28 2004). Therefore the vestibular system plays a crucial role in navigation and spatial  
29 orientation (Israël et al. 1996). Self-motion perception dysfunction and imbalance in case of  
30 vestibular lesion is becoming well described (Nakamura and Bronstein 1995, Cousins et al.  
31 2013), but less is known about vestibular spatial navigation.  
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41 Two main types of experiments have been led about vestibular perception or  
42 orientation :

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44 1: pointing responses with the eyes or the whole-body itself (Metcalf and Gresty 1992,  
45 Kanayama et al. 1995, Nakamura and Bronstein 1995), or with various devices indicating  
46 body orientation or velocity like button, tachometer, joystick (Okada et al. 1999, Seemungal  
47 et al. 2004, Cousins et al. 2013, Kyriakareli et al. 2013 and Tremblay et al. 2013).  
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51 2: "purely cognitive" responses (verbal magnitude estimate, reported perception) without  
52 pointing (Becker et al. 2000, Merfeld et al. 2005, Mergner et al. 1996, Cohen and Israël  
53 2004).  
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2 Although for some authors self-motion estimation is perception (Mergner et al. 1996,  
3 Becker et al. 2000), we believe that verbal estimate of motion magnitude or velocity is  
4 different from self-motion perception. Motion reproduction or return may be perception, but  
5 not magnitude estimate : the cognitive transduction from perception to its report in degrees or  
6 meters makes the difference, and this needs an internal representation (that self-motion  
7 reproduction does not need), i.e., imagery. Spatial navigation is "more cognitive" than  
8 perception, and it corresponds to one of those nowadays established higher vestibular  
9 functions (Besnard et al. 2015, Bigelow and Agrawal 2015, Lopez et al. 2012, Lopez 2013).

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16 Our hypothesis is that there is an effect of vestibular lesion on vestibular imagery,  
17 through damaged processing of self-motion information (acceleration, velocity and/or  
18 amplitude). Thus we examined with vestibular-lesioned patients and healthy participants how  
19 the vestibulo-ocular reflex (which indicates disorders) matches self-rotation imagery (or in  
20 other terms, how vestibular action matches cognition (Merfeld et al. 2005)).  
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## Material and Methods

### Subjects

We recorded 4 groups of vertigo patients (Table 1) :

Among the patients visiting a specialized medical consultation because of dizziness and/or postural instability, 4 groups of patients were selected for this retrospective study, after interrogation, physical examination, videonystagmography including oculomotor tests and checking for spontaneous or provoked nystagmus, rotatory tests with the analysis of VOR gain by cumulated slow phases, caloric test with cumulated slow phases velocity and also peak eye velocity. Patients with central signs (otoneurologic tests, MRI) were excluded.

\* dizzy patients with Bilateral Canal Paresis (BilCP), in the caloric test (sum of the 4 responses lower than  $20^{\circ}/s$  : Baloh et al. 1984) and in traditional rotary test (VOR gain  $< 0.2$ ).

\* patients exhibiting disabling vertigo since less than eight days : Acute Unilateral Canal Paresis (AcUCP), defined as a canal paresis superior to 80% in the caloric test according to Jongkees formula.

\* patients visiting after acute neuritis, more than three months before, because of persistent dizziness and without recovery at the caloric test : patients with Chronic Unilateral Canal Paresis (ChUCP).

\* patients with no vestibular symptom revealed by otoneurologic investigation considered as the Control Group (CG). The asymmetry between both sides computed with the Jongkees formula was lower than 25% and the directional preponderance was lower than  $2^{\circ}/s$ .

All the participants gave their written informed consent according to the guidelines of the hospital ethics committee.



