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ORIGINAL ARTICLE

Correlation between canal paresis and spontaneous nystagmus during early stage of acute peripheral vestibular disorders

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Abstract

Conclusions: This study demonstrates that the resolution period of spontaneous nystagmus (SN) may provide an indication of vestibular dysfunction on a particular day in the primary care setting. *Objective:* We aimed to predict canal paresis using fundamental observations of SN during the early stage of acute peripheral vestibular disorders. *Methods:* The study involved 87 patients who had recently experienced their first episode of acute spontaneous vertigo and direction-fixed horizontal nystagmus. Although they did not exhibit any other neurological deficits, they had been hospitalized with severe acute symptoms between 2004 and 2007. A correlation between the resolution period of SN and the results of laboratory caloric testing was reviewed. *Results:* The receiver operating characteristic analysis showed that the resolution period of SN may be a predictive indicator of unilateral vestibular hypofunction in the acute stage. In about half of the patients, SN disappeared on the third day after their initial visit. However, in 20% of the patients SN still persisted on the eighth day. Among the patients with SN, the prevalence of canal paresis increased with the increase in the resolution period of SN. When SN was observed on the fifth day, the prevalence was approximately 70%.

Keywords: *Acute peripheral vestibulopathy, acute isolated vertigo, vestibular neuritis*

Introduction

In acute peripheral vestibular disorders, patients usually suffer from acute symptoms of severe vertigo that persist longer than several hours, accompanied by intense nausea and vomiting. Almost all patients experience the onset of the disease as a surprise and are anxious [1]. The head thrust test or post-head-shaking nystagmus is known to be useful in predicting a significant unilateral vestibular hypofunction at the bedside of the patient [2]. However, it would be better to perform these dynamic vestibular tests in the sub-acute stage of the disease or later after spontaneous nystagmus (SN) and nausea/vomiting are reduced. During the early stage of acute peripheral vestibular disorder, oculomotor examination typically reveals nystagmus due to static vestibular imbalance by showing the mixed horizontal-torsional and direction-fixed SN that does not change direction with gaze and is suppressed with fixation, in the absence of other neurological symptoms or signs [2,3]. This study aimed to predict canal paresis (CP) using fundamental observations of SN during the early stage of acute peripheral vestibular disorders to help primary care providers and their patients.

Material and methods

This study reviewed the data of patients who were admitted to the otolaryngology ward of the university hospital and its affiliates between 2004 and 2007 for severe vertigo, nausea and/or vomiting, and gait impairment. The patients were referred primarily to

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either an emergency physician or an otolaryngologist, depending on the time of the patient's visit. Acute peripheral vestibular disorder was defined based on clinical features that suggested a possibly acute isolated vertigo of peripheral origin and was independent of the results of caloric testing subsequently performed. The criteria for inclusion were as follows: (1) a single episode of spontaneous vertigo that persisted for several hours, (2) the presence of a direction-fixed and horizontal SN that did not change direction with gaze and was suppressed with fixation compared with when Frenzel glasses were in place (we have included both mixed horizontal-torsional type and horizontal type); (3) no history of inner ear disease; (4) no cochlear symptoms; (5) no headache; and (6) no central nervous system involvement, as measured by neurological physical examination and urgent brain CT.

In clinical practice, central vestibular disorders occasionally present with features of peripheral vestibular nystagmus and a negative neurologic examination at the onset of a vertigo attack [3]. Patients were excluded from this study if they revealed central nervous system disorders on subsequent brain MRIs and neurological examinations during their course of hospitalization. They were also excluded if they exhibited unilateral sensorineural hearing loss on pure-tone audiometry. Finally, 87 patients were included in the study (33 men and 54 women aged 25–89 years; 31 patients at the university hospital and 56 patients at affiliates; Table I). SN was assessed daily in the morning at the bedside. Patients were administered anti-vertiginous and anti-emetic drugs without corticosteroids for several days to obtain relief from intense vertigo and nausea.

Table I. Summary of patients with acute peripheral vestibular disorder.

Parameter	Value
Subjects (n = 87)	
Sex (male/female)	33/54
Age (years)	61.5 ± 14.3
Acute symptoms	
Vertigo sensation (n = 87)	6.9 ± 7.6 days
Nausea and/or vomiting (n = 79)	3.1 ± 2.9 days
Difficulty in walking (n = 76)	3.0 ± 1.8 days
Findings	
Bedside test	
Nystagmus duration (n = 87)	5.3 ± 5.7 days
Laboratory vestibular test	
CP in caloric test	48.60% (35/72)
DP in rotational test	30.30% (10/33)

CP, canal paresis; DP, directional preponderance.

