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Vestibular Case Studies: A Case of CVA and Vestibular Paroxysmia

Jennifer Banovic B.S.
Illinois State University, jbanovi@ilstu.edu

Nicholas Stanley Ph.D. Au.D.
Illinois State University, nsstanl@ilstu.edu

Jennifer Banovic, B.S.

For Fulfillment of Doctor of Audiology Degree

Illinois State University, Normal, Illinois

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Abstract 1

Introduction: One of the leading causes of centrally based vestibular impairment is a cerebrovascular accident (CVA). Patients typically present with limb or facial weakness, dizziness and imbalance, impaired vision, and speech or language dysfunction, however, these symptoms can vary depending on the site of lesion. Case presentation: An 85-year-old female presented with dizziness, bilateral tinnitus, aural fullness, and aphasia, occurring after she suffered a CVA. Three months after the CVA, she was seen by a balance clinic for a videonystagmography (VNG) and audiologic evaluation. Discussion: CVAs can alter the blood supply to the inner ear and its nerve support, leading to neurotologic symptoms. Consideration of the stroke location, like the AICA or PICA, can help lead to an accurate diagnosis and therapy recommendations. Conclusion: Magnetic resonance imaging is strongly encouraged for CVA patients presenting with dizziness. Locating the site of lesion before vestibular testing can provide better patient outcomes and recommendations.

Key words: Vestibular, CVA, stroke, central nervous system, audiogram, VNG

Case Presentation 1

The Effects of a CVA on the Vestibular System: A Case Report

Introduction

Nearly 12% of vestibular patients have an impairment rising from the central nervous system (3). Of that 12%, CVAs are the most common causes of centrally based vestibular impairments. Depending on the site of lesion, otologic symptoms can vary. While strokes in a variety of cerebral sites can affect your inner ear, strokes that infarct the anterior inferior cerebellar artery (AICA) or the posterior inferior cerebellar artery (PICA) typically alter the neural and vascular flow to the inner ear system (2).

The PICA, which is sourced from the vertebral artery, has two common branches stemming from the vertebral artery trunk: the lateral branch and medial branch. An infarction of the medial PICA typically presents purely vestibular symptoms, including the following: severe vertigo, nausea, vomiting, dysarthria, and postural instability. Lateral PICA infarctions can lead to the following symptoms: dysmetria or ipsilateral limb, gait disturbance, and hypotonia of the ipsilateral limbs without the presence of severe vertigo or vomiting (2).

The AICA supplies blood flow to both the central and peripheral vestibular systems. A typical infarction of the AICA presents combined peripheral and central vestibular weaknesses, sensorineural hearing loss, gait ataxia, limb and facial sensory loss, and cerebellar dysmetria. Hearing loss in patients with AICA infarctions is typically permanent, however the vestibular symptoms can improve with central compensation (2). Because the AICA supplies blood to multiple structures that can stimulate vertigo, determining the exact site of lesion can be difficult. However, prolonged vertigo typically stems from an ischemia in both the central and peripheral structures (2).

Typically, oculomotor testing in CVA patients is abnormal (1). Through an MRI, the identification of an AICA, PICA, or other cerebral lesion can help clarify symptoms and point to better therapy recommendations after a vestibular impairment is diagnosed. It is recommended that an MRI be conducted in these patients before they are seen for vestibular testing.

Case Presentation

An 85-year-old female visited a balance clinic three months after suffering a CVA. She had been receiving physical therapy services at the clinic since the stroke occurred. She presented with dizziness, bilateral tinnitus, aural fullness, and aphasia. The patient described her dizziness as lightheadedness and imbalance, especially when walking. She reported that her symptoms used to last all day since the stroke occurred but had become intermittent in the weeks leading up to the appointment. The patient described "cricket-like" tinnitus that she reported to be louder on the right. She also described feeling "stopped up" ever since the CVA. The patient also had a life-long right lazy eye and history of cataract surgery. The patient had no prior history of an MRI being completed.

