

# Equilibrium test findings in patients with Bell's palsy

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## Abstract

**Objective:** We analyzed equilibrium test findings in patients with Bell's palsy to confirm that such findings occur not only in patients with Hunt's syndrome but also in patients with Bell's palsy.

**Methods:** Subjects were 83 patients with Bell's palsy, 45 patients with Hunt's syndrome, and 82 patients with unilateral chronic otitis media without history of vertigo or ear surgery (for control). They were treated in our inpatient department and underwent equilibrium testing during the period 1983–2002. All subjects were at least 20 years of age. The following were analyzed: (1) presence of vertigo, (2) presence of abnormal equilibrium test findings, (3) relation between the degree of paralysis and abnormal equilibrium test findings, and (4) relation between the degree of recovery from paralysis and abnormal equilibrium test findings. Equilibrium testing consisted of a gaze nystagmus test, a spontaneous nystagmus test, positional and positioning nystagmus tests, and a caloric test. The incidence of abnormal findings in each test was compared between the three groups.

**Results:** Abnormal equilibrium test findings were observed in patients with Bell's palsy. No association was observed between the results of equilibrium tests and the degree of facial paralysis in patients with Bell's palsy. However, some of the equilibrium test results were significantly associated with the degree of facial nerve recovery.

**Conclusions:** Although our study was limited in its lack of true control subjects, our data suggest that patients with Bell's palsy show a significantly high incidence of vestibular abnormalities.

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**Keywords:** Bell's palsy; Hunt's syndrome; Equilibrium test

## 1. Introduction

Bell's palsy and Hunt's syndrome are the most common causes of peripheral-type facial palsy. It is well known that patients with Hunt's syndrome often present with symptoms of the eighth cranial nerve. Several studies have shown abnormal equilibrium test findings not only in patients with Hunt's syndrome but also in patients with Bell's palsy [1–3]. To confirm this, we analyzed equilibrium test findings in patients with Bell's palsy or Hunt's syndrome who were treated in our department.

## 2. Subjects and methods

Eighty-three patients with Bell's palsy and 45 patients with Hunt's syndrome who were treated in our inpatient department and underwent equilibrium testing during the period 1983–2002 were investigated in this study. Eight patients with zoster sine herpete were included in the group with Hunt's syndrome. Bell's palsy was defined as the condition characterized by idiopathic facial palsy in the absence of vestibular and/or auditory symptoms relating to this palsy. Hunt's syndrome was defined as the condition characterized by peripheral-type facial palsy with herpes eruptions in the auricle and/or auditory meatus. Zoster sine herpete was defined as the condition characterized by peripheral-type facial palsy without herpes eruptions but with elevation of varicella zoster

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Table 1  
Sex and mean age of patients in this study

	Sex			Mean age	
	Male	Female	Total		
Bell's palsy	49	34	83	] * — * — *	48 (20 - 81)
Hunt's syndrome	17	28	45		49 (20 - 79)
Chronic otitis media	34	48	82		49 (20 - 76)

\* $P < 0.05$

virus antibody titer. All patients underwent equilibrium testing within 4 weeks after the onset of paralysis. We also analyzed preoperative equilibrium test findings of 82 patients with unilateral chronic otitis media without a history of vertigo or ear surgery. All subjects were at least 20 years of age. There were no significant differences in mean age between the three groups. The female-to-male ratio was significantly greater among patients with Hunt's syndrome or chronic otitis media than among patients with Bell's palsy (Table 1).

We analyzed the following: (1) presence of vertigo, (2) presence of abnormal equilibrium test findings, (3) relation between the degree of paralysis and abnormal equilibrium test findings, and (4) relation between the degree of recovery from paralysis and abnormal equilibrium test findings. Items (1), (3), and (4) were analyzed in patients with Bell's palsy or Hunt's syndrome. Item (2) was analyzed in patients with Bell's palsy, Hunt's syndrome, or chronic otitis media.

