

Alexander's Law Revisited

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Abstract

Background: It is a common occurrence in the balance function laboratory to evaluate patients in the post-acute period following unilateral vestibular system impairment. It is important to be able to differentiate spontaneous nystagmus (SN) emanating from peripheral vestibular system impairments from asymmetric gaze-evoked nystagmus (GEN) that originates from central ocular motility impairment.

Purpose: To describe the three elements of Alexander's Law (AL) that have been used to define SN from unilateral peripheral impairment. Additionally, a fourth element is described (i.e., augmentation of spontaneous nystagmus from unilateral peripheral vestibular system impairment) that differentiates nystagmus of peripheral vestibular system origin from nystagmus that originates from a central eye movement disorder.

Research Design: Case reports

Study Sample: Case data were obtained from two patients both showing a nystagmus that followed AL.

Intervention: None

Data Collection and Analysis: Videonystagmography (VNG), rotational, vestibular evoked myogenic potential (VEMP), and neuro-imaging studies were presented for each patient.

Results: The nystagmus in Case 1 occurred as a result of a unilateral, peripheral, vestibular system impairment. The nystagmus was direction-fixed and intensified in the vision-denied condition. The nystagmus in Case 2, by appearance identical to that in Case 1, was an asymmetric gaze-evoked nystagmus originating from a space-occupying lesion in the cerebello-pontine angle. Unlike Case 1, the nystagmus did not augment in the vision-denied condition.

Conclusions: Although nystagmus following AL usually occurs in acute peripheral vestibular system impairment, it can occur in cases of central eye movement impairment. The key element is whether the SN that follows AL is attenuated or augmented in the vision-denied condition. The SN from a unilateral peripheral vestibular system impairment should augment in the vision denied condition. An asymmetric GEN will either not augment, decrease in magnitude, or disappear entirely, in the vision-denied condition.

Key Words: Alexander's Law, central nervous system, gaze-evoked nystagmus, spontaneous nystagmus, vestibular

Abbreviations: AL = Alexander's Law; CT = computerized tomography; GEN = gaze-evoked nystagmus; MRI = magnetic resonance imaging; NI = neural integrator; VEMP = vestibular evoked myogenic potential; VNG = videonystagmography; VOR = vestibulo-ocular reflex; SN = spontaneous nystagmus; SPV = slow phase velocity; TC = time constant; VS = velocity storage

Sumario

Antecedentes: Es algo común en el laboratorio de función de balance el evaluar pacientes en periodo post-agudo, después de una trastorno unilateral de sistema vestibular. Es importante poder diferenciar entre un nistagmo espontáneo (SN) que emana de trastornos del sistema vestibular periférico, del

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nistagmo asimétrico evocado por la mirada (GEN) que se origina de trastornos en la motilidad ocular central.

Propósito: Describir los tres elementos de la ley de Alexander (AL) que han sido utilizados para distinguir el SN del trastorno unilateral periférico. Adicionalmente, se describe un cuarto elemento (p.e., aumento del nistagmo espontáneo a partir de un trastorno unilateral del sistema vestibular periférico) que diferencia el nistagmo de origen en el sistema vestibular periférico del nistagmo que se origina en un trastorno central del movimiento de los ojos.

Diseño de la Investigación: Reporte de casos.

Muestra del Estudio: Los datos de los casos fueron obtenidos de dos pacientes que mostraban nistagmo posterior a AL.

Intervención: Ninguna.

Recolección y Análisis de los Datos: Se mostraron los estudios de videonistagmografía (VNG), rotacionales, potenciales miogénicos evocados vestibulares (VEMP), y de neuro-imágenes para cada paciente.

Resultados: El nistagmo en el Caso 1 ocurrió como resultado de un trastorno unilateral, periférico, del sistema vestibular. El nistagmo tuvo dirección fija y se intensificó en condiciones de donde se niega la visión. El nistagmo en el Caso 2, en apariencia idéntico al del Caso 1, fue un nistagmo asimétrico evocado por la mirada, originado en una lesión ocupante de espacio en el ángulo pontocerebeloso. A diferencia del Caso 1, el nistagmo no aumentaba en condiciones donde se niega la visión.

Conclusiones: Aunque el nistagmo posterior a AL usualmente ocurre por trastornos agudos del sistema vestibular periférico, puede ocurrir en casos de trastornos centrales del movimiento de los ojos. El elemento clave consiste en diferencias si el SN que sigue a AL se atenúa o aumenta en condiciones donde se niega la visión. Un GEN asimétrico no aumentará, disminuirá en magnitud o desaparecerá enteramente, bajo condiciones que nieguen la visión.

Palabras Clave: Ley de Alexander, sistema nervioso central, nistagmo evocado por la mirada, nistagmo espontáneo, vestibular

Abreviaturas: AL = Ley de Alexander; CT = tomografía computarizada; GEN = nistagmo evocado por la mirada; MRI = imágenes por resonancia magnética; NI = integrador neural; VEMP = potencial miogénico evocado vestibular; VNG = videonistagmografía; VOR = reflejo véstibulo-ocular; SN = nistagmo espontáneo; SPV = velocidad de fase lenta; TC = constante de tiempo; VS = almacenamiento de velocidad