After nausea and vomiting were resolved, the patients were examined through routine laboratory evaluation of oculomotor-vestibular functions, which consisted of (1) spontaneous, positional, and gaze-evoked nystagmus; (2) pursuit, optokinetic system; and (3) caloric testing in sequence. The patients were also routinely evaluated using the sinusoidal rotational testing, galvanic body sway testing, and vestibular evoked myogenic potential testing in sequence [4]. Eye movements were recorded by the electronystagmographic (ENG) technique and sampled at 100 Hz with an A-D converter on a PC. Caloric stimuli consisted of alternate irrigation for 60 s with 6 L/min of cold and hot air, 24°C and 50°C, respectively. Asymmetry of vestibular function was calculated using the Jongkees formula. Significant CP corresponding to the evidence of unilateral vestibular dysfunction was defined as a response difference of 20% or more between the ears (laboratory-specific normal limits). The sinusoidal rotational test was performed with the patient's eyes closed in the dark with the head anteflexed to 30°. The patient was subjected to 0.1 Hz sinusoidal oscillations with a peak angular velocity of 75.4°/s. Directional preponderance (DP) was defined as a response difference of 10% or more between the ears (laboratory-specific normal limits).

In the present study, we aimed to predict the prevalence of unilateral vestibular hypofunction on a particular day in patients with SN using the resolution period of bedside SN (counted from the first visit) in patients with peripheral vestibules. First, receiver operating characteristic (ROC) analysis was performed to determine the predictability of the resolution period measures of SN; CP on laboratory caloric test was regarded as the gold standard. The area under the ROC curve (AUC) was estimated by the trapezoidal rule. An AUC > 0.60 was considered a highly predictive indicator. Second, the cumulative percentage of patients with SN in the early stage was examined. Third, among the patients with SN, the prevalence of CP with respect to a hospital stay was examined.

The duration of subjective symptoms was also examined. The relationship between the resolution of subjective symptoms and bedside SN was analyzed using Pearson's product-moment correlation coefficient. A correlation coefficient between 0.20 and 0.40 was considered mild correlation and 0.40–0.70 moderate correlation. A difference with a p value of < 0.05 was considered statistically significant.

Results

The visit was during the daytime (9:00 am to 5:00 pm) in 42% of the patients, during the evening or night (5:00 pm to 3:00 am) in 46%, and during the early morning (3:00 am to 9:00 am) in 12%. ROC analysis was performed to determine the predictability of the resolution period measures of SN (Figure 1). We found that the resolution period of SN could be a predictive indicator of unilateral vestibular hypofunction in the acute stage (AUC 0.63).

Figure 2A shows the cumulative percentage of patients with SN in the early stage. In about half of the patients, SN disappeared on the third day after the initial visit. However, in 20% of the patients SN still persisted on the eighth day. The prevalence of CP among the patients with SN is shown in Figure 2B. The prevalence increased with the increase in the duration of SN. For instance, when SN was observed on the third day, the prevalence was approximately 60%, and when SN was observed on the fifth day, the prevalence was approximately 70%. The duration of bedside SN did not correlate properly with DP in the subsequent laboratory rotational test (AUC 0.55).

Of the acute symptoms, nausea and/or vomiting and gait impairment resolved relatively quickly in most patients, but the resolution period of the vertigo

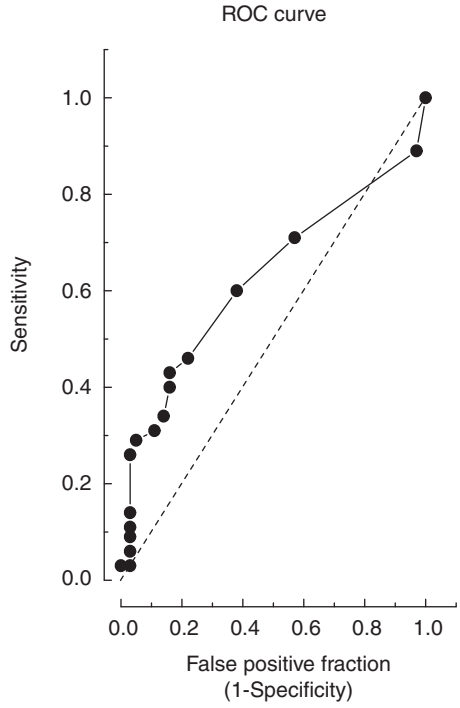

Figure 1. The receiver operating characteristic (ROC) curve indicates that the resolution period of spontaneous nystagmus in the acute stage may be a predictor of canal paresis (CP) in the caloric test. The area under the ROC curve was 0.63.

