We are IntechOpen, the world’s leading publisher of Open Access books
Built by scientists, for scientists

6,600 Open access books available
177,000 International authors and editors
195M Downloads

154 Countries delivered to
TOP 1% Our authors are among the most cited scientists
12.2% Contributors from top 500 universities

WEB OF SCIENCE™
Selection of our books indexed in the Book Citation Index in Web of Science™ Core Collection (BKCI)

Interested in publishing with us?
Contact book.department@intechopen.com

Numbers displayed above are based on latest data collected.
For more information visit www.intechopen.com
Chapter

Otoneurological Evaluation and Rehabilitative Considerations after Head Trauma

Maria Cristina Alves Corazza, Luíza Alves Corazza and Júlia Alves Corazza

Abstract

Head injuries due to traffic accidents, falls, gunshots and blows in sports fights, among others, with or without a skull or petrosal fractures, can lead to a Traumatic Labyrinth Concussion (TLC), defined as a disorder of the peripheral vestibular system comprising vestibular, auditory and neurovegetative signs and symptoms, which can persist for weeks or months after a traumatic injury. It is often accompanied by central nervous system (CNS) concussion, manifested by objective symptoms such as tachycardia, headache, thermoregulatory instability and mydriasis; and subjective complaints such as emotional disorders, memory loss, visual disorders, insomnia, hyperemotivity and behaviour disorders. Otoneurologic examination is relevant in the identification and topographic diagnosis of vestibular disorders. This chapter will verse on symptoms, audiometric and vestibular findings in TLC, as well as rehabilitation perspectives.

Keywords: labyrinth concussion, head injury, vertigo, hearing loss, dizziness

1. Introduction

Head injuries, from mild to severe, with or without cranial or petrosal fractures, may lead to traumatic labyrinth concussion (also referred to as labyrinth commotion) (TLC) – a vestibular disorder comprising auditory, vestibular and neurovegetative symptoms which may last up to weeks or months.

TLC may be concomitant to central nervous system (CNS) injury (diffuse axonal injury in brainstem/cerebellum, post-traumatic vestibular migraine, post-concussion syndrome) [1, 2] with autonomic signs, such as tachycardia, headache, thermoregulatory instability, mydriasis, as well as memory loss, behavioural changes, emotional lability, executive dysfunction, and gait disorders.

Otoneurologic evaluation is paramount in the identification and diagnosis of vestibular disorders.

This chapter will verse on symptoms, audiometric and vestibular findings in TLC, as well as rehabilitation and treatment perspectives.
2. Historical considerations

The most recent definition of Traumatic Labyrinth Commotion is that of an auditory or vestibular dysfunction in the absence of a temporal bone fracture (TBF) [3], still, its causes remain poorly understood. For a long time, post-traumatic vestibular symptoms were regarded as psychogenic, which explains the lack of established evaluation protocols and rehabilitation perspectives [4, 5].

Several pathologists have searched the correct definition and cause of traumatic labyrinth commotion, one of the first being Samuel Moos, in 1871 Prussia (nowadays, Germany) in a post-mortem assessment of a soldier who had suffered a glancing gunshot wound to his left mastoid, developing consequent hearing loss, due to a labyrinth haemorrhage; other reported possible mechanisms are cochleovestibular nerve traction injury and the travelling pressure wave theory, causing ciliary loss and cochlear damage [3].

Another possible clinical presentation is benign paroxysmal positional vertigo (BPPV) owed to dislodgement of otoconia from the macula of the utricle due to trauma, which should always be considered in patients with positional vertigo complaint after traumatic injuries, as well as utriculo-sacular injuries [2].

Romero characterised TLC as a disturbance of the inner ear with no evidence of macroscopic or radiological lesions, associated or not to central nervous system ailments, manifesting with vertigo, hypoacusis and tinnitus [6].