Acute unilateral peripheral vestibular system impairment is accompanied by spontaneous nystagmus (SN) with a fast phase that is directed toward the healthy ear. The slow phase velocity of the SN is greatest when gaze is directed toward the healthy ear (i.e., when gaze is directed toward the nystagmus fast phase), attenuates at central gaze, and may be absent when gaze is directed toward the ipsilesioned ear (i.e., when gaze is directed toward the nystagmus slow phase). These characteristics were first described by Alexander (1912), and they are referred to as "Alexander's Law" (AL). There is a third element of SN of peripheral vestibular system origin that became apparent only with the advent of electro-oculographic (EOG) instrumentation. SN at central gaze that occurs due to unilateral peripheral vestibular system impairment normally becomes augmented when vision is denied. That is, visual fixation, and specifically the pursuit eye movement subsystem, and vestibulocerebellum attenuates spontaneous vestibular nystagmus. Like SN that originates from a peripheral vestibular system impairment, asymmetrical (i.e., unilateral) gaze-evoked nystagmus (GEN) also may follow AL

when vision is present. However, since this nystagmus is evoked by gaze (i.e., hence the name), it normally is attenuated instead of being augmented when vision is denied. Accordingly, GEN is a clinical sign of central pathology. Patients with acute peripheral vestibular lesions may have SN with eyes open, and this finding can be misinterpreted as GEN. There are two ways to differentiate peripheral nystagmus (i.e., SN) from central nystagmus (i.e., GEN); nystagmus due to a peripheral vestibular lesion (i.e., SN) is more intense when the patient looks in the direction of the fast phases of the nystagmus (i.e., AL) and attenuates when gaze is directed toward the slow phases, and SN is suppressed with vision. Alternately, GEN is augmented by eccentric gaze and is reduced or absent when vision is denied.

The following are two case reports illustrating these concepts. The first case represents AL in a post-acute, unilateral vestibular system impairment. The second case represents an example of AL where the nystagmus occurred not because of peripheral vestibular impairment but instead because of a petroclival tumor compressing the cerebellum and brainstem. It is the third element, the augmentation or attenuation of

nystagmus when vision is denied (i.e., in the traditional SN test), that is critical for differentiating the possible causes of SN that follows AL.

CASE REPORTS

Patient 1

History

The patient was a 57-year-old male who reported dizziness with a first onset three years previous that occurred coincident with a coronary artery bypass procedure. It was the opinion of the physician managing his care that the patient might have had a stroke in the postoperative period. The patient reported increasing disequilibrium over the two-week period prior to presentation. This disequilibrium caused him to stumble when he attempted to walk. He also had right ear tinnitus for two weeks prior to his visit to the clinic. The patient was seen by a neurologist who requested an otolaryngology consultation to determine whether the patient had a vestibular neuronitis.

His past medical history was significant for coronary artery disease, insulin dependent diabetes, and chronic obstructive pulmonary disease.

Physical Examination

The Dix-Hallpike exam did not produce vertigo or nystagmus. The patient was too unsteady to perform the Fukuda stepping test.

Audiometric Results

Audiometric testing revealed a bilateral, moderate, flat, sensorineural hearing loss. Word recognition ability was excellent bilaterally. Acoustic immittance testing showed normal ear canal volumes and normal tympanic membrane (TM) mobility. Stapedial reflexes were present at expected intensities bilaterally. Accordingly, these results supported the contention that the patient had a moderate sensorineural hearing loss.

Magnetic Resonance Imaging (MRI)

The MRI study was essentially normal. There were only scattered bihemispheric and pontine white matter changes that were likely occurring secondary to chronic small vessel ischemic change. There was no acute infarct seen.

Balance Function Assessment

The videonystagmography (VNG) examination revealed a right-beating spontaneous nystagmus with a slow phase velocity of approximately 3.5°/sec on right

gaze. The SN attenuated to ~1°/sec at central gaze and was absent entirely when gazing to the left (see Figure 1a–c). This nystagmus increased in velocity to 21°/sec (i.e., from 1°/sec) when vision was denied (i.e., during testing for spontaneous nystagmus; see Figure 2). Saccade, pursuit, and optokinetic ocular motor subsystem tests were normal. The bithermal caloric test revealed an absent caloric response on the left (i.e., a 100% left unilateral weakness; see Figure 3), even in response to ice water. Peripheral vestibular system function was present on the right. Rotational testing revealed reduced vestibulo-ocular reflex (VOR) gains from 0.01 Hz through 0.16 Hz. This gain impairment was accompanied by phase leads across all frequencies tested (i.e., from 0.01 to 0.32 Hz), and asymmetries were present and consistent with the presence of a coexisting right-beating spontaneous nystagmus (see Figure 4a–b). The VEMP (vestibular evoked myogenic potential) examination showed symmetrical P13 latencies and amplitude asymmetries that were within normal limits (i.e., asymmetry of 19%; see Figure 5).

Treatment

The patient was enrolled in vestibular rehabilitation therapy for three months. At the end of therapy he was greatly improved. That is, he was independent in gait (i.e., able to ambulate without an assistive device) on all surfaces.

Patient 2

History

The patient was a 26-year-old female who was admitted to the hospital 30 weeks pregnant. The patient stated she was originally diagnosed with a tongue abscess two months prior to admission. A CT (computerized tomography) scan of the head was obtained at this time which showed an erosive process in the right clivus, although no comment was made about this lesion on the radiology report. Her main symptoms at this time were difficulty swallowing and mild dysarthria. She was placed on a course of antibiotics but her tongue symptoms did not improve. A repeat head CT scan was performed since the patient developed symptoms over a one-month period that included worsening headache, intermittent nausea and vomiting, right upper extremity weakness, difficulty in ambulation, and dysphagia. The CT scan showed hydrocephalus and progression of the skull base erosion. She was admitted for treatment.