Pure tone air and bone conduction testing found normal hearing from 250 Hz to 2000 Hz in the left ear, sloping to a mild and moderate hearing loss from 3000 Hz to 8000 Hz. In the right ear, testing revealed a mild conductive loss at 500 Hz, sloping to a mild sensorineural hearing loss from 1000 Hz to 3000 Hz, further sloping to a moderate mixed loss from 4000 Hz to 8000 Hz. Word recognition testing was excellent bilaterally. Tympanometry was consistent with proper middle ear function bilaterally. Results of the VNG found the following: in-direction gaze nystagmus to left and right with vision denied; spontaneous ocular flutter noted throughout test and more pronounced vision denied; reduced smooth pursuit to leftward targets; saccadic pursuit tended to undershoot for rightward targets; optokinetic testing was significantly reduced to the

left; and caloric irrigations revealed a significant 58% weakness to the right and borderline directional preponderance of 21% to the left.

Due to the right caloric weakness, a right peripheral pathology was diagnosed. A right central pathology was also diagnosed due to her difficulty tracking left-ward targets, both in smooth pursuit and optokinetic tracking, as well as the presence of ocular flutter. The patient was referred to an otolaryngologist due to the asymmetrical hearing loss. It was also highly recommended that she follow-up with her neurologist regarding the aural, peripheral, and central findings. Due to no previous history of an imaging study, it was recommended that the patient consult her neurologist regarding an MRI to determine the site of lesion. The patient was also encouraged to continue physical therapy, specifically vestibular retraining therapy.

Discussion

While nearly 200,000 cases of strokes occur annually, only 2.1% of those cases experience vertigo (4). While any CVA lesion can cause neurotologic symptoms, infarctions in the AICA and PICA are primarily the culprit for vertigo and acute hearing loss. Cerebellar infarctions in the PICA territory, typically present mainly vestibular symptoms, “characterized by severe vertigo, nausea, vomiting, prominent axial laterpulsion, dysarthria, and limb dysmetria” (2). Typically, AICA infarctions present with combined central and peripheral diagnosis, due to the presence of hearing loss, gait ataxia, cerebellar dysmetria, limb and facial sensory loss, and facial weakness (2).

While an MRI is needed to determine the site of lesion, vestibular and audiologic testing can determine the extent of the loss in the inner ear. VNG abnormalities often include dysfunction in oculomotor testing, as well as caloric weakness in some cases (1). By completing a comprehensive audiogram alongside a VNG, the extent of loss within the inner ear can be

determined. After completion of the diagnostic exams, accurate and beneficial recommendations can be made to help the patient rehabilitate.

Conclusion

Strokes are one of the most common causes of a central vestibular pathology (1). While an MRI is needed to determine the exact place of lesion, cerebellar strokes with an infarction of the PICA or the AICA are the most common causes of otologic symptoms (2). In the case presented, obtaining prior knowledge of an MRI could help guide testing and recommendations to streamline the patient's medical care. With the knowledge of the site of lesion, certain otologic symptoms could be explained and proper recommendations provided. Completing an MRI before conducting audiometric and vestibular testing can help determine the extent and cause of damage.

Abstract 2

Introduction: Vestibular Paroxysmia (VP) is a rare disease with symptoms such as episodic positional vertigo, tinnitus, and unilateral audiometric findings. Case presentation: A 68-year-old female reported to her local otolaryngologist with unilateral hearing loss in her right ear and vestibular symptoms. An MRI revealed VP, also known as a vascular loop, on her right side. Discussion: It is important to utilize a thorough case history in the diagnostic protocol for VP. An accurate case history and patient report can be the most important diagnostic tool in identifying the cause of symptoms. Conclusion: While diagnostic tests can at times definitively identify vestibular disorders, it is also important to consider the patient's reported symptoms when investigating their dizziness. In this case study, an accurate and thorough case history helped lead to an MRI which identified VP when diagnostic tests could not.