Also, there was a time in which it was believed labyrinth commotion was only related to head trauma severe enough to cause loss of conscience; yet, evidence has shown mild to moderate blunt trauma might lead to permanent neurosensorial hearing loss, compromising frequencies from 3.000 to 8000 Hz [7] or 4000 to 8000 Hz [8], as seen in noise-induced hearing loss [9, 10].

In 1976, TLC was regarded by Ganança and cols. as a peripheral syndrome, with the intensity of symptoms depending on the severity of the trauma, and that labyrinth areflexia was associated with mastoidal fracture, even when radiological evidence might have been absent, and traumatic facial palsy would be commonly found, often needing surgical correction [11].

This presentation is common in the context of commotions in the occipital region, leading to transversal temporal bone fractures (20%) or parietal/temporal commotions, leading to longitudinal fractures (80%). In transversal fractures, vestibular impairment tends to be unilateral; but may be bilateral, due to countercoup mechanism; in longitudinal fractures, anatomical damage usually comprises middle ear structures, with hearing loss, and vestibular symptomatology as a result of commotion of the membranous labyrinth [12, 13].

In Berman and cols’ sample, most patients with vertigo after head trauma presented with post-concussion syndrome (PCS): headache, irritability, memory loss, autonomic symptoms, vertigo, hyperacusis, fatigue, vision changes, disturbances in balance, confusion, dizziness, insomnia, neuropsychiatric symptoms and difficulty with concentration [1, 14].

The ICD-10 defines PCS symptoms above persisting longer than 3 weeks, although most patients recover in the first 7 to 10 days following an injury [14]. Post-concussion syndrome pathophysiology includes metabolic, autonomic nervous system damage, and microstructural injuries to the brain, leading to structural and microbiological changes, resulting even in increased atrophy and regional volume loss, in several cases [15].
Vertigo, in this context, might have been related to PCS, to TLC alone, to perilymphatic fistulae (with/without need of posterior surgical intervention) or to psychological factors (due to or worsened by these) [1, 16].

Jerger & Jerger observed sudden onset of hearing loss, fluctuating over the first 3 weeks to 6 months, with the persistence of acute frequencies impairment, albeit recovery of hearing thresholds to normal levels [9].

Type of hearing loss – neurosensory, conductive or mixed – depended on the mechanism of trauma, as well as lateralization – unilateral or bilateral [9]; tinnitus may last throughout the first three or four months or become permanent [17].

Word recognition performance varied as well: cochlear lesions result in proportional impairment considering pure-tone audiometry and word recognition, while in retro cochlear lesions, word recognition is worsened disproportionally to hearing loss. In pure central auditory system disturbances, pure-tone audiometry and word recognition may be normal [9].

Acoustic immittance is undisturbed in cochlear and retro cochlear insults and altered in middle ear disorders [9].

Considering electronystagmography, central [10] and peripheral [18–20] patterns may be present. Positional nystagmus may be temporary, according to [21], and may disappear a year following trauma; De Clercq described a post-traumatic alternating nystagmus lasting for years [22].

3. Personal data and discussion

A study conducted by the author analysed fifty cases of traumatic labyrinth commotion due to multiple causes.

Individuals underwent comprehensive anamnesis, otorhinolaryngological exam (to discard other ear, nose and throat affections which could be confounding factors), as well as audiological and vestibular assessment. Audiological evaluation consisted of pure-tone audiometry, word recognition testing and acoustic immittance. Vestibular assessment comprised static and dynamic balance testing – through Romberg, Romberg-Barré and Unterberger testing as well as gait evaluation – and vectonystagmography.

Positional stationary nystagmus was evaluated as present (including vertigo complaint) or not present, as it follows: supine, right-ear down, left-ear-down, supine and with head tilted down 30°, and seated [23].

Spontaneous nystagmus was assessed at the frontal plane, with open and closed eyes, and semi-spontaneous nystagmus with a 30° deviation towards right and left, upwards and downwards.