Equilibrium testing consisted of a gaze nystagmus test, a spontaneous nystagmus test, positional and positioning nystagmus tests, and a caloric test. We used electro-nystagmography (ENG) to record nystagmus. The gaze nystagmus test, spontaneous nystagmus test, and positional and positioning nystagmus tests were conducted with the use of Frenzel glasses. For the spontaneous nystagmus test, nystagmus was recorded with patients under three conditions in the sitting position with: (1) eyes closed, (2) eyes closed performing mental arithmetic, and (3) eyes open in the dark. The presence of nystagmus was used to define abnormality in the gaze nystagmus test, spontaneous nystagmus test, and positional and positioning nystagmus tests. The caloric test was performed by irrigating the external ear canal with 20 ml water at 30 °C or 44 °C or with 5 ml water at 20 °C, elevating the head, neck, and body in a straight line 30 °C from horizontal, with flexion at the hips. The bithermal caloric test was conducted in semidarkness with eyes open and with Frenzel glasses. Canal paresis (CP) was defined as a greater than 20% difference in caloric responses between the left and right ears. Directional preponderance (DP) was defined as a greater than 20% difference between leftward and rightward caloric responses. The monothermal caloric test was

conducted in darkness with eyes open. Caloric positional nystagmus was recorded before the monothermal caloric test and was subtracted from the caloric nystagmus response. Canal paresis in the monothermal test was defined as less than 10°/s of maximum slow-phase nystagmus velocity. Overall, equilibrium test results were considered abnormal if one or more of the equilibrium tests showed abnormalities.

The degree of paralysis was evaluated according to the grading system proposed by the Japan Society of Facial Nerve Research. The severity of facial nerve damage was categorized as complete paralysis (fewer than 8 points) or incomplete paralysis (more than 9 points). The degree of recovery from paralysis was categorized as complete (more than 36 points) or incomplete (fewer than 35 points). Statistical analysis was performed with Mann–Whitney  $U$ -test or  $\chi^2$ -test, as appropriate.

### 3. Results

#### 3.1. Presence of vertigo

None of the patients with Bell's palsy experienced vertigo, whereas 16 of 45 patients (36%) with Hunt's syndrome experienced vertigo.

#### 3.2. Abnormal equilibrium test findings

##### 3.2.1. Gaze nystagmus test

One of 83 patients (1.2%) with Bell's palsy, 2 of 45 patients (4.4%) with Hunt's syndrome, and 1 of 82 patients (1.2%) with chronic otitis media showed gaze nystagmus. There was no significant difference in the incidence of gaze nystagmus between the three groups of patients. (Table 2).

##### 3.2.2. Spontaneous nystagmus test

Thirty-one of 83 patients (37.3%) with Bell's palsy, 28 of 45 patients (62.2%) with Hunt's syndrome, and 17 of 82 male patients (20.7%) with chronic otitis media showed spontaneous nystagmus. There was a significant difference in the incidence of spontaneous nystagmus between these three groups of patients (Table 2).

Table 2  
Abnormal equilibrium test findings of patients in this study

	Type of paralysis	+	%	–	%	
Gaze nystagmus	Bell	1	1.2	82	98.8	
	Hunt	2	4.4	43	95.6	
	COM	1	1.2	81	98.8	
Spontaneous nystagmus	Bell	31	37.3	52	62.7	** *
	Hunt	28	62.2	17	37.8	
	COM	17	20.7	65	79.3	
Position nystagmus	Bell	11	13.3	72	86.7	** **
	Hunt	17	37.8	28	62.2	
	COM	5	6.1	77	93.9	
Positioning nystagmus	Bell	8	9.6	75	90.4	** **
	Hunt	14	31.1	31	68.9	
	COM	2	2.4	80	97.6	
Caloric test	Bell	6	7.2	77	92.8	** **
	Hunt	11	24.4	34	75.6	
	COM	5	6.1	77	93.9	
Overall	Bell	37	44.6	46	55.4	** ** *
	Hunt	31	68.9	14	31.1	
	COM	22	26.8	60	73.2	

Bell means Bell's palsy, Hunt, Hunt's syndrome, COM, unilateral chronic otitis media without history of vertigo or ear surgery, +, positive abnormal equilibrium test findings, –, negative abnormal equilibrium test findings.

\*  $P < 0.05$  \*\*  $P < 0.01$ .

### 3.2.3. Positional nystagmus test

Eleven of 83 patients (13.3%) with Bell's palsy, 17 of 45 patients (37.8%) with Hunt's syndrome, and 5 of 82 patients (6.1%) with chronic otitis media showed positional nystagmus. The incidence of positional nystagmus was significantly greater among patients with Hunt's syndrome than among patients with Bell's palsy or chronic otitis media (Table 2).

### 3.2.4. Positioning nystagmus test

Eight of 83 patients (9.6%) with Bell's palsy, 14 of 45 patients (31.1%) with Hunt's syndrome, and 2 of 82 patients (2.4%) with chronic otitis media showed positioning nystagmus. The incidence of positioning nystagmus was significantly greater among patients with Hunt's syndrome than among patients with Bell's palsy or chronic otitis media (Table 2).