Key words: Vestibular, Vestibular Paroxysmia, vascular loop, case history, VNG

Vestibular Paroxysmia: A Case Report

Introduction

Vestibular Paroxysmia (VP), otherwise known as a vascular loop, is a rare disease which can affect patients through symptoms of chronic positional vertigo, tinnitus, and unilateral audiometric findings (5). This occurs through a constriction of the anterior inferior cerebellar artery (AICA) around the eighth cranial nerve inside the internal auditory meatus (7). VP is considered a rare disease, contributing only 3%-4% to vestibular diagnoses a year. Very few VP case studies and research articles have been published leading to discrepancies between data and diagnostic factors (6, 8). These discrepancies emphasize the need for a stringent case history to assist in the diagnostic investigation. This method of case history analysis can lead to better patient outcomes.

Case Presentation

A 68-year-old female presented to the local otolaryngology clinic with complaints of right sided hearing loss in addition to episodic dizziness. She reported that dizziness was often triggered by movements, such as rolling over in bed or quick head turns, and lasted less than 2 minutes in intensity. She also reported a history of headaches, but denied aural fullness, otalgia, facial twitching, and persistent tinnitus. She was referred to the clinic's audiology department to complete a comprehensive audiologic evaluation before returning to the clinic's otolaryngologist. Comprehensive diagnostic protocol at this clinic involved acoustic reflex thresholds, tympanometry, pure tone air and bone audiometry, speech recognition testing, and word recognition scores using monitored live voice presentation of the W-22 word lists.

Pure tone air and bone conduction testing in the left ear revealed mild hearing loss rising to normal hearing at 500 Hz, precipitously sloping to a severe sensorineural hearing loss at 6000

Hz. Audiometric testing in the right ear revealed mild to moderate sensorineural hearing loss sloping at 6000 Hz to a profound hearing loss (Figure 1). A strong asymmetry, with the right ear being worse, was present. Speech recognition thresholds were 5 dB HL in the left ear and 35 dB HL in the right ear. Word recognition scores were 100% in the left ear at 60 dB HL, and 84% in the right ear at 85 dB HL with 65 dB HL of contralateral masking. Acoustic reflex thresholds at 500 Hz, 1000 Hz, and 2000 Hz were present both ipsilaterally and contralaterally in both ears. Tympanometry results were Jerger type A bilaterally, revealing normal compliance, pressure, and volume for both ears. After completion of the comprehensive audiologic evaluation, it was suggested by both the audiologist and otolaryngologist that the patient should return for additional neurodiagnostic auditory brainstem response (ABR) and videonystagmography (VNG) testing.

The neurodiagnostic ABR protocol was as follows: 90 dB nHL click stimulus with rarefaction polarity with a rate of 13.3/s, then increased to 81.1/s, followed by an 80 dB nHL click stimulus with rarefaction polarity at 13.3/s. ABR findings showed no abnormalities in latency or morphology (Figure 2). Following the neurodiagnostic ABR, a VNG was completed. Ocular motor testing showed normal central findings. The head shake and Dix Hallpike maneuver also revealed no instances of positional nystagmus. Bithermal caloric irrigation revealed a 22% unilateral weakness to the left, which was considered normal under the clinic's 25% abnormality threshold (Figure 3). Following the clinic's protocol, this patient was referred for an MRI because of reported symptoms relevant to an acoustic neuroma. At the time, an acoustic neuroma was suspected due to the unexplained asymmetry in her hearing, as well as, her dizziness.

MRI imaging revealed that the right AICA protruded through the right internal auditory canal causing neurovascular compression of the eighth cranial nerve. It was explained to her that this vascular loop was likely the cause of her asymmetric hearing loss as well as her positional vertigo. She was counseled on the results and chose to pursue hearing aids as a treatment option at that time. The patient did not wish to pursue treatment with pharmaceutical drugs such as carbamazepine which has been shown to decrease all auditory and vestibular symptoms (6).