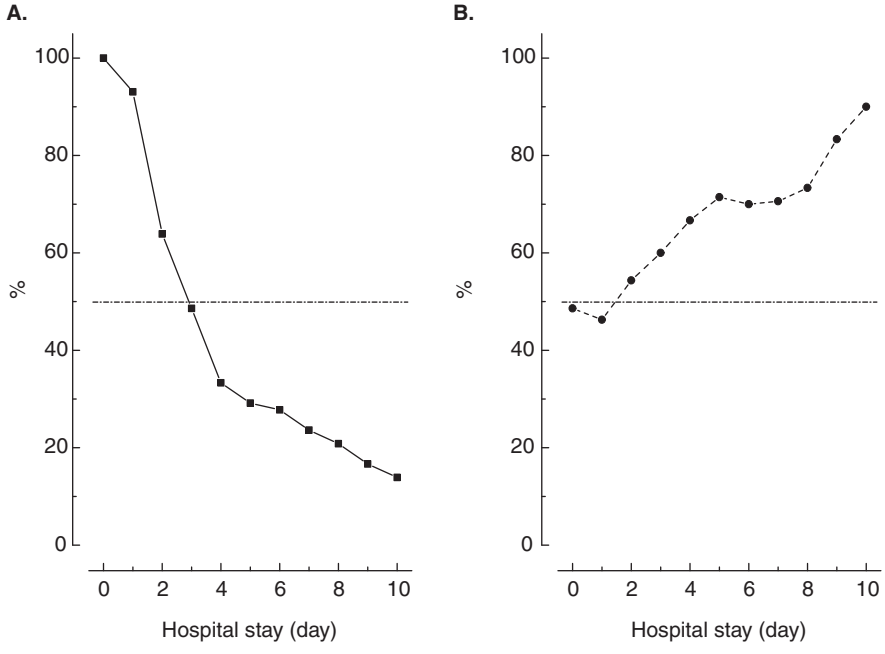

Figure 2. (A) The cumulative percentage of patients with acute peripheral vestibular disorder and spontaneous nystagmus (SN) with respect to the length of hospital stay in the acute stage ($n = 87$). On the third day from the initial visit, SN disappeared in about half of the patients; 20% of the patients still had SN on the eighth day. (B) The prevalence of canal paresis (CP) among these patients with SN ($n = 72$). These findings suggest that when SN is observed on the ninth day, the estimated prevalence rate of CP is approximately 90%. Zero indicates the date of admission.

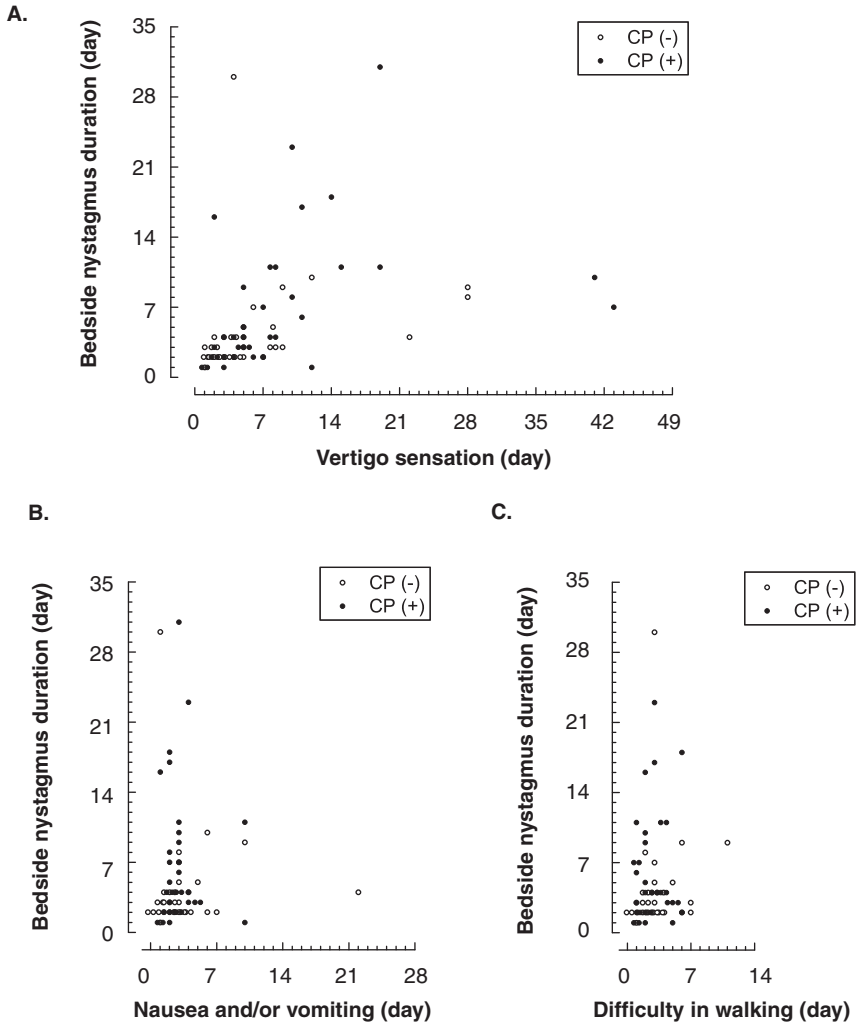

Figure 3. Relationships between resolution periods of acute symptoms and duration of bedside spontaneous nystagmus (SN). (A) Vertigo sensation. (B) Nausea and vomiting. (C) Difficulty in walking. Patients with significant canal paresis (CP) (●) and those without CP (○) are plotted separately. Longer duration of bedside SN correlated with longer resolution of vertigo sensation.