Vectonystagmography was developed using BERGER – VN 106/3, with three register channels. Skin was cleaned using an alcoholic solution; on each patient, an electrode was placed using an electrolyte paste for fixation on the lateral angle of each eye and on the frontal midline, forming an isosceles triangle, which made it possible to check horizontal, vertical, and oblique eye movements, providing precise measurements of the slow component angular velocity of nystagmus.

Calibration was made using the biological calibrator BERGER CB 115 and regularity of tracings was assessed to enable comparisons between studies. In testing of spontaneous nystagmus and semispontaneous nystagmus the presence, direction, inhibiting effect of ocular fixation (IEOF) and the maximum slow component angular velocity (SCAV) of nystagmus were assessed.
A BERGER TB-1131 visual stimulator was used for horizontal optokinetic nystagmus evaluation, concerning presence, direction, maximum SCAV with clockwise and anticlockwise movement of the light source and the preponderant direction of nystagmus was calculated. Pendular tracking evaluated the presence and type of curve.

A ROVER BR-3201 rotating descending pendular chair was used to investigate pre- and post-rotatory nystagmus by the pendular swing rotatory test with stimulation of the anterior, lateral and posterior semi-circular canals: presence, direction, frequency after anticlockwise and clockwise rotation and calculation of the preponderant direction were done.

A BERGER OC-114 water otocalorimeter was used with water temperatures of 44°C and 30°C for caloric testing with the patient's head and trunk tilted backwards by 60° for adequate stimulation of the lateral semi-circular canals. Stimulation time for each ear was 40 sec per ear at each temperature (44°C and 30°C) and responses were recorded with eyes closed and after with eyes open to observe EEOF. The direction, absolute values of SCAV and calculation of the preponderant direction and labyrinthic predominance of post-caloric nystagmus.

Anamnesis found vertigo and dizziness as the principal vestibular complaints, tinnitus (26%) as the main auditory complaint, followed by hyperacusis (20%) and hypoacusis (18%). Additional symptomatic findings are in Table 1 in Appendix A.

As to audiological findings, 76% had normal results and 24% had neurosensorial hearing loss; 96% had unaltered word recognition and 90% had unremarkable acoustic immittance (Table 2 in Appendix A).

Regarding vestibular findings, the totality of individuals had normal static and dynamic balance testing, normal oculomotor calibration, and absence of spontaneous and semi-spontaneous nystagmus with eyes open; 14 (28%) had positional vertigo, 3 (6%) had positional nystagmus and 13 (26%) had closed eyes spontaneous nystagmus with SCAV over 10°/s. One case (2%) had per-rotatory nystagmus areflexia (Table 3 in Appendix A).

Symptomatologic findings were consistent with previously reported data [1, 6, 9] and more recent evidence [13, 24–26].

Normal auditory function seemed to be expected [17, 21], although Griffiths, in 1979, reported an incidence of 56% of hearing loss in his sample [27]. When present, neurosensorial hearing loss was the most prevalent, consistent with findings of numerous studies in the field [7, 9, 10, 16, 27, 28].

In our sample, vestibular findings were consistent with a peripheral deficit syndrome, as Mangabeira, Albernaz & Ganância [18] and conflicting with Kirtane’s study, which found a higher incidence of central lesions, with absence of inhibitor effect of ocular fixation and enhanced caloric responses [10]. This might be due to different mechanisms of trauma and possible brainstem/central nervous system involvement, rather than pure TLC, in their sample, given that more recent studies have supported the fact that pure labyrinth commotion is a peripheral disorder, either by damage of the inner ear or of the vestibular nerve/apparatus [1, 13, 29].

Closed eyes spontaneous nystagmus with SCAV higher than 10°/s was reported in other studies [1, 22], as well as reduced caloric responses [1, 22].