Discussion

Neurovascular compression of the eighth cranial nerve can lead to vestibular paroxsymia. While previously known as "disabling positional vertigo", the name vestibular paroxysmia was adopted to be more specific of symptoms, as the former term accompanied vestibular characteristics seen in other conditions such as Meniere's disease or Benign Positional Postural Vertigo (BPPV) (5). VP occurs when the seventh or eighth cranial nerve is compressed inside the internal auditory meatus by an artery or vein. This compression causes a "bottleneck effect" of pressure inside the internal auditory canal and restricts blood flow and nerve stimulation to the inner ear. While seventh nerve compression can occur, MRI imaging shows eighth nerve compression in 95% of cases (5). In addition to this, while the PICA, vertebral artery, or a vein have been known to cause this nerve compression, the AICA seems to be the most common culprit of VP (5). While some studies show controversial findings on whether the presence of a vascular loop is the culprit of vestibular and auditory abnormalities, diagnostic investigation reveals no other explanation for the cause of symptoms (7). In addition, treatment with sodium-channel blockers such as carbamazepine, which targets the anatomical causes of VP, has been proven to lessen the degree of auditory and vestibular effects, further verifying that VP is the root of issues (6).

Certain diagnostic criteria must be present for VP to be considered. Diagnostic criteria is as follows for probable VP: the patient must endure at least five attacks of spontaneous vertigo a day, each lasting less than five minutes; symptoms must be spontaneous or provoked by body movements; the patient must present "stereotyped phenomenology"; and the symptoms must not be explained through another diagnosis (6). VP occurs either at an early age due to vertebrobasilar vascular anomalies, or between the age of 40 and 70 when increasing atherosclerosis and stronger pulsations due to arterial hypertension cause vascular elongation (5). Studies have also shown that men are twice as likely to have VP than women (5).

While some cases of VP can be suspected through a vestibular battery and case history, it is important to utilize an MRI in the diagnostic protocol. An MRI can help visualize the presence of a loop and lateralize the disorder. However, some studies show that the presence of a vascular loop can be a normal anatomical finding and that is important to consider the patients symptoms when pursuing a diagnosis with VP (7). Furthermore, an accurate case history can be the most important diagnostic tool in identifying the cause of vestibular symptoms.

Conclusion

While diagnostic tests can, at times, definitively identify vestibular disorders, it is also important to consider the patient's case history when investigating their dizziness. With the case study described above, an accurate and thorough case history helped lead to an MRI to identify the disorder when audiologic and vestibular diagnostic tests could not. Because VP's vestibular and otologic symptoms can imitate other conditions, it is vital to fully assess the patient's case history and complete diagnostic protocol for an accurate diagnosis and further treatment. In some cases of VP, like the case discussed in this article, the patient's case history can be a critical tool in pursuing a diagnosis when diagnostic tests can reveal inconclusive results.

References

1. Pimentel, Bianca Nunes, & Filha, Valdete Alves Valentins dos Santos. (2019). Evaluation of vestibular and oculomotor functions in individuals with dizziness after stroke. *Arquivos de Neuro-Psiquiatria*, *77*(1), 25-32.

2. Lee, H. (2009). Neuro-otological aspects of cerebellar stroke syndrome. *Journal of Clinical Neurology*, *5*(2), 65.

3. Strupp, M., & Brandt, T. (2008). Diagnosis and treatment of vertigo and dizziness. *Deutsches Arzteblatt international*, *105*(10), 173–180.

4. Rathore, S. S., Rathore, S. S., Hinn, A. R., Hinn, A. R., Cooper, L. S., Cooper, L. S., … Rosamond, W. D. (2002, November 1). Characterization of Incident Stroke Signs and Symptoms.