## Set-up

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3 A motorized rotating chair was used, driven by a PC computer controlling angular  
4 position and velocity, recorded at 100 Hz (see Israël et al. 2006).  
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8 A video nystagmography helmet (VNG Ulmer, Synapsys) was used to record the  
9 horizontal movements of one eye lit by infrared light. Sampling frequency of the camera was  
10 25 images/s. The subject was seated, the head forward tilted of 30°, leaning against a head  
11 rest individually adjusted. The test started with calibrating of eye position through saccades,  
12 during about 20 seconds. The subjects also wore a headphone to mask external auditory cues.  
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## Paradigm

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24 The seated subjects were passively rotated around the vertical axis in complete  
25 darkness.  
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29 All subjects were submitted to the same sequence of 8 successive trials in clockwise  
30 (CW) and counter-clockwise (CCW) directions (90°CW, 180°CCW, 270°CW, 360°CCW,  
31 90°CCW, 180°CW, 270°CCW and 360°CW). The task was to estimate the rotated angle and  
32 verbally give its magnitude after each trial. The velocity profile was trapezoid, with 70°/s<sup>2</sup>  
33 acceleration and 60°/s plateau velocity. Plateau duration increased with increasing angle.  
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40 The experimental test was preceded by a demonstration of some stimuli and a  
41 familiarization phase of how to verbally indicate the imposed rotation angles (either in  
42 degrees or in quarter-turns). Three or four trials were presented, until the participant felt  
43 confident with the procedure.  
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48 We waited about 40 s between successive trials (until extinction of the post-rotatory  
49 nystagmus). With only eight successive bidirectional rotations there is no habituation of the  
50 VOR and of vestibular sensations which can be observed after a hundred angular velocity  
51 steps (Clement et al., 2008), and this same number of rotations was used by Cousins et al.  
52 (2013).  
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## Data analysis

The analysis of this vestibular imagery task was performed off-line through its gain :  
VIR (Vestibular Imagery Response) gain = estimated angle / travelled angle.

The nystagmus during the tests was recorded for each subject and each trial, thus the gain of the VOR (maximal eye velocity of slow phases minus spontaneous nystagmus velocity divided by maximal chair velocity) was calculated.

Since our hypothesis was that a canal lesion would impair both reflexive and cognitive performances, we compared the intact and disordered sides (2) of all groups (4). Therefore we applied a repeated measures ANOVA over these 4 groups to both the VOR gain and the VIR gain, separately. The responses to the ipsilesional side (IL, for CW rotations in unilateral right-lesioned patients and for CCW rotations in left-lesioned patients) and the contralesional side (CL, for CCW rotations in right-lesioned patients and CW rotations in left-lesioned patients) of the patients groups were thereafter compared.

ANOVA was applied on the means and also on the standard deviations (SDs) of the means of the same groups and sides.

After the analysis of the effects of vestibular disorders on the VOR and on the VIR, we intended to reveal the relationship between both gains. A Spearman non-parametric rank correlation was applied over the VOR gain and the VIR gain, so that it could be computed for the different groups, on the two ipsilateral and contralateral sides separately and/or pooled.

## Results

### 1. VOR gain (Fig.1)

The ANOVA on mean VOR gain over all trials of all groups yielded a significant main effect of group ( $F(3,52) = 24.37, p < 0.0001$ ).

The post hoc analysis (Tukey) revealed that CG had the highest gain ( $0.61 \pm 0.19$ ) compared to AcUCP, ChUCP and BilCP ( $p = 0.02, 0.002, \text{ and } 0.0001$ , respectively). BilCP gain was lower than AcUCP and ChUCP ( $p = 0.0005, p = 0.0003$ , respectively), but AcUCP was not different from ChUCP.

ANOVA revealed also a significant effect of side ( $F(1,52) = 17.63, p = 0.00011$ ), as the mean VOR gain of the IL, i.e., the lesioned side was lower than that of the CL side.

ANOVA finally showed a significant interaction between group and side ( $F(3,52) = 18.75, p < 0.0001$ ) which was due to exclusively one group : the acute unilateral group, which had different ipsilateral ( $0.27 \pm 0.09$ ) from contralateral ( $0.49 \pm 0.15$ ) gains (post hoc Tukey,  $p = 0.00013$ ). With the other groups (ChUCP, BilCP, CG) no different VOR gains between IL and CL were observed ( $p = 0.21; p = 0.87, p = 0.98$ , respectively).