The importance of a full otoneurologic evaluation in the context of post-concussion patients was once more reasserted, as the patients might present with unremarkable evidence of balance disorders at physical examination, and, yet, their exams might be abnormal, as postulated by Haid and Graeffe [19].
4. Rehabilitation and treatment perspectives

Treatment should be tailored according to patient individual necessities, severity, and mechanism of trauma, assessed thoroughly by a comprehensive and integrated approach, involving doctors, physiotherapists, speech therapists/audiologists, psychotherapists, and patients’ families [4, 5, 26].

There is no definitive treatment for labyrinth concussion [30], as physiopathology is varied, therefore rehabilitation and treatment must be based on the patient complaint.

The first, and perhaps most important step is validation. Most patients who experience head trauma have subjective and sometimes hard-to-define complaints (take dizziness, for instance), not always well measured by objective tests, which for years led to the belief that those could be of a psychogenic nature, delaying the establishment of treatment protocols, even though of now it is known treatment should be initiated as early as possible, to mitigate pathophysiological chains involved [4, 5].

Patients with chronic vertigo are prone to work absenteeism [2], and, if present, psychiatric comorbidities are associated with poorer responses to treatment [27, 31, 32]. Screening tools, such as a full neurological and physiotherapeutic examination, as well as imaging examinations, must be used to identify the main mechanism of vertigo/dizziness, to provide specific therapeutic – if peripheral, vestibular rehabilitation may be useful; if cerebellar or due to disruptions on cerebellar pathways, coordination training should be proposed; if due to vision sequelae, vision directed therapy is indicated; if due to proprioceptive impairment, proprioception training might be appropriate; and if orthopaedic lesions are involved, specific treatment must be carried out [12, 14].

Neurotransmitters involved in vertigo pathophysiology are fundamentally Glutamate, acetylcholine and gamma-aminobutyric acid (GABA). Glutamate mediates synaptic transmission, mainly through amino-3-hydroxy-5-methylisoxazole-4-propionic acid (AMPA)–type glutamate receptors and maintains resting discharge potential and long-term modulation of stimuli through N-methyl-D-aspartate (NMDA)– type glutamate receptors. Acetylcholine appears to play an excitatory role in the vestibular nuclei and might be involved in vestibular compensation. GABA, as in other CNS structures, acts as an inhibitory neurotransmitter, as well as glycine. Noradrenaline, dopamine, serotonin and histamine are involved with less clear effects, working as modulators, especially regarding emesis [33].

Thus, pharmacological treatment of vertigo includes vestibular suppressant drugs, such as benzodiazepines, antihistamines, and anticholinergic agents, as well as calcium-channel antagonists (less used nowadays due to their known relation with drug-induced parkinsonism) [34, 35] and dopamine receptor antagonists. Although useful in the reduction of symptom intensity in the acute phase [33], their efficacy is little in post-concussion vertigo, and their use should be brief (maximum 3 weeks). Benzodiazepines may be particularly beneficial in co-morbidity with anxiety and depression, although should be briefly used, given they might lead to dependency [33]. Suggestions of use can be found in Table 4. In mild brain trauma, there are studies investigating the use of amantadine, yet the results are preliminary [5].

In case of lesions confined to the labyrinth, labyrinthectomy or vestibular nerve section might an option [36]. Hearing loss may be present and should be identified and treated, with hearing aids proper adapting, led by an experienced team of audiologists, and, in severe cases of labyrinth commotion, cochlear implantation may be required [37].
In rehabilitation of PCS, reassurance remains the major treatment, as most symptoms tend to resolve within 3 months; when headache is a main issue, medications such as amitriptyline [38], greater occipital nervous blocks [39], propranolol and indomethacin [40, 41] may be useful, depending on the characterisation of pain – as

**Vestibular Disorders After Concussion**

**Initial Symptomatic Management:**
- Vertigo
  - Benzodiazepine / Antihistamines / Anticholinergic / Dopamine Receptor Antagonists
- Headache: Depends on the characterization of pain
- Nausea / Emesis
  - Dopamine Receptor Antagonist / Serotonin 5-HT₃ Antagonist / Antihistamine