5. Brandt, T., Strupp, M., & Dieterich, M. (2016). Vestibular paroxysmia: a treatable neurovascular cross-compression syndrome. *Journal of Neurology*, *263 Suppl 1*, S90–S96. https://doi.org/10.1007/s00415-015-7973-3

6. Strupp, M., Lopez-Escamez, J. A., Kim, J. S., Straumann, D., Jen, J. C., Carey, J., Bisdorff, A., & Brandt, T. (2016). Vestibular paroxysmia: Diagnostic criteria. *Journal of Vestibular Research : equilibrium & orientation*, *26*(5-6), 409–415. https://doi.org/10.3233/VES-160589

7. de Abreu Junior, L., Kuniyoshi, C. H., Wolosker, A. B., Borri, M. L., Antunes, A., Ota, V. K., & Uchida, D. (2016). Vascular loops in the anterior inferior cerebellar artery, as identified by magnetic resonance imaging, and their relationship with otologic symptoms. *Radiologia Brasileira*, *49*(5), 300–304. https://doi.org/10.1590/0100-3984.2015.0069

8. Sousa, P., Costa, J., Duarte, D., & Trigueiros, N. (n.d.). Vestibular paroxysmia: A diagnosis not to forget. Retrieved March 09, 2021, from https://www.alliedacademies.org/articles/vestibular-paroxysmia-a-diagnosis-not-to-forget-10597.html

Figure 1 (Case 1)

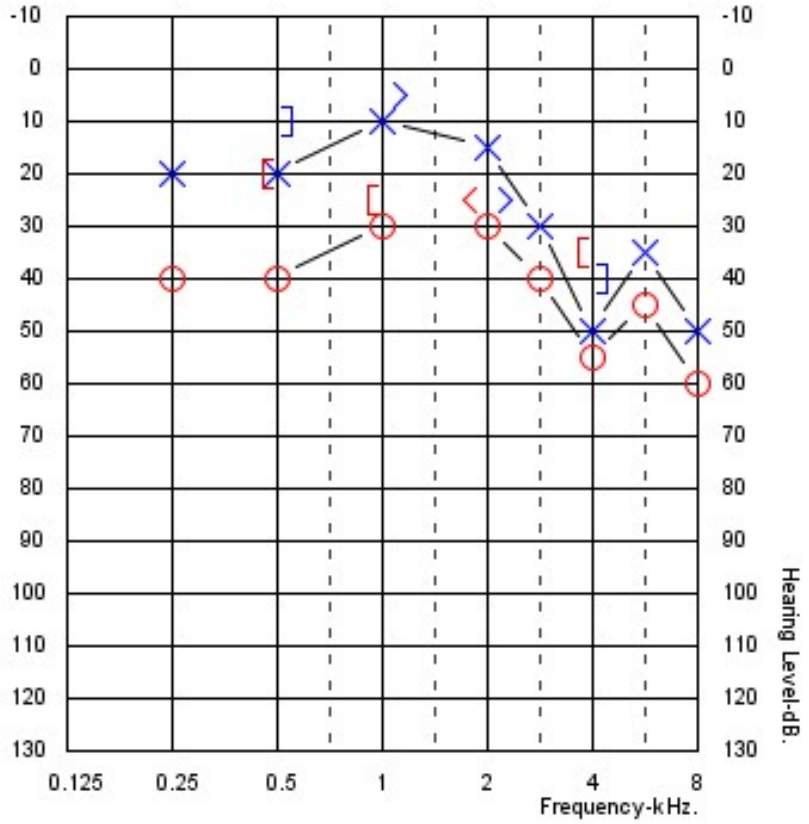

Figure 1: Audiometric testing found normal hearing from 250 Hz to 2000 Hz in the left ear, sloping to a mild and moderate hearing loss from 3000 Hz to 8000 Hz. In the right ear, findings found a mild conductive loss at 500 Hz, sloping to a mild sensorineural hearing loss from 1000 Hz to 3000 Hz, further sloping to a moderate mixed loss from 4000 Hz to 8000 Hz.