Comparing the VOR gains of the AcUCP and ChUCP groups, it was found that the VOR IL mean gain tended to improve (it became higher from AcUCP :  $0.27 \pm 0.09$  to ChUCP :  $0.36 \pm 0.17, p = 0.0002$ ) while the CL side gain slightly worsened (it decreased from AcUCP :  $0.49 \pm 0.14$  to ChUCP :  $0.41 \pm 0.20, p = 0.0132$ ) during compensation.

There was also a significant difference between the VOR gain SDs of the different groups ( $F(3,52) = 4.69, p = 0.0057$ ), with the SDs exhibiting a similar pattern of group effect as the means. However there was no main effect of side on the VOR gain SDs ( $F(1,52) = 0.25, p = 0.62$ ), unlike with the means. But there was a significant group X side interaction on the SDs ( $F(3,52) = 3.52, p = 0.02$ ), due to BilCP IL gain SD lower than that of ChUCP IL and of CG IL ( $p = 0.04 \text{ and } 0.032$ , respectively).

## 2. VIR gain (Fig.2)

The angle estimation or vestibular imagery response (VIR) gain, i.e., the ratio between the estimated angle amplitude and the chair rotation amplitude, was then computed and analysed.

The ANOVA on mean VIR gain over all trials of all groups yielded a significant main effect of group ( $F(3,52) = 9.31, p = 0.00005$ ). So the different patients groups had different mean VIR gains.

The post hoc Tukey test showed that the VIR gain of the BilCP group was clearly and significantly lower than all the other groups gains ( $p = 0.005$  for AcUCP,  $p = 0.00023$  for ChUCP, and  $p = 0.00033$  for CG), while these other groups were not significantly different from each other. Indeed, as can be seen on the Fig.2, the mean VIR gains of the BilCP group were around 0.6 while the other groups were close to 1. On the VIR gain there was no main effect of side ( $F(1,52) = 0.28, p = 0.6$ ) nor interaction between side and group ( $F(3,52) = 1.3, p = 0.29$ ). This result is distinct from the VOR gain, where the AcUCP group had different IL and CL gains.

Finally, there was no main effect of group on the VIR gain SDs ( $F(3,52) = 1.58, p = 0.20$ ), neither main effect of side ( $F(1,52) = 0.016, p = 0.90$ ) nor group X side interaction ( $F(3,52) = 1.04, p = 0.38$ ).

## 3. VOR vs VIR gains (Fig.3)

The differences and also the correlations between VOR gain and VIR gain were thereafter examined.

It was first noted that the mean VOR gain was significantly lower than the mean VIR gain ( $F(1,52) = 239.39, p < 0.0001$ ). This was the case for all groups ( $F(3,52) = 1.69, p = 0.18$ ), and both sides ( $F(1,52) = 1.89, p = 0.17$ ).

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2 Then the correlation between the VOR gain and the VIR gain was computed for each  
3 group of subjects, including all the individual responses. It was revealed that exclusively the  
4 BilCP group exhibited a significant correlation ( $r = 0.225$ , two-tailed  $p = 0.027$ ).  
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6 We ultimately computed the coefficients of variation (SD/mean) of both these gains,  
7 in order to be able to safely compare the means (Fig.3). ANOVA on the coefficient of  
8 variation of the VOR gain over all groups was highly significant ( $F(7,104) = 6.61$ ,  $p <$   
9  $0.0001$ ). This was due on the CL side to the coefficient of BilCP ( $56 \pm 49\%$ ) larger than  
10 AcUCP, ChUCP, and CG (Tukey test,  $p < 0.001$  for each). ANOVA on the VIR gain  
11 coefficient was also highly significant ( $F(7,104) = 5.95$ ,  $p < 0.0001$ ), and the post-hoc Tukey  
12 test indicated on the CL side that the coefficient of BilCP ( $51 \pm 27\%$ ) was larger than AcUCP  
13 ( $p < 0.05$ ), ChUCP ( $p < 0.001$ ), and CG ( $p < 0.001$ ). The coefficient of BilCPIL ( $43 \pm 22\%$ )  
14 was also larger than that of CGIL ( $p < 0.01$ ).  
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24 There was no significant difference between the coefficients of variation of the VOR  
25 and VIR gains ( $F(1,52) = 1.22$ ,  $p = 0.27$  on the IL side, and  $F(1,52) = 0.085$ ,  $p = 0.77$  on the  
26 CL side). The coefficient of the BilCP group was the highest for both VIR and VOR, mostly  
27 for CL (i.e., CCW) rotations, as can be seen on Fig.3.  
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## Discussion