sensation varied widely among the patients (Table I and Figure 3). Correlations were observed between nausea/vomiting and the other two symptoms (correlation coefficient: vertigo sensation 0.403, $p < 0.001$; difficulty in walking 0.673, $p < 0.001$). Longer SN duration correlated with a longer resolution period of vertigo sensation (correlation coefficient 0.357, $p = 0.002$). No correlations were found between SN and the other two symptoms (correlation coefficient: nausea/vomiting −0.005, $p = 0.969$; difficulty in walking 0.110, $p = 0.390$).

Discussion

In clinical practice, a patient with a first episode of acute vertigo is often referred to a non-specialist, such as a general or an emergency physician, depending on the medical context among different countries and districts [5–7]. A large office-based survey by the National Ambulatory Medical Care reported that almost 70% of all outpatients with dizziness, who were aged 25 years and older, were initially examined by primary care physicians, such as general practitioners, family physicians, and general internists, and only 10% were examined by specialists such as otolaryngologists and neurologists [5]. Of all the patients who were referred to primary care physicians with the chief complaint of dizziness, 1.5% were hospitalized and 4.4% were referred to a specialist [5]. Vertigo/dizziness accounts for 2.5–3.5% of all the visits to the emergency departments [6,7]. Several reports have reported a clinically valid management of neurological emergencies; the focus would usually be on determining whether vertigo is related to a life-threatening condition [8–10]. Once a diagnosis of

vertigo of peripheral origin is made, it is best to estimate the evidence of vestibular hypofunction as the next step.

SN, characterized as horizontal, direction-fixed, and suppressed with fixation, in the absence of other neurological symptoms or signs reflects an imbalance in tonic neural activity in the vestibular nuclei due to sudden loss of input unilaterally from the labyrinth or the vestibular nerve. This study indicates that the resolution period of bedside SN could provide an indication of CP on a particular day. For instance, if SN presents on the fifth day after onset, the suspicion of it being CP is estimated to be approximately 70% (Figure 2B). A prospective study with 6 months follow-up showed that the degree of asymmetry of the caloric CP and age would influence balance performance and perceived symptoms after acute unilateral vestibular disorders [11]. A primary care physician may consult a specialist regarding the confirmation and degree of CP by means of a laboratory caloric test and additional treatment on the third day or later. A specific exercise improves residual imbalance and persistent dizziness [12–14]. Corticosteroids may be a treatment option because their administration in the early stage may improve the recovery of peripheral vestibular function in the long term [15] and facilitate central vestibular compensation [16,17].

Recently, Yagi et al. [18] analyzed the rotation axis of SN during the early stage of vestibular neuritis by means of an infrared charge-coupled device (CCD) camera. They determined that the rotation axis corresponds to the pathology of the superior vestibular nerve branch in all patients examined. In general, the axis initially tends to be mixed horizontal-torsional, and subsequently its orientation gradually changes during the recovery course of the early stage, as the anterior canal branch recovers faster than the horizontal canal branch, and ultimately draws closer to the horizontal [18]. This phenomenon is noticeable when the peripheral vestibular nystagmus is carefully observed using an infrared CCD camera. As mentioned in the Methods section, it is important to keep in mind that central vestibular disorder occasionally presents with a direction-fixed pure horizontal nystagmus [19,20] and, although rare, presents with a direction-fixed mixed horizontal-torsional nystagmus [21].

This study also examined symptomatic outcomes of patients who had experienced the first episode of acute isolated vertigo and direction-fixed horizontal nystagmus with symptoms severe enough to warrant hospitalization. Of the acute symptoms, nausea and/or vomiting (vestibulo-autonomic response) and gait impairment (vestibulo-spinal response) resolved relatively quickly in most patients, but the resolution period of vertigo (vestibular perception) varied widely among the patients. The duration of bedside SN (vestibulo-ocular response) was associated with the resolution of vertigo sensation, whereas it was not associated with the other two symptoms.

The presence of SN in vestibular disorders is a fundamental examination, which can be observed at the bedside in a matter of minutes without difficulty. In conclusion, observation of SN during the early stage of acute peripheral vestibular disorders provides useful information to non-specialists in predicting vestibular dysfunction, the timing of consultation, and planning of additional treatment.

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