Physical Examination

The patient was awake and alert; however, her speech was dysarthric. She was alert and oriented

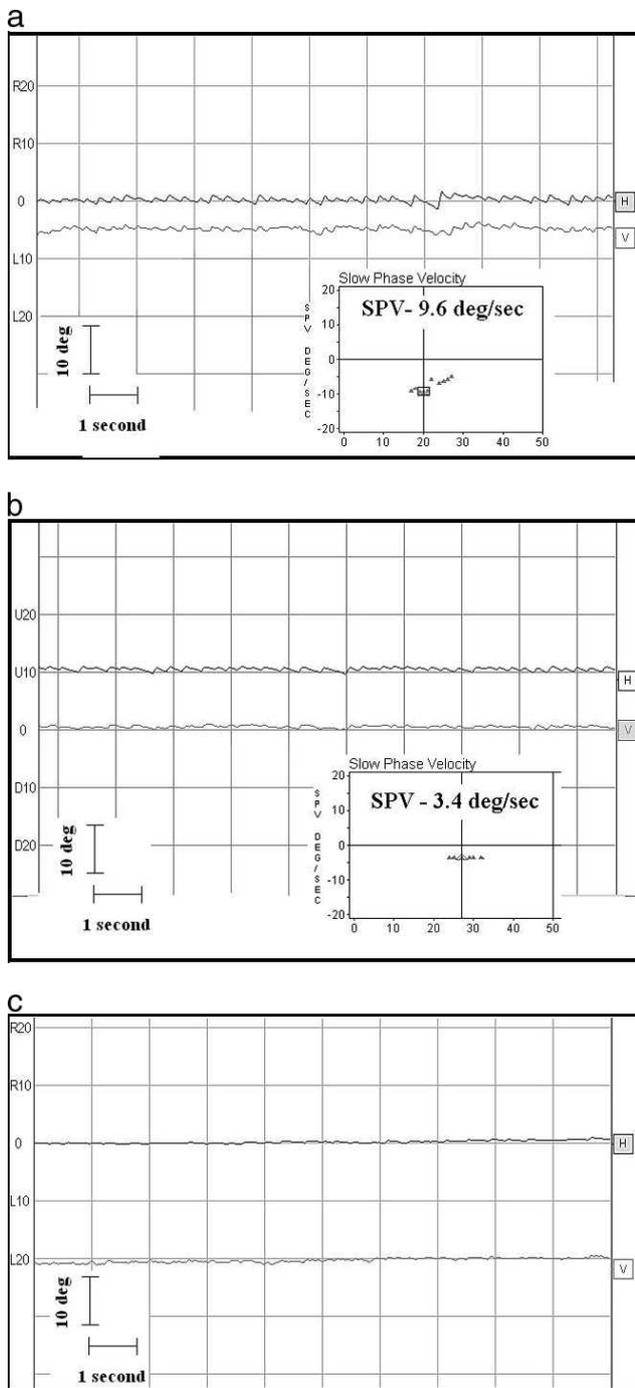


Figure 1. Gaze testing results for Case 1. (a) The patient is gazing to the right, in the direction of the fast phase of the SN. Notice that the patient is generating a right beating SN with a slow phase velocity (SPV) of $\sim 10^\circ/\text{sec}$. (b) The patient is gazing center. In this position the SPV has attenuated to $\sim 3^\circ/\text{sec}$ consistent with Alexander's Law. (c) The patient is gazing in the direction of the slow phase of the SN. Notice that the SN is absent.

times four. Her pupils were equal, round, and reactive to light. Her extraocular eye movements were intact; however, she demonstrated a right-beating nystagmus on right gaze. The patient commented that she experienced diplopia when she looked to the right. She had decreased sensation on the entire right side of

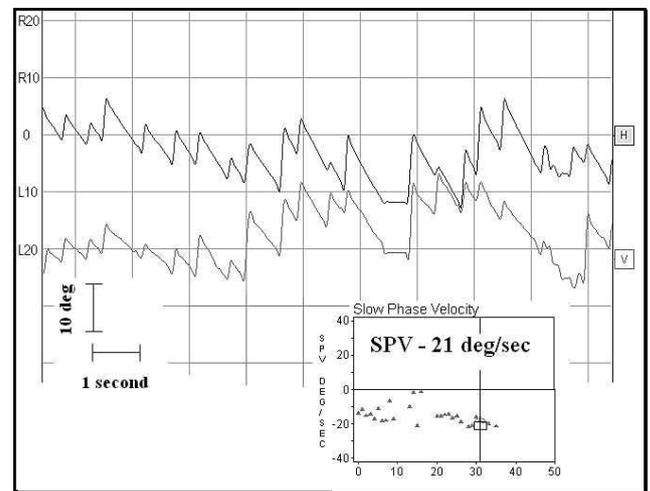


Figure 2. Spontaneous nystagmus test for Case 1 patient. In comparison to the SPV of the right beating SN with visual fixation present, in the vision denied condition, the SN increases in velocity from $\sim 3^\circ/\text{sec}$ to $21^\circ/\text{sec}$.

her face. Her facial muscle strength was a House-Brackmann Grade I (i.e., normal strength). Her palate elevated symmetrically. Her tongue deviated to the right. She had full strength on cranial nerve XI testing. Fiberoptic laryngeal examination showed a complete paralysis of the right vocal cord (i.e., impairment of cranial nerve X). On motor examination it was difficult to assess pronator drift, as she was very unsteady and had an unstable right upper extremity, with ataxia. Her left upper extremity strength was judged to be 5/5 (i.e., normal strength). Her left lower extremity strength was judged to be 5/5 (i.e., normal strength). Her right upper extremity strength was judged to be 4/5 throughout (i.e., slight weakness). Her right lower extremity was 5/5 (i.e., normal strength) throughout. She demonstrated dysmetria upon finger-to-nose

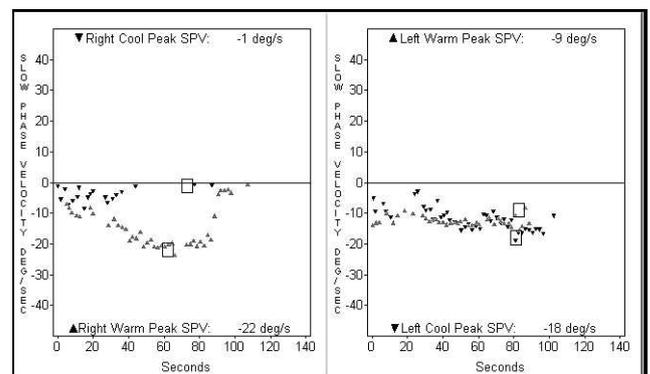


Figure 3. Caloric test result for Case 1 patient. Notice the bias toward right slow phase velocities in the caloric velocity plots. The magnitude of this bias is roughly equivalent to the magnitude of the spontaneous nystagmus in the vision denied condition.