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6 The aim of this study was to establish the effect of vestibular lesion on vestibular  
7 imagery. Subjects had to estimate verbally their passively travelled rotation angles in  
8 complete darkness. The VOR was measured, so it could be compared with self-rotation  
9 imagery, in vestibular-lesioned patients and healthy participants.

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13 The VOR gain was lower in patients with unilateral lesions, for ipsilateral rotations.  
14 The healthy subjects had the highest gain and the bilateral group the lowest, on both rotation  
15 sides.  
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18 A deficit of vestibular imagery was found exclusively in patients with bilateral  
19 hyporeflexia, on both sides. The performance in vestibular imagery was good in the control  
20 group and correct in the unilateral patients.  
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24 Finally we found a significant correlation between the efficiency of the VOR and that  
25 of vestibular imagery, exclusively in the bilateral patients.  
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### 1 VIR is excellent in control subjects

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35 The VIR gain of the control group was close to 1 and symmetrical.  
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37 It should be recalled that because of the trapezoid velocity profile of the stimuli, long  
38 plateaus of constant velocity (for the largest angles) were sometimes used. But cognitive  
39 mechanisms did probably fill in for the decaying vestibular signal, enabling subjects to  
40 perceive displacements longer than what the characteristics of the system would allow. The  
41 vestibular (and somatosensory) cue that must have arisen when the chair stopped at the end of  
42 a trial, mostly was interpreted as a signal to end the internally maintained percept of an  
43 ongoing rotation (Becker et al. 2000, 2002).  
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49 It should be noted that the angle imagery method in the present study is very different  
50 from Cousins et al.'s (2013) velocity estimation method, which was the perceived slowing of  
51 rotational velocity, more intuitive and probably more approximate than angle estimation.  
52 However a dissociation was found between VOR and vestibulo-perceptual response in the  
53 acute unilateral patients, with both methods.  
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1 The imposed angles may seem too easy to estimate in our protocole, but everyone who  
2 tried (this can be done with an office chair) knows that this imagery exercise is extremely  
3 difficult in complete darkness. So with another less easy sequence the responses would  
4 certainly have been less accurate also for healthy subjects, but we feared that many trials  
5 would have been unusable because too difficult for several patients.  
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## 10 **2 VIR is correct in AcUCP ipsilateral**

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13 The correct VIR gain on both sides in the unilateral patients groups is an important  
14 result of the present study. It generated a dissociation in the vestibular processing, since the  
15 VOR gain of the lesioned side was quite low.  
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18 The fact that the VIR gain in AcUCP, even IL, was close to the VIR gain in CG  
19 suggests several hypotheses :  
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22 A/ there is immediately after lesion either a central component of the vestibular  
23 compensation phenomenon (Allum and Ledin 1999; Fetter and Dichgans 1990) or a peripheral  
24 component with the otolith system (Maire and van Melle, 2000). However this hypothesis is not  
25 supported by Metcalfe and Gresty (1992), who did not find immediate central compensation  
26 phenomenon in their unilateral patients after complete vestibular nerve section.  
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29 B/ perception uses both the excitation and the inhibition of lateral canals and does not  
30 need time for compensation as for the VOR. The possible extra-reflexive VIR pathway seems  
31 less lateralized than the reflexive VOR pathway : perception is not impaired like the VOR, as  
32 if the information transmitted by the two labyrinths would merge higher and would remain  
33 strong enough for the brain to rebuild perception. Cousins et al. (2013) asked whether a single  
34 labyrinth is capable of detecting motion in both directions, and the response is in the unilateral  
35 patients : when the lesioned side (IL) is stimulated, the healthy side (CL) is also stimulated.  
36 This is why the AcUCP and ChUCP groups had a correct VIR gain, by comparison with the  
37 BilCP.  
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40 So to explain the good VIR in the acute unilateral patients, we propose that the central  
41 pathways of rotation perception need information from both labyrinths, one safe and at least a  
42 weak input from the other one, in order to build good imagery immediately after lesion. VIR  
43 is the result of the stimulation of the two vestibules and consequently is not impaired in acute  
44 unilateral canal paresis.  
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### **3 VIR is affected only in BilCP**