Figure 2 (Case 1)

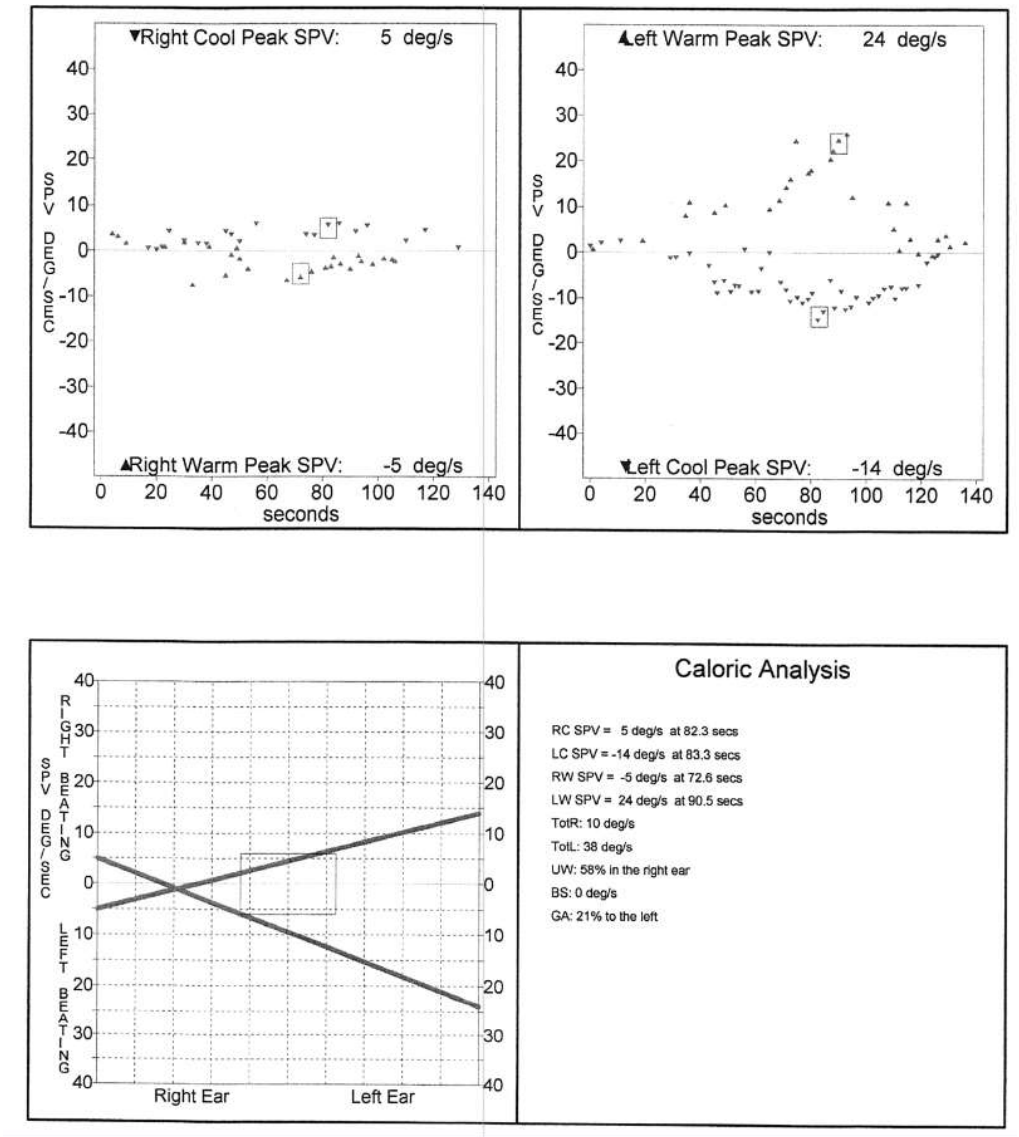

Figure 2: Caloric SPV and butterfly chart analysis depicting a right peripheral weakness of 58% and a borderline directional preponderance of 21% to the left.