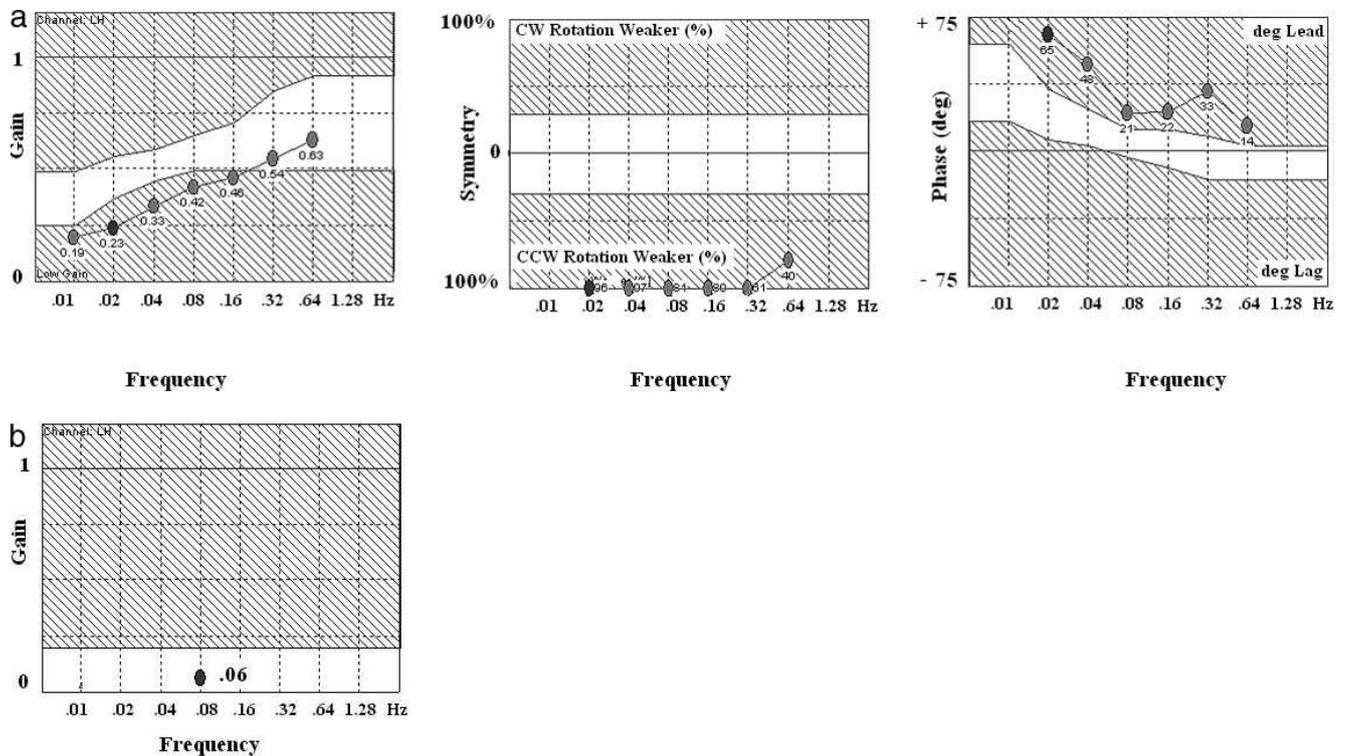


Figure 4. Rotational test result for Case 1 patient. The patient shows a reduction in low frequency VOR gain consistent with the loss of velocity storage (a). Further the patient shows multifrequency abnormal phase leads and a VOR gain asymmetry consistent with the direction of the slow phase of the SN (i.e., rotation toward the left results in greater nystagmus slow phase velocities than rotation toward the right). The patient shows normal VOR suppression (b).

testing with the right upper extremity suggesting cerebellar impairment on the right side.

Review of her MRI of the brain (see Figure 6a–c) obtained without contrast showed approximately 5 cm right-sided petro-clival mass with compression of the brainstem and right cerebellum. The lesion was isointense with brain on T1-weighted pre-contrast images. It showed increased signal postcontrast. The mass was slightly hyperintense on T2-weighted images with

minimal amount of surrounding edema. The mass abutted the right side of the fourth ventricle with widening of the foramen of Lushcka. The medulla was severely compressed to a diameter of 4 mm. There was extensive tumor involvement in the clivus and sphenoid bone. The tumor extended inferiorly to the hypoglossal canal and foramen magnum. There did not appear to be abnormal signal near the jugular foramen, but the tumor did not appear to originate in the jugular bulb. She did have enlarged temporal horns and a mild amount of ventriculomegaly. Angiography was performed, and no feeder vessels or tumor blush were seen. This tumor was consistent with a malignancy because of its deeply erosive nature and rapid growth.