**Medical:** Neurologic, Visual, Otorhinolaryngologic, Orthopaedic and Psychiatric

**Audiologic:** Pure-tone and High Frequency Audiometry, Vocal Audiometry, Tinnitus Evaluation, Timpanometry, Acoustic Reflex Testing, Word Recognition Testing

**Vestibular Assessment:** Static and Dynamic Balance Testing, Gait Evaluation, Dix-Hallpike and Head Roll Test, Electronystagmography or Vestroystagmography, V-HIT, Posturography, Psychotherapy

**Figure 1.**
Vestibular disorder management after head trauma.
post-concussive headaches often mimic primary headaches, such as migraine, hemicrania continua and paroxysmal hemicrania.

Even patients with PCS may develop chronic vertigo or dizziness, chronic balance problems, or chronic cognitive impairment, which should also be assessed and treated.

Balance stability is one of the most multi-layered subjects in the field, given the complexity that underlies the mechanism of posture and gait control, comprising higher level processing, sensorial cues, proprioception, coordination, tone, strength, vestibular and visual cues [42]. Disruption in any of these points – neurologic, orthopaedic, psychologic, ophthalmologic, or audiologic – produces postural instability and, consequently, gait disturbances [43].

As such, the perfect balance rehabilitation should be able to evaluate all these fields, hence why a rehabilitation team is required. Posturography, kinematic evaluation, and sensorial integration in balance clinical testing are tools in the identification of the predominant deficiencies and help guide and plan therapy [12].

Otoneurologic assessment is not usually performed in the initial or follow-up of post-concussion patients [44, 45], although, as explained above, it might have benefit, including treatment planning.

A review by Nagib & Linens has brought evidence that vestibular rehabilitation (VRT), associated with light aerobic exercise or cervical spine therapy reduced Dizziness Handicap Index scores, improving quality of life and balance [46], whereas a study by Murray, Meldrum and Lennon also suggests that VRT might be useful in mild brain trauma, although more studies are necessary to fully establishment of the practice [47]. This evidence was also supported by the study of Park, Ksiazek and Olson, on VRT in adolescents who suffered sports-related concussion [25].

Wearable sensors and applications, if available, may be helpful in screening of brain injury extent through biomarkers [48], as well as monitoring physiological [49, 50], biological and gait data [51], to improve rehabilitation techniques, and prevent new trauma, as with fall detectors, detections of epileptic seizures and arrhythmias, as well as evaluate treatment efficacy [52]. Virtual and augmented reality systems have been also studied in the rehabilitation field, with promising results [53, 54], as well as audio-feedback [55].

Physical activity and aerobic exercise should always be encouraged, initially supervised, and, once the patient regains independence, unsupervised, as it correlates with autonomic regulation [24] cognitive recovery, by supporting neuroplasticity during the post-acute period [56], and re-establishing hippocampal homeostasis [57], due to activation of neuroprotective and antiapoptotic pathways, as well as mediating release of substances by bone, liver and muscle, augmenting brain production of brain-derived neurotrophic factor signalling (BDNF) [58-60], as well as improving quality of life and decreasing levels of anxiety, depression and permanent post-concussive symptoms [61-63] (Figure 1).

5. Concluding remarks

Traumatic Labyrinth Commotion may present with various otoneurological symptoms and signs, predominating vestibular over auditory alterations. Otoneurologic, audiological, neurological, and physical evaluations as well as image screening are relevant to treatment, which should be individualised and may comprise non-pharmacological, pharmacological and sometimes surgical interventions. It is suggested that vestibular rehabilitation may be useful in peripheral and central causes

5
of vertigo and dizziness, as well as physical exercise may be an ally on rehabilitation of post-concussed patients.