Figure 1 (Case 2)

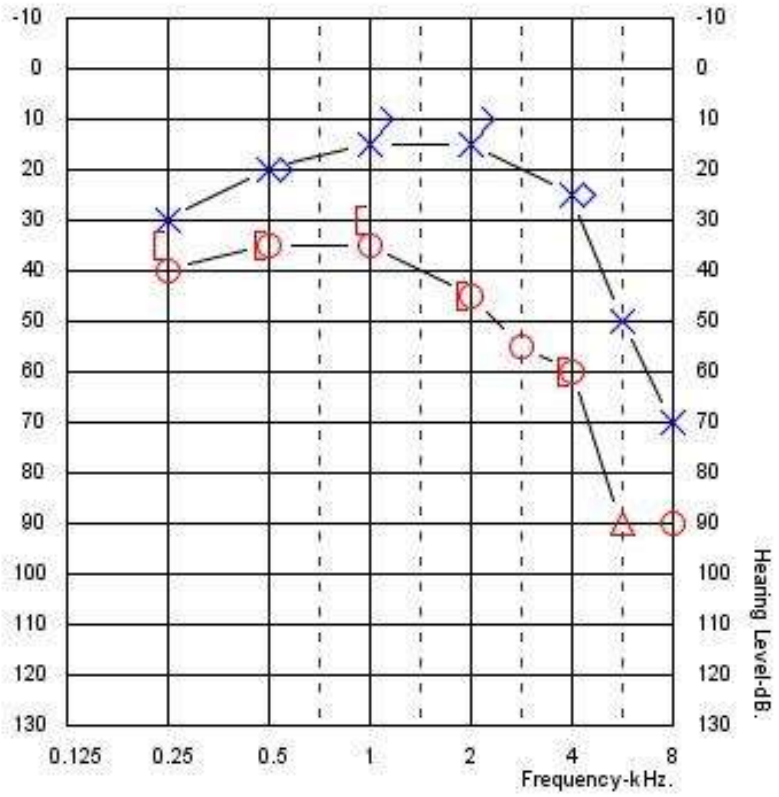

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Figure 2 (Case 2)

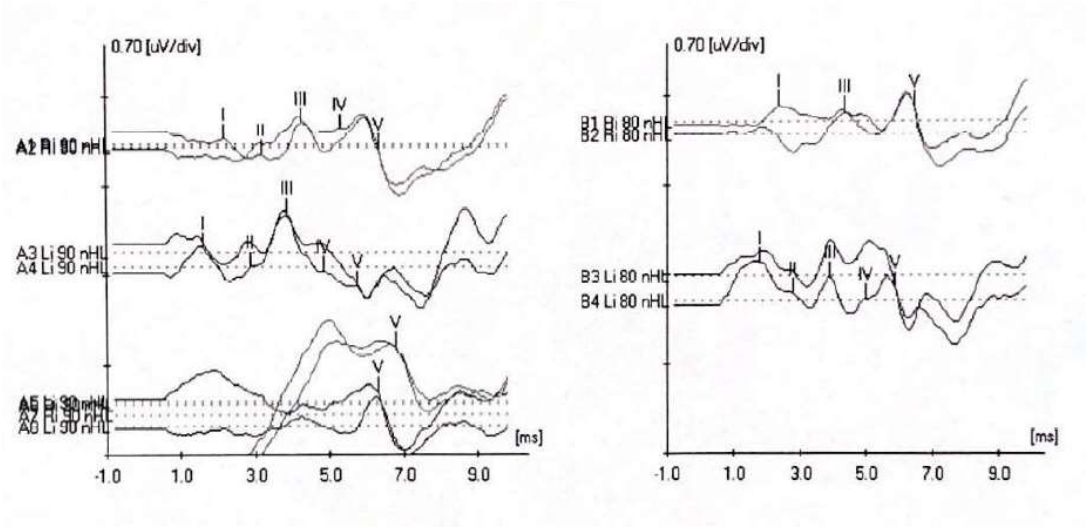

Figure 2: Neurodiagnostic recordings for CP. While wave V was marked as delayed in the right ear, no abnormalities were present.