### 3.2.5. Caloric test

Six of 83 patients (7.2%) with Bell's palsy, 11 of 45 patients (24.4%) with Hunt's syndrome, and 5 of 82 patients (6.1%) with chronic otitis media showed CP or DP. The incidence of CP or DP was significantly greater among patients with Hunt's syndrome than among patients with Bell's palsy or chronic otitis media (Table 2).

### 3.2.6. Overall evaluation of equilibrium tests

One or more of the equilibrium tests showed abnormalities in 37 of 83 patients (44.6%) with Bell's palsy, 31 of 45 patients (68.9%) with Hunt's syndrome, and 22 of 82 patients (26.8%) with chronic otitis media. The incidence of abnormal equilibrium test findings was significantly greater among patients with Bell's palsy or Hunt's syndrome than among patients with chronic otitis media. The incidence of abnormal equilibrium test findings also differed significantly between patients with Bell's palsy and patients with Hunt's syndrome (Table 2).

### 3.3. Relation between the degree of paralysis and abnormal equilibrium test findings

The incidence of gaze nystagmus was significantly greater among patients with incomplete Bell's palsy than among patients with complete Bell's palsy. No association was observed between the results of equilibrium tests, with the exception of the gaze nystagmus test, and the degree of facial paralysis among patients with Bell's palsy. No association was observed between the results of equilibrium tests and the degree of facial paralysis among patients with Hunt's syndrome (Table 3).

Table 3  
Abnormal equilibrium test findings in relation to the degree of paralysis

	Degree of paralysis	Bell				Hunt			
		+	%	-	%	+	%	-	%
Gaze	complete	0	0.0	66	100.0	1	3.0	32	97.0
nystagmus	incomplete	1	5.9	16	94.7	1	8.3	11	91.7
Spontaneous	complete	24	36.4	42	63.6	20	60.6	13	39.4
nystagmus	incomplete	7	41.2	10	61.1	8	66.7	4	33.3
Position	complete	9	13.6	57	86.6	12	36.4	21	63.6
nystagmus	incomplete	2	11.8	15	89.5	5	41.7	7	58.3
Positioning	complete	7	10.6	59	89.6	8	24.2	25	75.8
nystagmus	incomplete	1	5.9	16	94.4	6	25.0	6	50.0
Caloric test	complete	6	9.1	60	91.2	8	24.2	25	75.8
	incomplete	0	0.0	17	100.0	3	25.0	9	75.0
Overall	complete	30	45.5	36	54.7	22	66.7	11	33.3
	incomplete	6	35.3	11	62.5	9	75.0	3	25.0

Bell means Bell's palsy, Hunt, Hunt's syndrome, +, positive abnormal equilibrium test findings, –, negative abnormal equilibrium test findings.  
\* $P < 0.05$ .

### 3.4. Relation between the degree of recovery from paralysis and abnormal equilibrium test findings

Seventy-seven patients with Bell's palsy and 43 patients with Hunt's syndrome were evaluated with respect to the degree of recovery from paralysis.

In patients with Bell's palsy, there were significant associations between the degree of recovery from paralysis and abnormal findings in the positional and positioning nystagmus tests. No association was observed between the degree of recovery from facial paralysis and overall results of the equilibrium tests (Table 4).

There was no significant association between the degree of recovery from paralysis and abnormal equilibrium test findings in patients with Hunt's syndrome (Table 4).

## 4. Discussion

According to previous reports, abnormal equilibrium test findings are seen in patients with Bell's palsy [1–3]. Abnormal equilibrium test findings were seen in 4–82% of patients described in these reports. Our findings were similar to those of Adour and Doty [2] and Yagi et al. [5]. The

Table 4  
Abnormal equilibrium test findings in relation to recovery from paralysis

	Recovery	Bell				Hunt			
		+	%	-	%	+	%	-	%
Gaze	good	0	0.0	56	100.0	1	3.3	29	96.7
nystagmus	poor	1	4.8	20	95.5	1	7.7	12	92.3
Spontaneous	good	21	37.5	35	63.2	17	56.7	13	43.3
nystagmus	poor	6	28.6	15	72.7	10	76.9	3	23.1
Position	good	3	5.4	53	94.9	10	33.3	20	66.7
nystagmus	poor	7	33.3	14	68.2	6	46.2	7	53.8
Positioning	good	2	3.6	54	96.6	10	33.3	20	66.7
nystagmus	poor	5	23.8	16	77.3	4	30.8	9	69.2
Caloric test	good	2	3.6	54	96.5	7	23.3	23	76.7
	poor	4	19.0	17	81.8	3	23.1	10	76.9
Overall	good	22	39.3	34	61.8	20	66.7	10	33.3
	poor	10	47.6	11	50.0	10	76.9	3	23.1

Bell means Bell's palsy, Hunt, Hunt's syndrome, +, positive abnormal equilibrium test findings, –, negative abnormal equilibrium test findings.  
\* $P < 0.05$  \*\* $P < 0.01$ .

incidence of abnormal findings was higher in reports by Philipszoon [1] and Lämmler and Fisch [3] than in other reports, including ours. Koizuka et al. [4] speculated that the differences between reports are due to differences in the test period.