**Abbreviations**

TLC  Traumatic Labyrinth Concussion  
CNS  Central Nervous System  
TBF  Temporal Bone Fracture  
PCS  Post-Concussion Syndrome  
ICD-10  International Classification of Diseases 2010  
IEOF  Inhibiting Effect of Ocular Fixation  
SCAV  slow component angular velocity  
BNDF  Brain Derived Neutrophic Factor  
AMPA  amino-3-hydroxy-5-methylisoxazole-4-propionic acid  
NMDA  N-methyl-D-aspartate  
GABA  Gamma-aminobutyric Acid  
VRT  Vestibular Rehabilitation Therapy  
BPPV  Benign Paroxysmal Positional Vertigo

**A. Appendix**

<table>
<thead>
<tr>
<th>Symptom</th>
<th>No of cases</th>
<th>(%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Vertigo</td>
<td>32</td>
<td>64.0%</td>
</tr>
<tr>
<td>Dizziness</td>
<td>24</td>
<td>48.0%</td>
</tr>
<tr>
<td>Tinnitus</td>
<td>13</td>
<td>26.0%</td>
</tr>
<tr>
<td>Headache</td>
<td>11</td>
<td>22.0%</td>
</tr>
<tr>
<td>Hyperacusis</td>
<td>10</td>
<td>20.0%</td>
</tr>
<tr>
<td>Nausea</td>
<td>9</td>
<td>18.0%</td>
</tr>
<tr>
<td>Hypoacusis</td>
<td>9</td>
<td>18.0%</td>
</tr>
<tr>
<td>Ear pressure/fullness</td>
<td>8</td>
<td>16.0%</td>
</tr>
<tr>
<td>Sweating</td>
<td>7</td>
<td>14.0%</td>
</tr>
<tr>
<td>Phobias</td>
<td>7</td>
<td>14.0%</td>
</tr>
<tr>
<td>Pallor</td>
<td>6</td>
<td>12.0%</td>
</tr>
<tr>
<td>Depression</td>
<td>6</td>
<td>12.0%</td>
</tr>
<tr>
<td>Tachycardia</td>
<td>5</td>
<td>10.0%</td>
</tr>
<tr>
<td>Anxiety</td>
<td>5</td>
<td>10.0%</td>
</tr>
<tr>
<td>Sensation of “turning off”</td>
<td>4</td>
<td>8.0%</td>
</tr>
<tr>
<td>Darkening of vision</td>
<td>3</td>
<td>6.0%</td>
</tr>
<tr>
<td>Memory loss</td>
<td>3</td>
<td>6.0%</td>
</tr>
<tr>
<td>Falls</td>
<td>2</td>
<td>4.0%</td>
</tr>
<tr>
<td>Symptom</td>
<td>No of cases</td>
<td>(%)</td>
</tr>
<tr>
<td>-------------------------------</td>
<td>-------------</td>
<td>-------</td>
</tr>
<tr>
<td>Light-headedness</td>
<td>2</td>
<td>4.0%</td>
</tr>
<tr>
<td>Sleep disorders</td>
<td>2</td>
<td>4.0%</td>
</tr>
<tr>
<td>Vomiting</td>
<td>1</td>
<td>2.0%</td>
</tr>
<tr>
<td>Word recognition problems</td>
<td>1</td>
<td>2.0%</td>
</tr>
<tr>
<td>Feeling of liquid inside ear</td>
<td>1</td>
<td>2.0%</td>
</tr>
<tr>
<td>Ear pain</td>
<td>1</td>
<td>2.0%</td>
</tr>
<tr>
<td>Diplacusis</td>
<td>0</td>
<td>0.0%</td>
</tr>
</tbody>
</table>

Table 1.
Prevalence of symptoms in 50 cases of traumatic labyrinth concussion.