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3 There was no dissociation with the BilCP group, since this group had poor gains in  
4 both VIR and VOR.  
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8 The VIR gain of the BilCP group was lower than all the other groups, on both sides. In  
9 order to explain this low VIR gain in BilCP we suggest that it could be on both sides a  
10 consequence of a failure of the velocity storage mechanism (Raphan et al. 1979), which does  
11 not extend enough the vestibular stimulation during the longer velocity plateaus used in our  
12 test.  
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18 Furthermore, the compensation is not immediate and cannot be achieved even with  
19 time, when the two labyrinths are affected.  
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23 For the BilCP group the coefficients of variation (SD/mean) of both the VIR and VOR  
24 gains were larger than 50%. This suggests that the patients were unsuccessfully trying to find  
25 complementary information, sensory or cognitive. Moreover this variation could exhibit a  
26 tired or a very tiring system between the differently efficient VIR and VOR, with recruitment  
27 followed by exhaustion.  
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33 According to Jandl et al. (2015), cerebellar activity during spatial navigation in BilCP  
34 patients may reflect increased non-vestibular efforts to counteract navigation deficits. Jandl et  
35 al. suggested a change in navigational strategy of BilCP patients, from allocentric to more  
36 sequence-based.  
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### **4 Correlation between the VOR and VIR gains.**

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47 The correlation between VOR gain and VIR gain was examined, as did Kanayama et  
48 al. (1995). For ipsilateral stimulation, Kanayama et al. found significant correlations for self-  
49 rotation and vestibular remembered saccades versus VOR gain. But the feedback that the  
50 subjects received after each trial might have improved their responses and instigated the  
51 correlation. Great underestimates of passive rotations were found in the present experiment in  
52 patients with bilateral hyporeflexia, together with very low VOR gain. Furthermore a  
53 significant correlation between VOR gain and VIR gain was found, exclusively with the  
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2 BilCP group. Thus VOR and VIR gains were indeed correlated, but only with the most  
3 deficient vestibular-lesioned patients.

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5 This correlation may be the manifestation of increased recruitment for these subjects,  
6 who were looking for complementary information to assist their deficient vestibular  
7 information.  
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11 Acute vestibular vertigo induces a drastic mismatch between vestibular, visual and  
12 somatosensory signals, which may lead to distortions of the body schema (Lopez 2012).  
13 Indeed internal models govern perceptions, while simple filtering governs the human VOR  
14 (Merfeld et al., 2005). Thus qualitatively different mechanisms contribute to human VOR and  
15 perceptual responses. Furthermore vertigo probably adds a perceptual noise disturbing signal  
16 detection (Cousins et al. 2013). Indeed perceptual thresholds are higher than VOR thresholds,  
17 thus vestibular brainstem mechanisms are more sensitive to angular acceleration than cortical  
18 conscious mechanisms. This is why the search for a correlation between the perceptual  
19 response and the sensory reflex one is legitimate.  
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29 Moreover a different pathway might be recruited by the BilCP patients, as Hitier  
30 (2015) reports that four different pathways transmit vestibular information to the cortical  
31 centers involved in cognition. The BilCP group might be specifically concerned by the  
32 pathway close to the cerebellum, which receives direct projections from the vestibular nerve,  
33 since Jandle et al. (2015) suggested that patients with bilateral vestibular lesions rely on the  
34 cerebellum for spatial navigation.  
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41 Kyriakareli et al. (2013) measured thresholds of perception and slow phase velocity  
42 under galvanic stimulation. They found a larger threshold enhancement for VOR than for  
43 motion perception, indicating a partial dissociation between cortical processing of VOR and  
44 perceptual responses. We found in the present study that the BilCP group exhibited a low  
45 VOR and also a low VIR gain, but the AcUCP IL group had a low VOR and a correct VIR  
46 gain, thus we indeed found partial dissociation.  
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## 5. VOR