Lämmler and Fisch [3] and Koizuka et al. [4] studied the relation between the degree of paralysis and abnormal equilibrium test findings. Lämmler and Fisch [3] reported no association between equilibrium test findings and the degree of facial paralysis in patients with Bell's palsy. Koizuka et al. [4] reported that patients with CP had significantly more severe paralysis than patients without CP. Our data showed no association between equilibrium test findings and the degree of facial paralysis in patients with Bell's palsy.

Lämmler and Fisch [3] and Koizuka et al. [4] also discussed the relation between the degree of recovery from paralysis and abnormal equilibrium test findings. According to Lämmler and Fisch, only the evolution of spontaneous vestibular signs (particularly of positional nystagmus) indicated whether the lesion was reversible [3]. They did not conclude that vestibular signs present in Bell's palsy should be used for prognostic assessment of the lesion. Koizuka et al. [4] reported that patients without visual suppression showed poor recovery from paralysis, even though CP and DP were not dependent upon the degree of facial nerve recovery. Some of the equilibrium test results showed significant association with the degree of facial nerve recovery in our study. However, we could not conclude that vestibular signs are prognostic factors in cases of Bell's palsy because no association was observed between the degree of facial paralysis recovery and the overall evaluation of the equilibrium test results.

Abnormal equilibrium test findings in patients with Bell's palsy were observed in previously reported studies and also in the current study. Abnormal findings were observed less frequently in patients with Bell's palsy than in patients with Hunt's syndrome and were observed more frequently in patients with Bell's palsy than in patients with unilateral chronic otitis media without a history of vertigo or ear surgery. To evaluate the significance of abnormal equilibrium test findings in patients with Bell's palsy, it is important to compare patients with Bell's palsy and normal subjects. Only Adour and Doty [2] evaluated spontaneous and positional nystagmus in normal control subjects. They reported that 4% of their normal control subjects showed spontaneous or positional nystagmus, whereas 1.2–35% in other studies showed spontaneous or positional nystagmus. Thus, the reported percentages vary widely, and normal control groups should be included in each study. Because persons who would have qualified as normal control subjects had not undergone equilibrium testing in our department, we compared patients with Bell's palsy and patients with unilateral chronic otitis media without a history of vertigo or ear surgery. Although the patients with unilateral chronic otitis media were not true normal control subjects, our data

suggest that patients with Bell's palsy show a high incidence of vestibular abnormalities.

Philipszoon [1] reported that Bell's palsy is a disorder of the blood vessels in the temporal bone that affects different organs (cochlea, labyrinth, and facial nerve) individually or in combination. He speculated that this vascular disorder affects not only the facial nerve but also the labyrinth. Adour and Doty [2] did not agree that localized vascular disease in the temporal bone causes facial palsy and vestibular signs. Their findings indicate that idiopathic facial paralysis is a polyneuropathy involving the eighth cranial nerve, and they believe that Bell's palsy is a viral disorder, on the basis of its similarity to diseases of viral origin.

It was recently postulated that herpes simplex virus (HSV) is the causative agent of Bell's palsy. McCormick [6] first suggested this hypothesis based on the finding that HSV is the cause of recurrent cold sores, in all likelihood related to latency of the virus within the trigeminal ganglia. They speculated that HSV may be present in the geniculate ganglia, where it can cause neuropathy of the seventh nerve when it travels down the nerve axons. Murakami et al. [7] detected the HSV-type 1 (HSV-1) genome by polymerase chain reaction in 79% of patients with Bell's palsy but not in patients with Hunt's syndrome or in other control subjects. They concluded that HSV-1 infection of the facial nerve is directly related to the pathogenesis of Bell's palsy. This theory has been accepted, and acyclovir is currently used in the management of Bell's palsy.

Our findings are similar to those of Adour and Doty [2]. Therefore, we support their hypothesis that Bell's palsy is a polyneuropathy involving the eighth cranial nerve caused by a viral disorder. We suggest that virus such as HSV is the causative agent of vestibular signs in Bell's palsy. Further investigation is needed to fully elucidate the relation between HSV infection and vestibular signs in patients with Bell's palsy.

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