Audiometric Testing

The patient showed normal pure tone sensitivity in both ears. Word recognition testing was excellent bilaterally. Acoustic immittance testing showed normal TM mobility, and ipsilateral stapedial reflexes were present bilaterally but showed elevated thresholds bilaterally (i.e., stapedial reflex thresholds of 100–105 dB HL for frequencies 500–2000 Hz). Crossed stapedial reflexes were bilaterally absent.

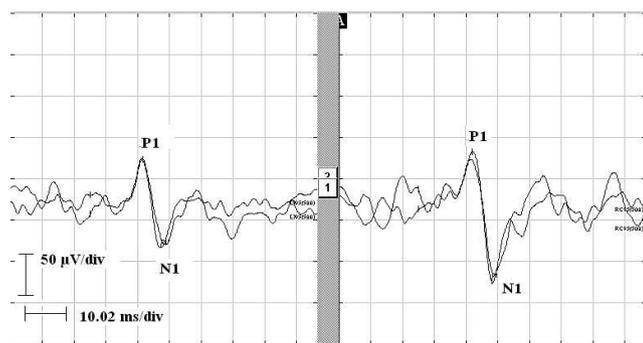
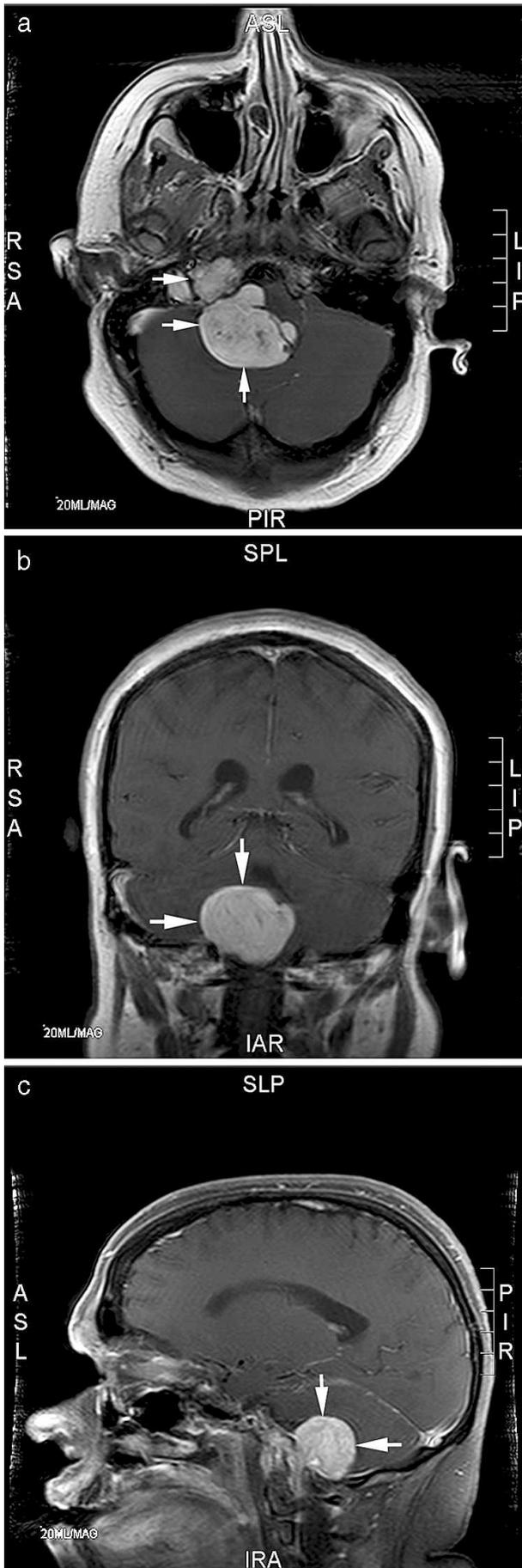


Figure 5. Vestibular evoked myogenic potential (VEMP) tracings for Case 1 patient. The latencies of the P13 components were 16.75 msec and 16.92 for left (i.e., left figure) and right ears (i.e., right figure), respectively. The VEMP amplitude asymmetry of 19% was not significant.



Both findings were suggestive of bilateral VIIIth cranial nerve impairments and pontine level brainstem impairments. Auditory brainstem response (ABR) testing showed absolute Wave V latencies to be slightly delayed on the left side (i.e., 6.11 msec for the right ear and 6.37 msec for the left ear). Technically poor recording conditions resulted in increased physiological interference (i.e., increased EMG contamination) and an inability to resolve Wave I to compute a Wave I–Wave V interwave interval.

Balance Function Assessment

VNG testing revealed a coarse right-beating SN on rightward gaze on informal testing (see Figure 7a). This nystagmus was attenuated at center gaze (see Figure 7b) and was absent on leftward gaze (see Figure 7c). The nystagmus velocity was attenuated in the vision-denied condition (see Figure 8). The SN contaminated the pursuit and optokinetic examinations. That is, pursuit for both tests was saccadic when targets were moving in the direction of the fast phase of the SN. The caloric test was bilaterally normal (see Figure 9). The patient showed a 7% unilateral weakness and a 5% directional preponderance. VOR suppression (i.e., fixation suppression) was abnormal.

Rotational testing (see Figure 10a–b) showed the patient to have normal VOR gain and phase for frequencies 0.02 Hz, 0.8 Hz, and 0.32 Hz. The patient demonstrated a VOR asymmetry at 0.02 Hz and 0.08 Hz. That is, consistent with the right-beating GEN, the patient showed reduced slow phase velocity (SPV) when the chair was oscillating toward the left and greater SPV when the chair oscillated to the right.

The vestibular evoked myogenic potential (see Figure 11) was present and of normal latency on the left side and was absent on the right side.