<table>
<thead>
<tr>
<th>Audiological findings</th>
<th>No° of cases</th>
<th>(%)</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Tonal audiometry</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Normal</td>
<td>38</td>
<td>76.0%</td>
</tr>
<tr>
<td>Neuro-sensorial hearing loss</td>
<td>12</td>
<td>24.0%</td>
</tr>
<tr>
<td>Conductive hearing loss</td>
<td>0</td>
<td>0.0%</td>
</tr>
<tr>
<td>Mixed hearing loss</td>
<td>0</td>
<td>0.0%</td>
</tr>
<tr>
<td><strong>Word recognition testing</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Normal</td>
<td>48</td>
<td>96.0%</td>
</tr>
<tr>
<td>Altered</td>
<td>2</td>
<td>4.0%</td>
</tr>
<tr>
<td><strong>Acoustic immittance testing</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Normal</td>
<td>45</td>
<td>90.0%</td>
</tr>
<tr>
<td>Altered</td>
<td>5</td>
<td>10.0%</td>
</tr>
</tbody>
</table>

Table 2.
Prevalence of audiological findings in 50 cases of traumatic labyrinth concussion.

<table>
<thead>
<tr>
<th>Vestibular findings</th>
<th>No. of cases</th>
<th>(%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Normal balance (static and dynamic)</td>
<td>50</td>
<td>100.0%</td>
</tr>
<tr>
<td>Regular eye movement calibration</td>
<td>50</td>
<td>100.0%</td>
</tr>
<tr>
<td>Symmetric optokinetic nystagmus</td>
<td>50</td>
<td>100.0%</td>
</tr>
<tr>
<td>Pendular tracking types I or II</td>
<td>50</td>
<td>100.0%</td>
</tr>
<tr>
<td>Symmetric per-rotatory nystagmus</td>
<td>37</td>
<td>74.0%</td>
</tr>
<tr>
<td>Symmetric post-caloric nystagmus</td>
<td>24</td>
<td>48.0%</td>
</tr>
<tr>
<td>Positional vertigo</td>
<td>14</td>
<td>28.0%</td>
</tr>
<tr>
<td>Post-caloric nystagmus directional preponderance</td>
<td>14</td>
<td>28.0%</td>
</tr>
<tr>
<td>Spontaneous nystagmus with closed eyes (&gt; 10°/s)</td>
<td>13</td>
<td>26.0%</td>
</tr>
</tbody>
</table>
### Vestibular findings

<table>
<thead>
<tr>
<th>Vestibular finding</th>
<th>No. of cases</th>
<th>(%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Directional preponderance of per-rotatory nystagmus to lateral semi-circular</td>
<td>13</td>
<td>26.0%</td>
</tr>
<tr>
<td>channels stimulation</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Post-caloric nystagmus unilateral hyporeflexia</td>
<td>9</td>
<td>18.0%</td>
</tr>
<tr>
<td>Directional preponderance of per-rotatory nystagmus to vertical semi-circular</td>
<td>4</td>
<td>8.0%</td>
</tr>
<tr>
<td>channels stimulation</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Positional nystagmus</td>
<td>3</td>
<td>6.0%</td>
</tr>
<tr>
<td>Bilateral post-caloric nystagmus hyporeflexia</td>
<td>2</td>
<td>4.0%</td>
</tr>
<tr>
<td>Per-rotatory nystagmus areflexia</td>
<td>1</td>
<td>2.0%</td>
</tr>
<tr>
<td>Unilateral post-caloric nystagmus areflexia</td>
<td>1</td>
<td>2.0%</td>
</tr>
<tr>
<td>Bilateral post-caloric nystagmus areflexia</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>Post-caloric unilateral nystagmus hyperreflexia</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>Post-caloric bilateral nystagmus hyperreflexia</td>
<td>0</td>
<td>0</td>
</tr>
</tbody>
</table>

**Table 3.**

*Prevalence of vestibular findings in 50 cases of traumatic labyrinth concussion.*

