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ACUTE PERIPHERAL VESTIBULAR DEFICITS AFTER WHIPLASH INJURIES

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We report 3 patients who had acute peripheral vestibular dysfunction minutes to hours after a car collision with whiplash injury without head trauma. The accident was a frontal collision in 1 case, a rear impact in the second, and lateral in the third. All patients complained immediately of cervicalgia, headache, acute vertigo with a sensation of erroneous body movements, and slipping of image with head movements. A sudden sensation of tilting of the environment when driving, tinnitus, and hyperacusis were also described. The otoneurologic findings showed bilateral canalolithiasis in 1 patient and an acute peripheral vestibular deficit in 2 patients. Tilt of the subjective visual vertical was measured in all patients. Cerebral magnetic resonance imaging yielded normal findings. As angular and linear accelerometers, the vestibular organs are directly exposed to high forces generated by whiplash mechanisms. Vertigo generated by peripheral vestibular lesions is probably underestimated in whiplash injuries and may often be incorrectly attributed to cervical or cerebral lesions.

KEY WORDS — otolith organ, semicircular canal, subjective visual vertical, vertigo, whiplash injury.

INTRODUCTION

Biomechanically, whiplash is a dynamic and inertial event that is not caused by a direct blow to the neck or head.[1] It corresponds to an acceleration-deceleration mechanism of energy transfer to the neck. The most common causes are car accidents such as rear-end, frontal, or lateral collisions. Such impact might generate bony and/or soft tissue injuries of the head and neck ("whiplash injuries"), which in turn may lead to a variety of clinical manifestations ("whiplash-associated disorders").[2] The symptomatology is often polymorphous. The most common complaints are cervicodynia, headache, and scapulodynia. Dizziness and vertigo are reported in 25% to 50% of cases, depending upon the study.[3,4] Auditory disorders such as tinnitus and hearing impairment are described in 14% and 5% of cases, respectively.[4] More complex complaints are reported: memory disorders, concentration disorders, and visual disturbances in 31%, 34%, and 24%, respectively.[4] Otoneurologic findings of 3 patients with an acute peripheral vestibular deficit beginning some minutes to hours after a car accident with whiplash injury are reported and discussed.

PATIENTS AND METHODS

All patients underwent a complete otoneurologic examination including history, clinical vestibular examination, pure tone audiogram, brain stem auditory evoked potentials (BAEPs), electronystagmography (ENG), and measurements of subjective visual vertical (SVV) by the monocular method of modified Maddox glasses as described previously.[5]

Electronystagmography consisted of recording spontaneous nystagmus with (light) and without (darkness) visual fixation; positional nystagmus with the head in hyperextension, then turned to the right and to the left (positions of Rose); and optokinetic nystagmus at speeds of 25°/s, 50°/s, and 75°/s (rotation to left and right) with whole retinal field stimulation. This was followed by an examination of smooth pursuit, a rotatory pendular test (undamped rotation of 360° in 20 seconds; sinusoidal frequency of 0.05 Hz with a peak velocity of 60°/s) with (light) and without (darkness) visual fixation suppression, and a bithermic caloric test with recordings of nystagmus duration after irrigation of each ear for 20 seconds with 20 cm^3 of water at 44°C and 30°C and with ice water if needed. The corneoretinal potentials were recorded for all examinations simultaneously on both eyes with horizontal and vertical leads. Criteria of abnormality were defined as follows: presence in darkness of horizontal (≥1 Hz) spontaneous nystagmus, and rotatory, vertical positional nystagmus; irregular smooth pursuit, irregularity, and gain of <50% of the optokinetic nystagmus (normal value, 100%); and asymmetry of nystagmic responses (side difference ≥ 25%) to caloric and rotatory pendular testing.

Case 1. A 57-year-old man was the seat-belted driver of an automobile during a frontal collision that occurred at a speed of approximately 100 km/h. Im-

mediately after the impact, he complained of cervicodynia and positional transient vertigo on head rotation toward the left. During the following days, he described a feeling of erroneous movements on driving the car, particularly when executing short curves, as well as an episode of subjective vertical tilt of the environment toward the left during a rear maneuver with his car. During the following weeks and months after the accident, he suffered from repeated episodes of vertigo with dizziness, nausea, vomiting, and sensations of images slipping during head and body movements, as well as sensations of erroneous movements. He also reported difficulties of concentration at his workplace, as well as disturbances of comprehension in discussions during meetings. These problems disappeared progressively after several weeks. Otoneurologic examination was performed 2 months after the accident.

Clinical vestibular examination showed transient geotropic rotatory nystagmus during the Hallpike maneuver to the left and transient upper vertical nystagmus with a geotropic rotatory component for the Hallpike maneuver to the right. The SVV was tilted 5° toward the left. The first ENG showed normal smooth pursuit, decreased gain (34%) of optokinetic nystagmus at 75°/s during rotation toward the left, a preponderance of the left nystagmus (44%) during rotatory pendular testing, and symmetric caloric responses at 44°C and 30°C (side difference, 11%). Audiological findings revealed a high-frequency sensorineural hearing loss on the left side, which had been known for several years, and normal hearing in the other ear. Brain stem auditory evoked potentials and findings on cerebral magnetic resonance imaging (MRI) were normal. No persistent cervical disorder was found on follow-up clinical examination. Eighteen months after the accident, the vertigo had disappeared and the follow-up ENG findings were normal.

Case 2. A 22-year-old woman was the seat-belted driver during a rear-end collision that occurred with an impact speed of about 60 km/h while her car was stopped at a red light. Three hours after the event, she complained of dizziness, slipping of images with head and body movements, and mild cervicodynia. Several hours later, during the night, she complained of acute vertigo with ataxia and vomiting, as well as hyperacusis and tinnitus on both sides. She also complained of concentration disturbances, especially on reading, for several weeks after the accident. During examination showed a spontaneous right second-degree nystagmus, a permanent positional right nystagmus during the Rose maneuvers, irregular smooth pursuit, decreased gain of optokinetic nystagmus to 30% and 10% at 50°/s and 75°/s, respectively, during rotation toward the right, a preponderance of right nystagmus (side difference, 42%) during rotatory pendular testing, and left areflexia during caloric testing at 44°C and 30°C (side difference, 100%; Fig 1A). The SVV was tilted 5° toward the left. The cerebral MRI findings were normal. No persistent cervical disorder was found on follow-up clinical examination.

The acute dizziness episodes decreased progressively and disappeared after several weeks. However, erroneous perception of movements such as a feeling of attraction toward the left remained, especially during quick changes of body positions. Five months after the accident, the follow-up ENG showed normal smooth pursuit, persistent decreased gain of optokinetic nystagmus of 44% and 15% at 50°/s and 75°/s, respectively, during rotation toward the right, and hyporeflexia of the caloric response in the left ear (side difference, 32%). The rotatory pendular test results were normal (side difference, 20%; Fig 1B).

Case 3. A 56-year-old woman was the seat-belted driver during a lateral collision that occurred at a speed of about 50 km/h while her car was stopped at a red light. Immediately after the impact, she complained of headache and cervicodynia. During the following days, she described an acute dizziness and a feeling of erroneous movements on walking. During the following months, she suffered from recurrent positional vertigo episodes with nausea, as well as a sensation of erroneous movements on walking. Since the accident, she has suffered from problems with concentration, difficulties with memory and ideation, and difficulty falling sleep.

Otoneurologic examination was performed 14 months after the accident. Clinical vestibular examination showed transient right nystagmus during the Hallpike maneuver to the left. The pure tone audiogram and BAEPs were normal. The ENG showed a permanent positional right nystagmus during the Rose maneuver to the right, an irregular smooth pursuit, a preponderance of right nystagmus during rotatory pendular testing (side difference, 30%) with a subtotal visual suppression of the per-rotatory nystagmus, and mild left hyporeflexia on caloric testing at 44°C and 30°C (side difference, 29%). The SVV was

diometry and BAEPs were normal. The first ENG

points of tenderness on the left side.

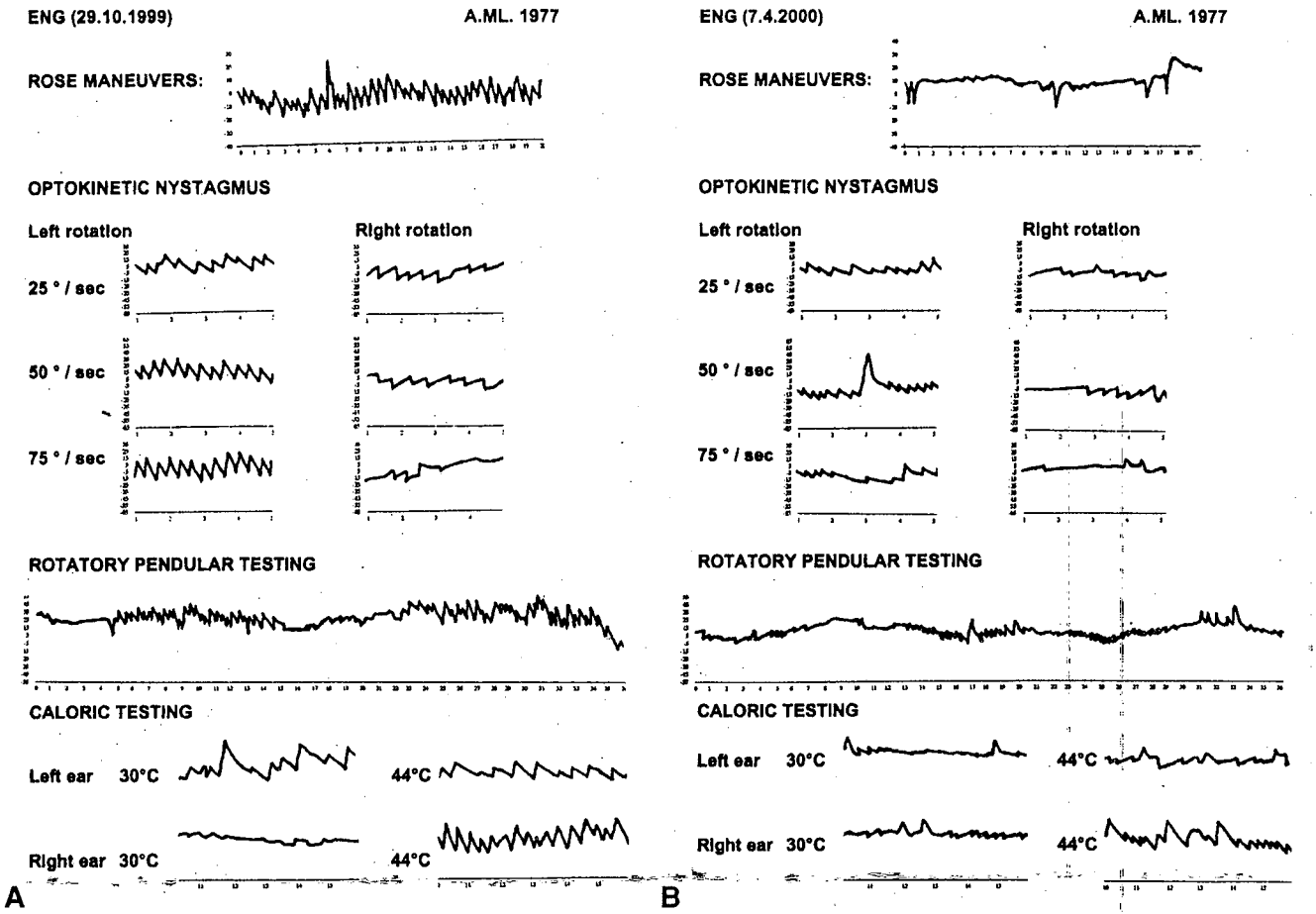

Fig 1. (Case 2) Electronystagmographic findings **A)** 2 days and **B)** 5 months after rear-end collision.

DISCUSSION

In 1995, the Québec Task Force on Whiplash-Associated Disorders proposed a classification of whiplash injuries into 4 grades depending on the neck's symptoms (see Table[6]). Grade I corresponds to complaints of neck pain without physical signs, and grade IV neck pain is associated with cervical bone fracture or dislocation. The 2 intermediate grades, II and III, correspond to neck complaints associated with musculoskeletal signs and neurologic signs, respectively. Auditory and vestibular symptoms such as hearing impairment, tinnitus, vertigo, and dizziness may be present in all grades of the classification.

CLINICAL CLASSIFICATION OF QUEBEC TASK FORCE FOR WHIPLASH-ASSOCIATED DISORDERS[6]

*Grade**	*Clinical Symptoms*
0	No complaint about neck; no physical sign(s)
I	Neck complaint of pain, stiffness, or tenderness only; no physical sign(s)
II	Neck complaint and musculoskeletal sign(s)

Neck pain is the most common symptom described after whiplash injury mechanisms and is reported in 88% to 100% of cases, depending on the study. Visual disturbance, auditory symptoms, and vertigo are described in 8% to 21%, 4% to 18%, and 17% to 25% of cases, respectively.[3]

From the otoneurologic point of view, Oosterveld et al[4] demonstrated that of 262 patients investigated 6 months to 5 years after a whiplash injury, 85% complained of persistent dizziness such as rotatory vertigo (50% of cases), and 35% complained of erroneous body sensations (floating sensations). Tinnitus was present in 14% of patients, and unilateral or bilateral hearing loss was reported in 5% of cases. Visual disturbances such as blurred vision and focusing impairment were described by 24% of patients. The ENG findings showed spontaneous, positional nystagmus, gaze nystagmus, and disturbances of smooth pursuit and of optokinetic nystagmus. Saccade impairments may be present more than 1 year after an accident.[7] Disturbances of the vestibulo-ocular reflex are also

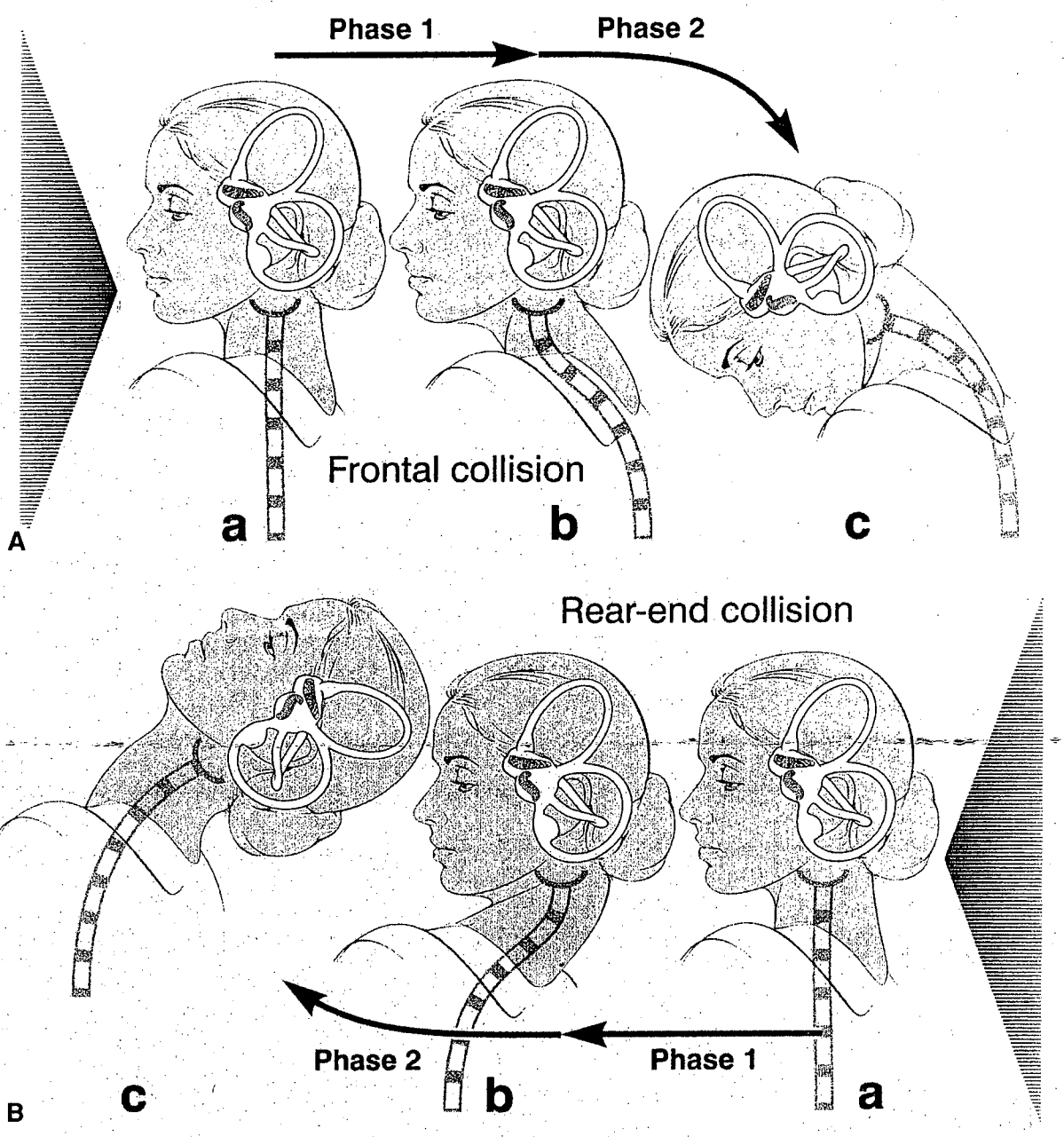

Fig 2. Positions of otolith organs during horizontal translational displacement of head in **A)** frontal collision and **B)** rear-end collision.

Otoneurologic findings in our patients included complaints of positional vertigo, sensations of erroneous movements and sudden tilting of the environment, and dizziness with ataxia that lasted for several days. These were consistent with a bilateral canalolithiasis (case 1) and an acute otolithic and horizontal semicircular canal deficit (cases 2 and 3) present

tibular deficit.[12,13] The progressive increased gain of the optokinetic response shown on the follow-up ENG might be interpreted as a sign of beginning central compensation for the peripheral vestibular deficit. In the literature, a directional preponderance of optokinetic nystagmus has also been described in cases of unilateral peripheral vestibular disorders such as

ous nystagmus generated by the left peripheral ves-

part of the ocular tilt reaction, which corresponds to

clinical signs of lesions attributed to the otolithic organs or graviceptive pathways. This is well documented as occurring after surgical vestibular deafferentation,[15-17] as well as after peripheral acute vestibular deficits such as unilateral sudden cochleovestibular loss and sudden idiopathic unilateral peripheral vestibular loss.[5,18-20] A tilt of the SVV after canalolithiasis is also measurable, but only in a small percentage of patients (17%) that is not statistically significant.[21]

Regarding case 3, it was interesting to note that the SVV remained tilted more than 1 year after the otolithic lesion. Such a finding was also observed in the long-term evolution of SVV after surgical peripheral vestibular deafferentation and interpreted as an incomplete otolithic compensation of the peripheral deficit.[22] Frontal and rear-end collisions generate a significant strain on neck and head structures. During this acceleration-deceleration event, the force acceleration might reach 5 to 30 g, depending on the speed of impact.[23] As angular and linear accelerometers, the vestibular organs directly encounter such acceleration-deceleration movements. During the initial phase, the head undergoes a horizontal translational displacement relative to the torso. This is called protraction in the case of a frontal collision and retraction in a rear-end collision (Fig 2). In both situations, the force of translation generated by the impact is recorded by the otolithic organs, especially the utriculus. Depending on the acceleration force, one can hypothesize that the "slipping" movement during the head translation generates otolith displacements or damage of the sensorineural cells, especially the hair cells. One can hypothesize that the acute or persistent dizziness and feeling of erroneous movements might be correlated to transient or permanent lesions of these structures, perhaps similar to the mechanism that has been described for the cochlear hair cells after noise exposure.

Lesions of the vestibular organs, particularly the otolithic organs, after whiplash injuries are probably underestimated by attributing dizziness and vertigo symptoms mainly to cervical damage and lesions of the central nervous system. Furthermore, the otolithic dysfunction seems not to be directly correlated to the severity of the whiplash injury. Indeed, patients 1 and 2 were classified as grade I and patient 3 as grade II on the Quebec Task Force classification system (see Table).

Various complaints such as lack of concentration, decreased efficiency, disturbance of intellectual faculties, and depression could also be manifestations of the peripheral vestibular dysfunction. Indeed, these symptoms are often clinically observed by patients after peripheral vestibular deficit that remains incompletely compensated.

A complete otoneurologic examination, including measurements of otolithic function, should be undertaken as soon as possible after the accident, that is, within the first days to weeks. The aims would be to demonstrate the presence of an acute peripheral vestibular lesion in order to have objective findings in case of possible future litigation and to treat the peripheral vestibular dysfunction appropriately and quickly by vestibular physiotherapeutic training.

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Vestibular Deficits after Whiplash Injuries

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USE: When trauma patients present with tinnitus, dizziness or visual field deficits

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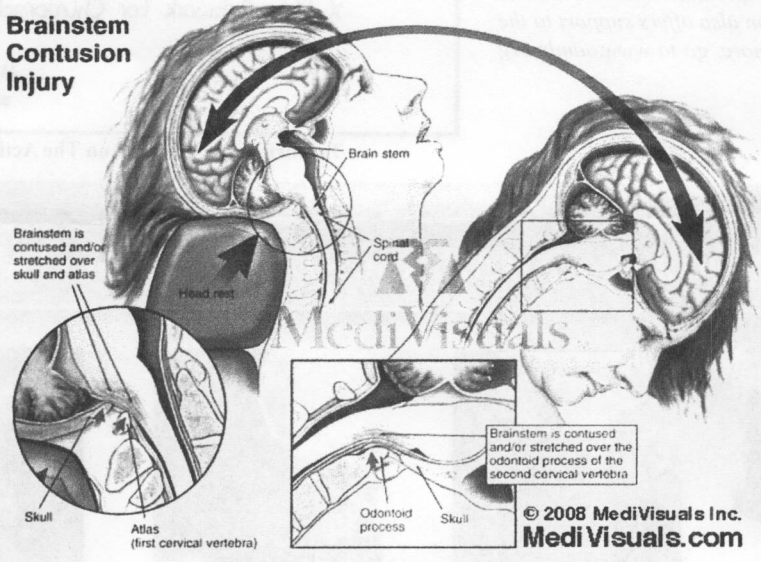
This manuscript addresses one of many important issues to keep in mind when evaluating patients that present with neck or head trauma. In the case of whiplash type injuries, it is the inertia of the trauma that causes the injury. The context of this paper was relative to whiplash injuries without direct head trauma; however, any type of acceleration/deceleration syndrome can produce symptoms of tinnitus, dizziness or visual field deficits (falls, fights, contact sports). It is reported that "dizziness and vertigo are reported in 25 percent-50 percent of [whiplash] cases."



When evaluating a patient that complains of the above symptoms, in what ways do you strive to objectify those subjective complaints? Determining the proper diagnostic test will assist you in a proper and timely medical specialty referral.

In this paper, the authors reviewed three individual cases where the patients had experienced acute peripheral vestibular dysfunction following car accidents. The following cases were discussed and, as you can see, the speed and collision type varies.

1: Case #1: "Fifty-seven-year-old male, frontal collision, speed approx 100km/hr."

2: Case #2: "Twenty-two-year-old female, rear-end collision, speed approx 60km/hr."

3: Case #3: "Fifty-six-year-old woman, lateral collision, speed approx 50km/hr."

Most importantly, "Lesions of the vestibular organs, particularly the otolithic organs after whiplash injuries, are probably underestimated by attributing dizziness and vertigo symptoms mainly to cervical damage and lesions of the central nervous system." They also stated in the article that "otolithic dysfunction seems not to be directly correlated to the severity of the whiplash injury".

It was determined that these types of symptoms can be found in all five categories of whiplash injury; therefore, it is most important to evaluate the patient properly to obtain an accurate diagnosis.

Two tests that are specific to these injuries and subjective complaints include:

1. Brainstem Auditory Evoked Potential (BAEP): This test, as the name implies, targets the brainstem area and is a recording of the electrical activity coming from the brain stem.

2: Electronystagmography (ENG): Electronystagmography is a test to look at voluntary and involuntary eye movements.

It evaluates the acoustic nerve, which aids with hearing and balance.

The paper also references that Oosterveld, et al., 1991, "demonstrated that of 262 patients investigated six months to five years after a whiplash injury, 85 percent complained of persistent dizziness such as rotary vertigo (50 percent of cases), and 35 percent complained of erroneous body sensations (floating sensations). Tinnitus was present in 14 percent of patients and unilateral or bilateral hearing loss was reported in 5 percent of cases.

Finally, it was stated that, "Lesions of the vestibular organs, particularly the otolithic organs after whiplash injuries, are probably underestimated by attributing dizziness and vertigo symptoms mainly to cervical damage and lesions of the central nervous system. A complete otoneurological examination should be undertaken as soon as possible after the accident, that is, within the first days to weeks.

Each issue, a clinical topic will be provided by Drs. Mark Studin & William J. Owens of the American Academy of Medical Legal Professionals (AAMLP), which is a national non-profit organization comprised of doctors and lawyers. The purpose of the organization is to provide its members with current research in trauma and spinal-related topics to keep the professional on the cutting edge of healthcare. Members may also sit for a Diplomate examination and be conferred a DAAMLP. The organization also offers support to the individual member's practice. To learn more, go to www.aamlp.org or call 1-716-228-3847.