Treatment

Surgical resection was attempted. An intraoperative biopsy showed the mass to be a “small round blue cell tumor” consistent with a primary malignancy. It was decided that surgery could not be curative, so the tumor was “debulked” to provide relief of brainstem compression. Final pathology showed a primitive malignancy most likely an osteosarcoma.

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Figure 6. Post-contrast T-1 weighted axial (a), coronal (b), and sagittal (c) MRI images of patient in Case 2. The images show the tumor with compression of right cerebellum and brain stem. Extensive erosion into the clivus may be seen.

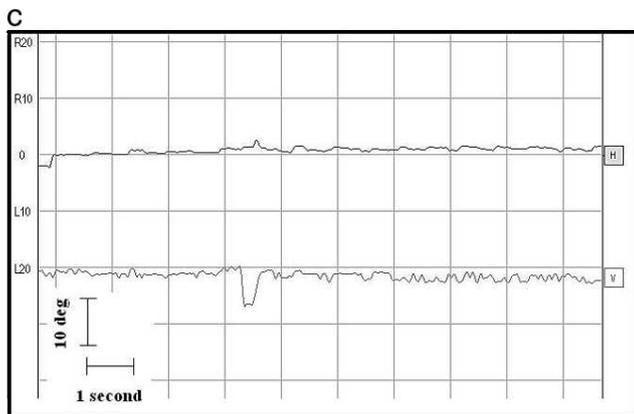
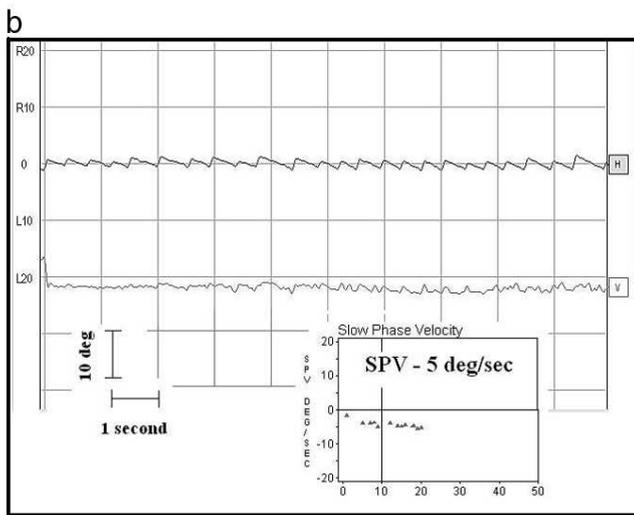
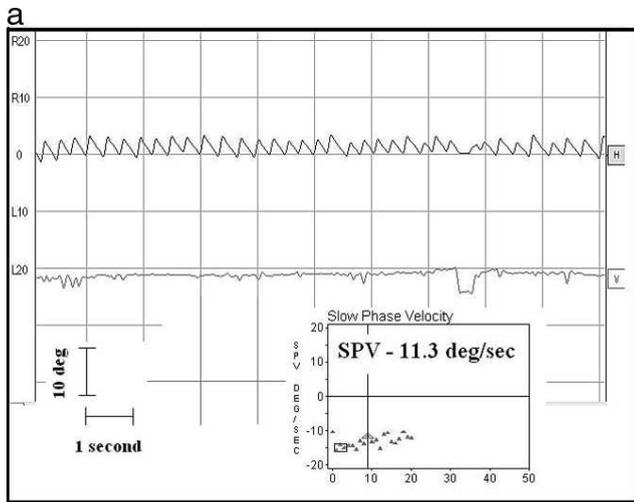


Figure 7. Gaze testing results for Case 2. (a) The patient is gazing to the right, in the direction of the fast phase of the SN. Notice that the patient is generating a right beating SN with a slow phase velocity (SPV) of $\sim 11^\circ/\text{sec}$. (b) The patient is gazing center. In this position the SPV has attenuated to $\sim 5^\circ/\text{sec}$ consistent with Alexander's Law. (c) The patient is gazing in the direction of the slow phase of the SN. Notice that the SN is absent.

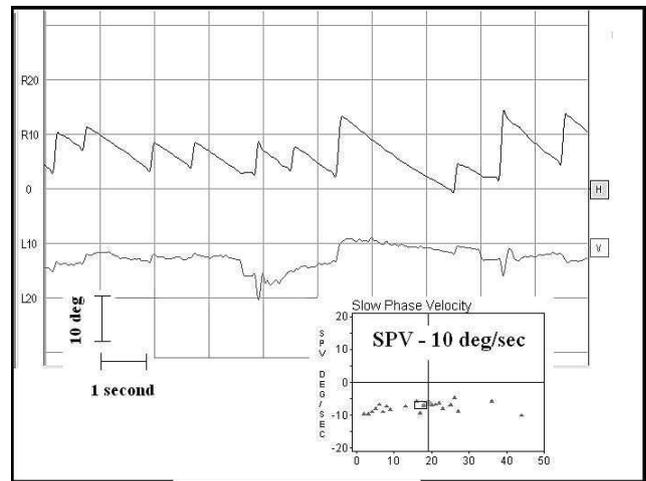


Figure 8. Spontaneous nystagmus test for Case 2 patient. Notice that in the vision-denied condition the slow phase velocity (SPV) is intermediate between that occurring in the "gaze right" and "center gaze" conditions (i.e., SPV of $\sim 10^\circ/\text{sec}$). The patient does not demonstrate the augmentation of the SN that is observed in Case 1.