<table>
<thead>
<tr>
<th>Medication</th>
<th>Dosagea</th>
<th>Class</th>
<th>Side Effects and Precautions</th>
</tr>
</thead>
<tbody>
<tr>
<td>Meclizine</td>
<td>Oral tablets 12.5–50 mg every 4–6 h or chewable tablets 25 mg tid</td>
<td>Antihistamine, anticholinergic</td>
<td>Sedating, dry mouth, urinary retention, bradycardia, precaution in prostatic enlargement and glaucoma</td>
</tr>
<tr>
<td>Clonazepam</td>
<td>0.5 mg orally bid</td>
<td>Benzodiazepine</td>
<td>Mildly sedating, incoordination, hallucinations, drug dependency</td>
</tr>
<tr>
<td>Scopolamine</td>
<td>0.5 mg patch every 3 days</td>
<td>Anticholinergic</td>
<td>Topical allergy, dry mouth, bradycardia, urinary retention, precaution in glaucoma</td>
</tr>
<tr>
<td>Dimenhydrinate</td>
<td>50 mg orally, IM or IV every 4–6 h</td>
<td>Antihistamine, anticholinergic</td>
<td>Sedating, dry mouth, urinary retention, bradycardia, precaution in prostatic enlargement and glaucoma</td>
</tr>
<tr>
<td>Diazepam</td>
<td>2–10 mg (one dose) given acutely orally, IM or IV or 2 mg orally bid</td>
<td>Benzodiazepine</td>
<td>Sedating, respiratory depressant, drug dependency, contraindicated in closed angle glaucoma</td>
</tr>
<tr>
<td>Lorazepam</td>
<td>0.5 mg orally or IM bid</td>
<td>Benzodiazepine</td>
<td>Mildly sedating, incoordination, hallucinations,</td>
</tr>
</tbody>
</table>
### Medication Dosage

<table>
<thead>
<tr>
<th>Medication</th>
<th>Dosage*</th>
<th>Class</th>
<th>Side Effects and Precautions</th>
</tr>
</thead>
<tbody>
<tr>
<td>Metoclopramide</td>
<td>10 mg orally tid or 10 mg IM</td>
<td>Dopamine antagonist, stimulates, upper gastrointestinal motility</td>
<td>Drug dependency, contraindicated in closed angle glaucoma</td>
</tr>
<tr>
<td>Promethazine</td>
<td>25 mg orally every 6–8 h, 25 mg rectally every 12 h or 12.5 mg IM every 6–8 h</td>
<td>Antihistamine</td>
<td>Restlessness or drowsiness, extrapyramidal reaction, contraindicated in closed-angle glaucoma</td>
</tr>
<tr>
<td>Ondansetron</td>
<td>4 mg orally or IV</td>
<td>Serotonin 5-HT3 antagonist</td>
<td>Headache, diarrhoea, fever</td>
</tr>
</tbody>
</table>

*Recommended dosage for adults. Bid = twice daily; IM = intramuscularly; IV = intravenously; tid = three times daily.

Table 4. Pharmacological options to mitigate vertigo.

### Author details

Maria Cristina Alves Corazza*, Luíza Alves Corazza† and Júlia Alves Corazza‡

1 University of Western São Paulo - College of Medicine, Presidente Prudente, SP, Brazil

2 Santa Marcelina Hospital - Department of Neurology, São Paulo, SP, Brazil

3 Federal University of Mato Grosso do Sul - College of Computing, Campo Grande, MS, Brazil

*Address all correspondence to: criscorazza40@gmail.com

†These authors contributed equally.

‡Draft review and formatting.
References


37(1):139-145. Funding Information: This work was supported by the Assistant Secretary of Defense for Health Affairs under Award No. W81XWH-15-1-0620. Opinions, interpretations, conclusions and recommendations are those of the author and are not necessarily endorsed by the Department of Defense. Copyright: © Copyright 2020, Mary Ann Liebert, Inc., publishers