Figure 3 (Case 2)

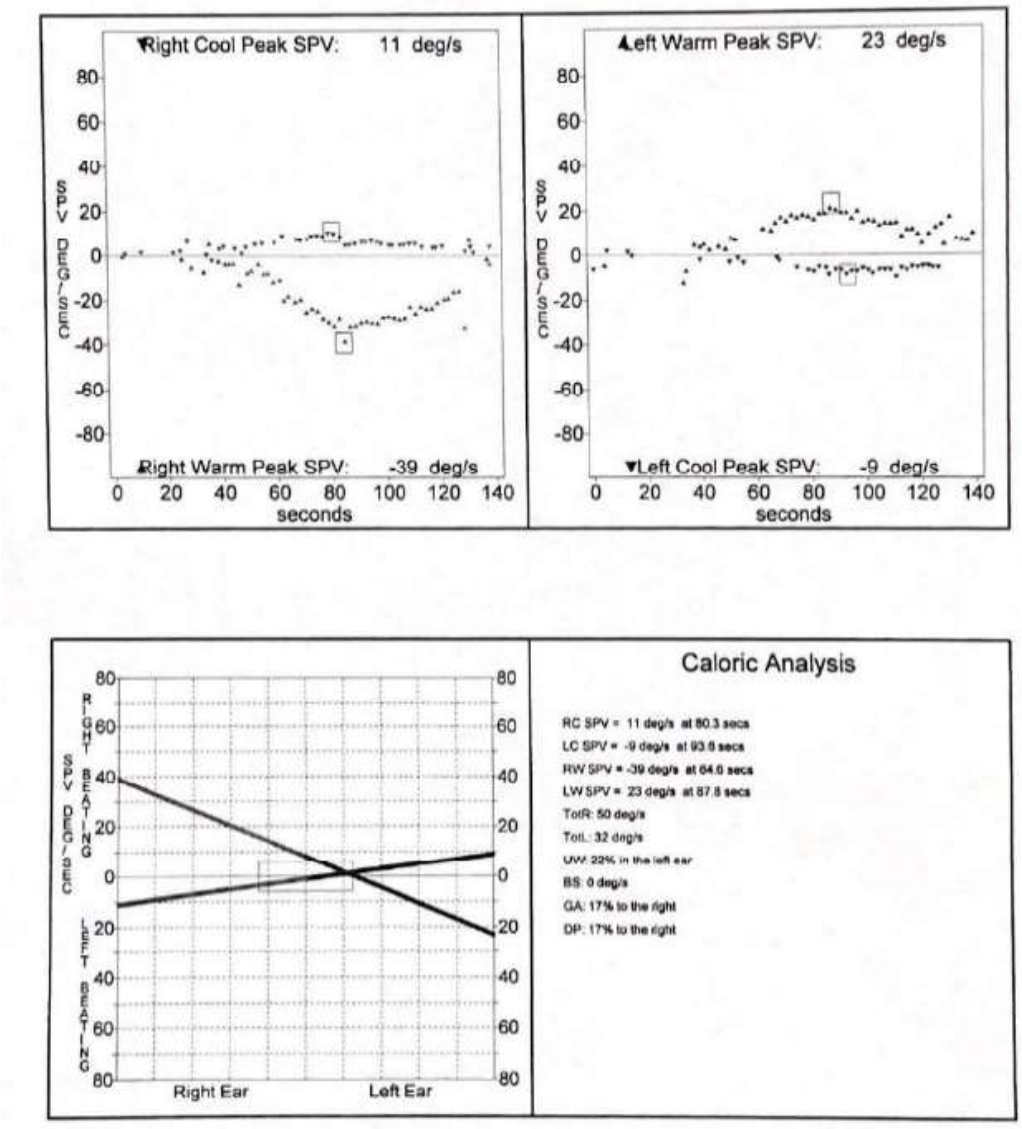

Figure 3: Caloric SPV and butterfly chart analysis depicting a 22% weakness in the left ear and directional perponderance of 17% to the right. This was considered normal under this clinic's protocol.