COMMENT

The mechanisms underlying AL have been understood only in the last two decades. Prior to this, it was felt that eye mechanics produced a greater SPV when the eye was deviating toward midline (i.e., away from the fast phase of the nystagmus beat and toward the eye's "resting" position) than when moving away from midline. Thus, a longer muscle would be capable of contracting further and pulling the eye further (i.e., the slow phase of the nystagmus beat) before the correcting saccade would bring the eye back to its starting point. In the same vein, shorter muscles (i.e., muscles that have been full contracted) would be expected to produce lower slow wave velocities since

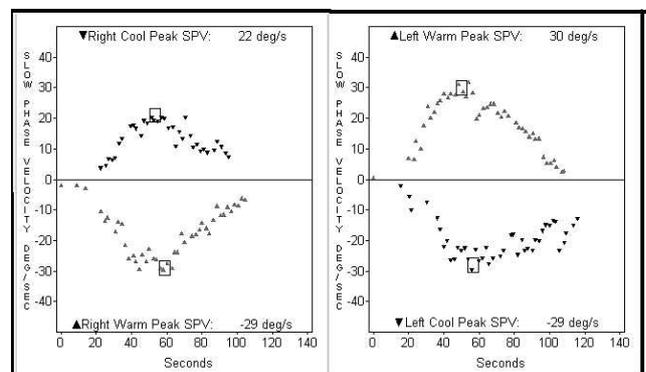


Figure 9. Caloric test result for Case 2. Notice that even though the patient generates what might have appeared to be a SN in the vision-denied condition, the caloric responses were symmetrical without a right directional preponderance. This is evidence that the nystagmus is not occurring due to a tonic asymmetry in peripheral vestibular system function but instead represents an impairment in gaze-holding (i.e., a gaze-evoked nystagmus).

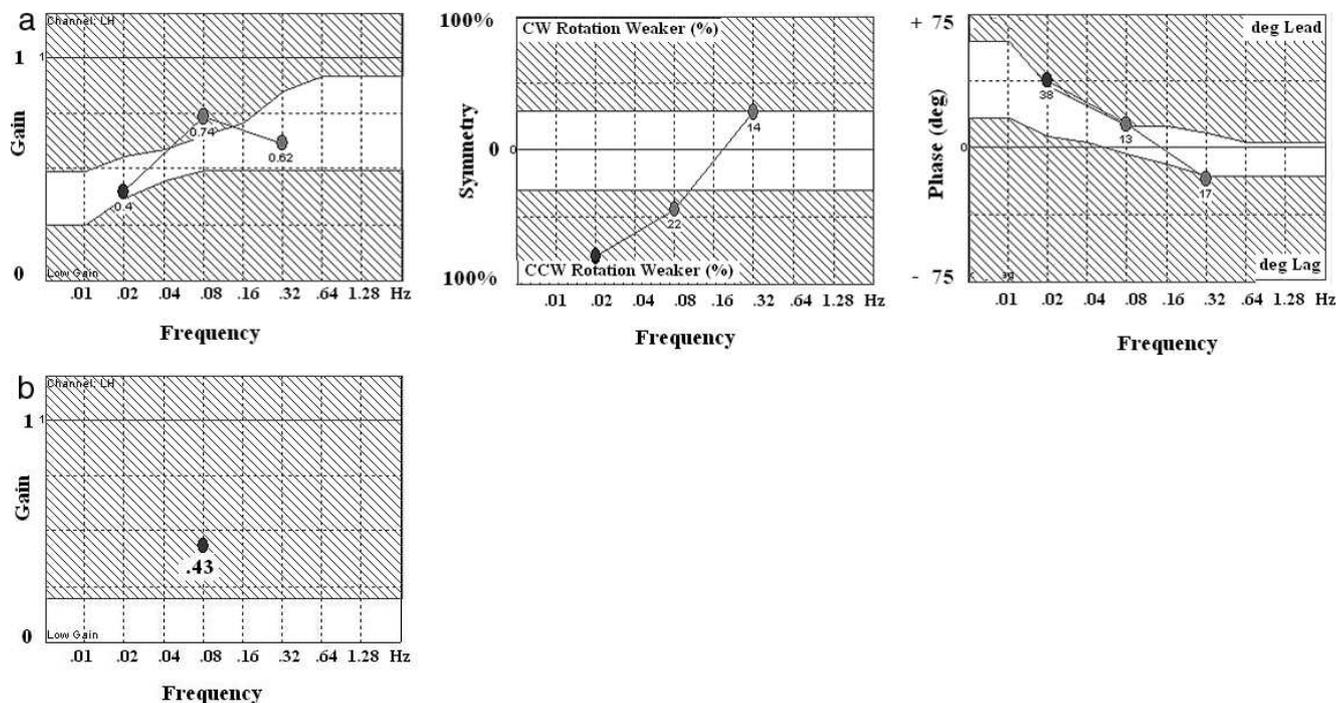


Figure 10. Rotational test result for Case 2. (a) The patient demonstrates normal VOR phase and gain but shows a VOR asymmetry in middle and lower frequencies (i.e., left-beating nystagmus is weaker than right-beating nystagmus). (b) Result of VOR suppression test showing that the Case 2 patient has asymmetrically impaired VOR suppression. That is, the patient is attempting to stare at a fixed target moving with the chair as the chair is oscillating left and right. The patient is generating nystagmus (an abnormal finding) for rightward oscillations only. This impaired VOR suppression is consistent with that seen in lesions that impair neural connections between the vestibulocerebellum and the vestibular nuclei.

they were already contracted to their limits (e.g., the left lateral rectus muscle when the eyes are deviated to the left in a patient with a right-beating SN). However, this explanation for AL appears to be incorrect (Robinson, 1981).

Contemporary thought is that AL is produced by two adaptive processes that are initiated by unilateral vestibular system impairment (Robinson et al, 1984). The first process is the “defeat” of velocity storage (VS). VS is a central vestibular system process by which the flow of electrical activity from the periphery (i.e., the afferent electrical signal from the end organ) is stored and the gated to drive the VOR in such a way that the

eye response becomes roughly double the actual duration of the electrical activity flowing through the nerve into the vestibular nuclei. In fact, the VOR time constant (TC; i.e., the time required to reduce the nystagmus SPV to 37% of its initial intensity) without velocity storage is ~6 sec and with velocity storage is ~18 sec (Hain and Zee, 1992). The physiological significance of VS is to extend the low frequency response of the system by two orders of magnitude from ~0.10 Hz without VS to ~0.001 Hz with VS intact. From a practical standpoint, the loss of VS should reduce the TC of the VOR and hasten the decrease in the maximum velocity of the SN that occurs after a unilateral vestibular impairment (and the physiological symptoms that accompany the SN).

The second process that contributes to Alexander's Law is a change in the “leakiness” of the neural integrator (NI). The NI is a function that is distributed within the brainstem and cerebellum (i.e., the flocculonodular lobe of the cerebellum, and nucleus prepositus hypoglossi and medial vestibular nucleus in the pons; Leigh and Zee, 1999). The NI provides the eye movement system with the ability to move the eyes to an eccentric position and for the eyes to maintain their position once they have arrived at the target. It is significant to note that the electrical drive from the vestibular nuclei contribute to gaze holding. The role of the NI is to calculate the level of electrical “tone” that

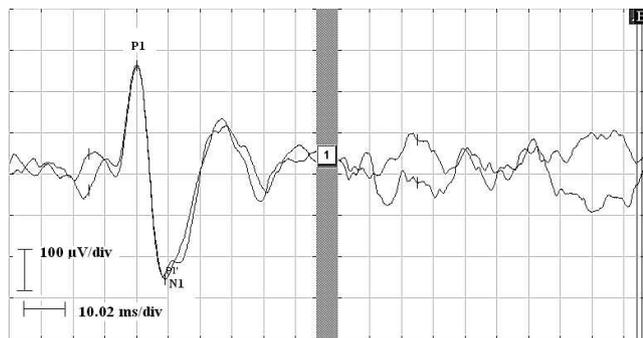


Figure 11. VEMP test result for Case 2 patient. The VEMP is absent on the right side and present with a latency of 18.25 msec on the left side.

is required to hold the eyes in their new position and to maintain that position for the duration of the eye movement. A “leaky” NI cannot maintain a constant electrical tone, and accordingly, the eyes drift back from their eccentric position to midline. This, in fact, is the origin of gaze-evoked nystagmus (GEN). That is, if the eyes are driven from midline to a rightward target, and if the NI is “leaky” once the eyes reach the target, the electrical drive that is routed to the agonist muscles will show an exponential decay and the eyes will drift back to midline. The vision system then initiates a corrective saccade to bring the eyes back to the target. This repetitive drift, corrective saccade, drift, corrective saccade, and so on is the origin of GEN. The GEN usually is bidirectional. That is, gaze to the right initiates a right-beating GEN, and gaze to the left produces a left-beating GEN. However if a patient is generating a right-beating SN, gaze to the right will result in an enhanced SN, gaze to the left will result in a cancellation of the right-beating SN by the left-beating GEN. Thus, the “leaky” NI produces at least one direction of gaze where SN is absent (i.e., in the direction of the slow phase of the SN) and gaze is stable.

Case 1 represents a classic example of AL in a unilateral, peripheral, vestibular system impairment. The right-beating SN shows a SPV that is greatest in magnitude when the patient gazes in the direction of the fast phase, and the nystagmus velocity attenuates when the patient’s gaze is directed toward the slow phase. The key element that is not described in AL that is “diagnostic” in this case is the augmentation of the SN when vision is denied. For this patient, the SPV of the SN in the vision-denied condition increases sixfold over that generated at center gaze and twofold over that generated in the gaze-right condition.

Case 2 also demonstrated features similar to those of Case 1. However, in the vision-denied condition, the SN increased only marginally (i.e., intermediate between the SPV observed at midline gaze and at right gaze). In this case it was clear that the “gazing” was the origin on the nystagmus. Thus, the patient had an asymmetrical (i.e., unilateral) GEN. That is, the effect of tumor compression on the brainstem and cerebellum produced a unilaterally leaky NI. Further evidence that the nystagmus was caused by central nervous system disease and not the peripheral vestibular system was the symmetrical caloric responses, and normal phase and gain measures on rotational testing. Thus, in this case, the nystagmus was generated from a central gaze-holding impairment, not from peripheral vestibular system impairment (Leigh and Zee,

1999). Asymmetric horizontal GEN *always* is caused by a brain lesion usually involving the brainstem or cerebellum when the lesion is focal. These cerebello-pontine angle tumors usually are large. Very large tumors can compress the cerebellum and brainstem but often do not cause purely central vestibular dysfunction. This patient did not have any involvement of the peripheral end organ or nerve. Her symptoms were purely from central compression. As occurred in this case, the direction of the fast phase of the GEN usually denotes the side of the lesion and/or the side where the effects of compression are most severe (Baloh and Honrubia, 2001).

SUMMARY

The two case reports illustrate that what appears to be a spontaneous nystagmus that follows Alexander’s Law is not, in and of itself, diagnostic for peripheral vestibular system impairment. It is the augmentation of the SN in the vision-denied condition that becomes diagnostic for an acute or post-acute, unilateral, peripheral vestibular system impairment. Likewise, it is the attenuation or lack of augmentation of the SN that occurs in the vision-denied condition that defines asymmetric gaze-evoked nystagmus that occurs due to significant central nervous system disease affecting the brainstem and cerebellum. The behavior of the eye movements in the vision-denied condition can be observed best with video-nystagmography eye movement recording techniques.

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