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3 While the VOR gain of AcUCP was very different between IL and CL sides, in  
4 ChUCP a symmetrical gain was observed. This was the effect of vestibular compensation  
5 (Fetter and Dichgans, 1990; Halmagyi et al. 1990; Maire and Melle 2000). Interestingly, the  
6 VOR gain of the IL side in ChUCP was higher than the VOR gain of the IL side in AcUCP,  
7 and inversely the VOR gain of healthy side of ChUCP was lower than the VOR gain of the  
8 healthy side of AcUCP (Fig.1). So both sides grew closer to each other, from AcUCP to  
9 ChUCP, in an attempt to balance the state of both semi-circular canals. As if vestibular  
10 compensation would lead to an increasing gain in lesion side and a decreasing gain in the  
11 healthy side (Maire and Melle 2000, Halmagyi et al. 1990).

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20 The VOR gain of the BilCP group was low on both sides as expected since we  
21 included in this group patients with low responses in the caloric test and poor VOR gain in  
22 rotatory test.  
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27 Moreover, the VOR gains were close to those reported in the literature for healthy  
28 subjects (Clement et al. 2008; Baloh et al. 1986). Indeed the gain of the control group was  
29 considered normal in our experimental conditions : passive rotation in darkness without  
30 fixation and with a cognitive task (imagery of the travelled angle).  
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## 39 Conclusion

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42 As it had been found that vestibular information contributes to update our internal  
43 visual maps (Israël et al. 1999), we here specify and broaden this vestibular imagery process.  
44 The present study shows the complex relationship between vestibular imagery and VOR, and  
45 our test could contribute to another assessment of the spatial handicap of vestibular patients. It  
46 seems particularly interesting for patients with bilateral canal paresis and could be used to  
47 confirm this diagnosis.  
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54 Spatial imagery can be vestibular, and we finally found a correlation between  
55 vestibular imagery and vestibular reflex, but this correlation appeared exclusively with  
56 bilaterally disordered vestibular patients. These patients might have tried to rely on imagery,  
57 less deficient than the VOR. Because imagery is intuitive, and does not need training. Indeed  
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1 the task was very simple, even though it was not easy to respond accurately, and this is why  
2 BilCP was distinct from the other groups. It is also with an extremely simple task (counting  
3 seconds) that we had found a correlation between vestibular stimulation and time perception  
4 (Israël et al., 2004). But vestibular temporal imagery is beyond the scope of the present paper.  
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11  
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15 allowing us to realise this study with his motorised Barany chair.  
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Groups	Number	Age	Sex (male / female)	Symptoms	Inclusion criteria: Caloric Test
BilCP	12	58 (17)	7 / 5	Dizziness	Sum of the 4 responses < 20 °/s
AcUCP	11	43.4 (11)	5 / 6	Acute vertigo in the first week	Canal paresis asymmetry > 80 %
ChUCP	20	62.5 (7)	14 / 6	Neuritis with dizziness persistent > 3 months	Canal paresis remaining > 60 %
CG	13	51,5 (17)	6 / 7	No otoneurologic symptom now and before	Symmetric with responses > 10°/s for each ear

Ages are expressed as mean (SD)



## Figure captions

**Fig.1** Mean VOR (vestibulo-ocular reflex) gain + standard deviation of the four groups, with IL (filled bars) and CL (striped bars) rotation directions. The X-axis indicates the group.

**Fig.2** Mean VIR (vestibular imagery response) gain, same as Fig.1.

**Fig.3** Scatter graph of the coefficients of variation of the VOR and the VIR gains, for the four groups.







