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ORIGINAL ARTICLE

Benign paroxysmal positional vertigo in swimmers

Yüzücülerde benign paroksismal pozisyonel vertigo

Songül AKSOY, PhD.,[1] Levent SENNAROĞLU, M.D.[2]

Objectives: This study was designed to evaluate the relationship between swimming and benign paroxysmal positional vertigo (BPPV).

Patients and Methods: This prospective study was comprised of 30 subjects (15 females, 15 males, mean age 15.6 years; range 11 to 23 years) who had been swimming regularly for at least two years. Twenty subjects (9 females, 11 males; mean age 15.4 years; range 11 to 21 years) with no vertigo and ear complaints comprised the control group. The Dix-Hallpike maneuver was used in all the swimmers and controls to diagnose BPPV.

Results: Four swimmers (13%) exhibited characteristic findings of BPPV, being unilateral in three, and bilateral in one. Interestingly, no swimmer experienced vertigo during swimming. The characteristics of nystagmus were typical of posterior semicircular canal BPPV in all cases. None of the subjects in the control group exhibited symptoms or findings of BPPV. Patients with BPPV underwent the Epley maneuver for therapy. All were free of vertigo after one month. There was no relationship between swimming and BPPV with respect to the frequency and duration of swimming.

Conclusion: Swimming may be one of the etiological factors of BPPV. It appears that rapid head movements during swimming cause otoconia to be dislodged from the macula and enter the semicircular canals.

Key Words: Labyrinth diseases; swimming; vertigo/etiology/physiopathology.

Amaç: Bu çalışmada yüzme ile benign paroksismal pozisyonel vertigo (BPPV) arasındaki ilişki değerlendirildi.

Hastalar ve Yöntemler: Bu ileriye dönük çalışma, aktif olarak iki yıldan fazla yüzme sporu ile ilgilenen 30 bireyden (15 kadın, 15 erkek, ort. yaş 15.6; dağılım 11-23) oluşan bir yüzücü grubu ile yürütüldü. Kontrol grubu, herhangi bir kulak ve vertigo yakınması olmayan 20 bireyden (9 kadın, 11 erkek, ort. yaş 15.4; dağılım 11-21) oluşturuldu. Tüm yüzücülerde ve kontrol grubunda BPPV'yi açığa çıkarmak için Dix-Hallpike manevrası kullanıldı.

Bulgular: Dört yüzücüde (%13) BPPV'nin karakteristik özellikleri gözlendi; bunların üçünde tek taraflı, birinde iki taraflı tutulum vardı. İlginç bir şekilde, bu hastaların hiçbirinde yüzme sırasında vertigo oluşmamıştı. Tüm bireylerde nistagmus karakteristikleri tipik olarak posterior semisirküler kanal BPPV'sine uymaktaydı. Kontrol grubundaki hiçbir bireyde BPPV semptomlarına ya da bulgularına rastlanmadı. Dört yüzücüye BPPV tedavisi için Epley manevrası uygulandı ve bir ay sonra tüm yüzücülerde vertigo kayboldu. Yüzme ile BPPV arasında yüzme sıklığı ve süresi açısından herhangi bir ilişki saptanmadı.

Sonuç: Yüzme, BPPV için etyolojik faktörlerinden biri olabilir. Muhtemelen, yüzme sırasındaki ani baş hareketleri, otokonianın makuladaki yerinden ayrılıp, semisirküler kanala girmesine neden olmaktadır.

Anahtar Sözcükler: Labirent hastalıkları; yüzme; vertigo/etyoloji/fizyopatoloji.

◆ *Department of [2]Otolaryngology, and [1] Section of Audiology and Speech Pathology, Hacettepe University (Hacettepe Üniversitesi [2]Kulak Burun Boğaz Hastalıkları Anabilim Dalı, [1]Odyoloji ve Konuşma Bozuklukları Ünitesi), Ankara, Turkey.*

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◆ *Correspondence (İletişim adresi): Dr. Levent Sennaroğlu. 28. Cadde, Hardem Apt., 7/8, