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Benign Paroxysmal Positional Vertigo in Mountain Bikers

Dominique Vibert, MD; Robin C. Redfield, PhD; Rudolf Häusler, MD

We evaluated 4 men who had benign paroxysmal positional vertigo (BPPV) that occured several hours after intensive mountain biking but without head trauma. The positional maneuvers in the planes of the posterior and horizontal canals elicited BPPV, as well as transitory nystagmus. This was attributed to both the posterior and horizontal semicircular canals (SCCs) on the left side in 1 patient, in these 2 SCCs on the right side in another patient, and to the right posterior SCC in the other 2 patients. The symptoms disappeared after physiotherapeutic maneuvers in 2 patients and spontaneously in the other 2 patients. Cross-country or downhill mountain biking generates frequent vibratory impacts, which are only partially filtered through the suspension fork and the upper parts of the body. Biomechanically, during a moderate jump, before landing, the head is subjected to an acceleration close to negative 1*g*, and during impact it is subjected to an upward acceleration of more than 2*g*. Repeated acceleration-deceleration events during intensive off-road biking might generate displacement and/or dislocation of otoconia from the otolithic organs, inducing the typical symptoms of BPPV. This new cause of posttraumatic BPPV should be considered as an injury of minor severity attributed to the practice of mountain biking.

Key Words: benign paroxysmal positional vertigo, inner ear, mountain bike, otoconia, sport, vertigo.

INTRODUCTION

Transient vertigo elicited after quick changes of head position is characteristic of benign paroxysmal positional vertigo (BPPV). Its causation is attributed to disorders of vestibular organs, more specifically to dislocation of otoconia that fall from the utricle to the semicircular canals (SCCs). The dislodged otoconia are believed to generate BPPV in erroneously stimulating the angular accelerometers of the head during its movements. On the basis of his histopathologic descriptions of basophilic deposits on the cupula of the posterior SCC, Schuknecht[1] correlated the clinical signs of BPPV to his findings and named this entity "cupulolithiasis." Some years later, Parnes and McClure[2] reported free-floating particles in the endolymph of the posterior SCC during its surgical occlusion. They hypothesized that BPPV might be due also to the displacement of otoconial particles into the SCC itself; they proposed the term "canalolithiasis" to designate the clinical signs of BPPV.[2]

Benign paroxysmal positional vertigo represents one of the most common peripheral vestibular diseases that frequently occur after acute peripheral vestibular deficits, head trauma, and whiplash injuries.[3-7] Clinically, there are specific positional maneuvers that are used to induce the pathological transient rotatory, horizontal, or vertical nystagmus that suggests which SCC is involved. Benign paroxysmal positional vertigo most frequently affects the posterior SCC, and is detected with the positional maneuver described by Dix and Hallpike[3] in 1952. Benign paroxysmal positional vertigo of the horizontal SCC is less frequently reported and was first described by McClure.[8] The positional maneuver that elicits transient horizontal nystagmus consists of rolling the head from one side to the other side with the patient in the supine position. Little is known about BPPV of the anterior SCC.[9] After head trauma, one may also observe BPPV of both the posterior and horizontal SCCs.[10,11]

During the past 10 years, the increased worldwide popularity of off-road bicycling or mountain biking has also revealed an exponential rise of minor and more serious related injuries.[12-14] To our knowledge, we describe the first reported cases of acute BPPV that occurred in mountain bikers several hours after cross-country and downhill activities. We discuss the possible pathophysiological mechanism underlying this new cause of BPPV, which can be considered posttraumatic in origin.

PATIENTS AND RESULTS

The 4 healthy adult male patients were free of any previous otoneurologic symptoms. They underwent an otoneurologic examination including a history-

From the Department of Otorhinolaryngology–Head and Neck Surgery, Inselspital, University of Berne, Berne, Switzerland (Vibert, Häusler), and the Department of Engineering Mechanics, United States Air Force Academy, Colorado Springs, Colorado (Redfield).

Correspondence: Dominique Vibert, MD, Neurotology, Dept of ENT–Head and Neck Surgery, Inselspital, University of Berne, 3010 Berne, Switzerland.

taking, a clinical vestibular examination, and a pure tone audiogram.

The diagnosis of BPPV was based on the typical history and on the clinical observation of a transient nystagmus during the positional maneuvers for BPPV of the posterior or horizontal SCCs, elicited on the side of the affected ear. No auditory symptoms were reported, and pure tone audiograms were normal in all patients.

Case 1. A 48-year-old man complained of acute transient vertigo on lying down in bed. Because of the vertigo, he had to sleep with a couple of pillows to avoid the symptoms. The vertigo occurred several hours after a hard session of cross-country off-road biking with strong vibrations during downhill racing. There was neither a fall nor head trauma.

Three days after the onset of symptoms, the clinical vestibular examination showed a transient counterclockwise rotatory nystagmus during the Dix-Hallpike maneuver to the left and transient downbeat nystagmus for the Dix-Hallpike maneuver to the right. The left horizontal SCC maneuver (turning the head to the left side with the patient in a supine position) elicited a transient horizontal-geotropic nystagmus. These clinical findings were attributed to a BPPV of both posterior and horizontal SCCs on the left side. A physiotherapeutic Semont maneuver was successful; the patient became free of symptoms. One week later, the clinical vestibular examination findings were normal.

Case 2. A 32-year-old man complained of recurrent transient rotatory vertigo on turning the head to the left, as well as on getting in and out of bed for several days. The day before the onset of the first symptoms, he had performed an intensive session of cross-country off-road biking for several hours. There was neither a fall nor head trauma.

The clinical vestibular examination performed 1 week after the first symptoms showed a transient counterclockwise rotatory nystagmus during the Dix-Hallpike maneuver to the right side, as well as a transient horizontal-geotropic nystagmus elicited on the right horizontal SCC maneuver. These findings were attributed to a BPPV of both the posterior and horizontal SCCs on the right side. The symptoms disappeared after performance of a Semont maneuver. The patient mentioned sensations of floating on walking during the day after the physiotherapeutic treatment. The findings of a clinical examination performed 1 week later were normal, and the patient was free of symptoms.

Case 3. A 42-year-old man who underwent regular hard cross-country off-road bike training complained of recurrent acute transient vertigo during head movements, as well as floating sensations that had lasted for several months. The symptoms occurred regularly the day after training and were recurrent during the 24 to 48 hours thereafter. The clinical vestibular examination showed a transient counterclockwise rotatory nystagmus during the Dix-Hallpike maneuver to the right side. These findings were attributed to a BPPV of the right posterior SCC. Because of decreased symptoms during the clinical examination, a physiotherapeutic maneuver was not performed. The symptoms disappeared completely in the following days.

Case 4. A 55-year-old man complained of acute transient vertigo during head movements, dizziness, a floating sensation on walking, and nausea. The symptoms appeared about 1 hour after a very rough, speedy downhill off-road bike ride. Neither a fall nor head trauma was reported.

The clinical vestibular examination using the Dix-Hallpike maneuver to the right side showed a transient clockwise-upbeat nystagmus, which was attributed to BPPV of the right posterior SCC. Because of very limited symptoms at the time of examination, the patient did not want a physiotherapeutic maneuver. Several days later, he reported that he was free of symptoms.

DISCUSSION

Invented by Californians in the 1970s, off-road bicycling or mountain biking has become one of the most popular sport activities of the past 3 decades. The number of mountain bikers has increased regularly throughout the world. It includes participants of many ages, from 8 to 81 years,[12,15] the majority of whom are male. In 2000, out of 25,000 members of the American National Off-Road Bicycle Association, 89% were men and 11% were women.[16]

During the past 10 years, the increased popularity of this sport throughout the world has also revealed an exponential rise of minor and serious injuries due to its practice.[12-14] In accordance with the American National Athletic Injury Reporting System, the severity of injuries is divided into 3 different grades.[12] Skin wounds and contusions are classified as injuries of minimal severity; muscle strains and sprains, of minor severity. Severe injuries include fractures, ligament tears, joint dislocations, and cerebral concussions. Depending on the material and methods of studies, the classification of the severity of injuries is extremely different. In a study carried out among subscribers of a German-language mountain bike magazine, the written survey revealed that 90% of the respondents had 1 to 3 injuries on participat-

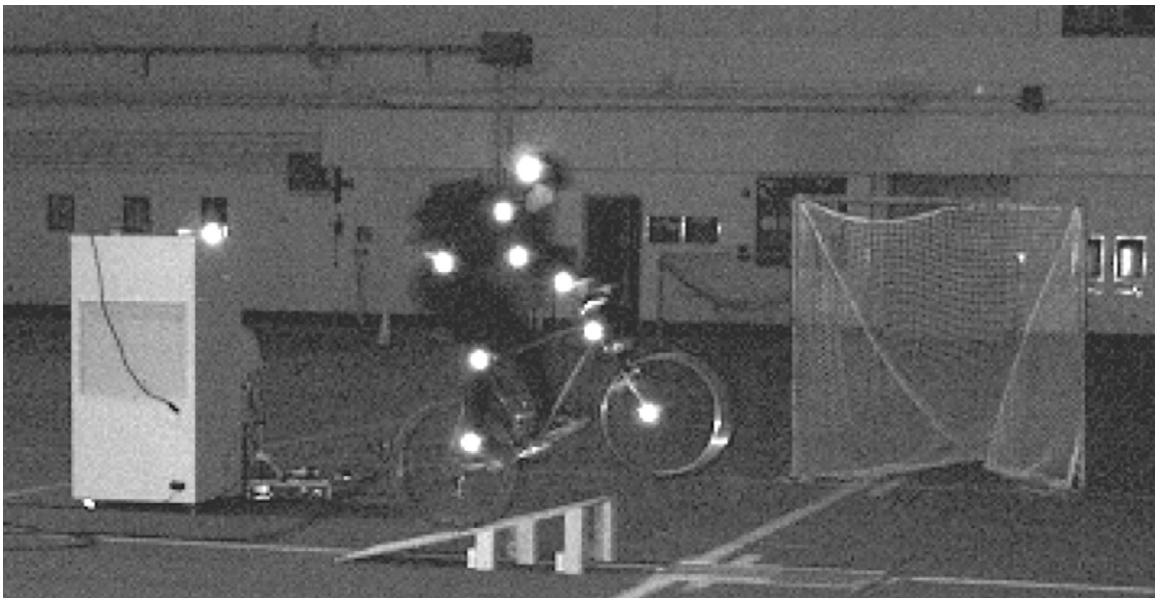

Fig 1. Bicycle and rider marker locations during test run. (Reprinted[17] with permission of the Instrumentation, Systems, and Automation Society. Copyright© 2004 ISA. All rights reserved.)

ing in their sport for an average of 3.7 years. Ninety percent of these injuries were of minimal or minor severity. The other 10% (the severe injuries) involved the shoulders, hands, and fingers, as well as the head.[12] In a recent multicenter study of the trauma centers of the Vancouver area, the data showed an exponential rise in severe injuries during the past 10 years, with a high proportion of head injuries, representing 12% of the nonorthopedic injuries.[14] The most frequent clinical presentation was a loss of consciousness (concussion), but bleeding or contusion of cerebral tissue also occurred.

In our 4 patients, the otoneurologic symptoms of BPPV occurred several hours after cross-country or downhill mountain bike training. In such situations, the cyclist rides at a high speed, doing multiple turns and jumps on rugged terrain, generating many shocks and vibratory impacts that are only partially filtered through the suspension fork and the upper parts of the body.

Biomechanically, during a moderate jump maneuver of 15 inches (about 38 cm) in height, the vertical accelerations that are measured at the beginning of the jump and on landing vary as a function of locations of the markers on the rider and the bike (Fig 1[17]). During jumping, all markers on the rider (head, shoulder, hip) and on the bike (head tube, front axle, seat) show an acceleration near a negative 1*g* until landing. During impact, the front axle of the bike records an upward acceleration of nearly 4*g*. Acceleration reaches about 2.6*g* on the shoulder markers and more than 2*g* on the head markers (Fig 2[17]).

Vestibular organs and, more specifically, the utricle and saccule as linear accelerometers of the head directly encounter such acceleration-deceleration movements. According to the biomechanical measurements of the vertical accelerations of the head during jump and impact, it is conceivable that during intensive mountain bike training, such repeated acceleration-deceleration events might generate displacement and/or dislocation of otoconia from the utricle. In case of dislocation, the otoconia might be dispatched either into the posterior, horizontal,

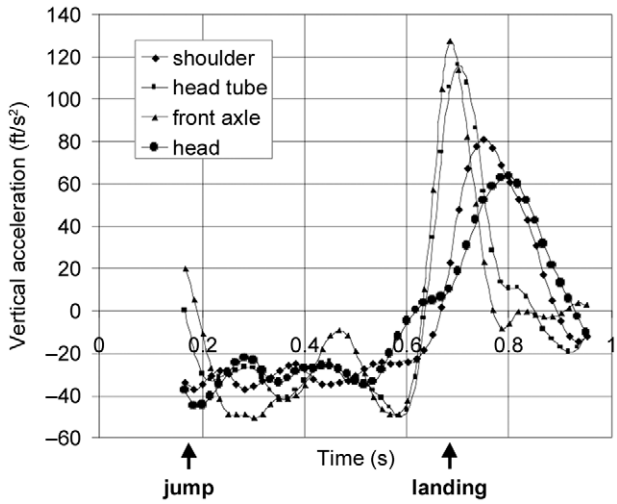

Fig 2. Vertical accelerations measured by key markers during jump. Graphic shows vertical accelerations measured by key markers located on bicycle and on rider between beginning of jump and landing. Triangles and squares represent vertical accelerations, respectively, of front axle and head tube markers on bicycle. Diamonds and circles represent vertical accelerations, respectively, of shoulder and head markers of rider. 1*g* = 29.9 ft/s^2 or 9.1 m/s^2. (Data from Redfield et al.[17])

or both semicircular canals, with the consequently typical symptoms of BPPV. The floating sensations described by patient 3, as well as the nearly sitting position on sleeping of patient 1, could be explained either by displacement of otoconia on the otolithic macula or by a diffuse transient disorder of the sensorineural cells of the utricle due to the repeated acceleration-deceleration events. The pathophysiological mechanisms that underlie the symptoms might be comparable to those in cases of otolithic dysfunction after whiplash injuries.[7] Indeed, during the initial phase of whiplash, the head undergoes a horizontal translational displacement relative to the torso. Whiplash induces a protraction movement of the head in the case of frontal collision and a retraction movement in the case of rear-end collision. The forces of translation generated by the impact are recorded by the otolith organs. The "slipping" movement during the head translation due to the acceleration forces is hypothesized to induce otolith displacements or damage of the sensorineural cells. During downhill and cross-country biking, the component of head translation is probably reduced because of the contraction of the cervical musculature.

CONCLUSIONS

Intensive cross-country and downhill training by off-road biking is a new cause of posttraumatic BPPV without head trauma. Regarding the sensorineural disorder of the vestibular organs induced by such acceleration-deceleration events, we propose classification of this posttraumatic BPPV as an injury of minor severity in accordance with the American National Athletic Injury Reporting System.

Otolaryngological practitioners, as well as sport and trauma physicians, should be aware of this possible origin of posttraumatic vertigo in order to treat it quickly with physiotherapeutic maneuvers and to have objective findings in case of possible future insurance litigation.

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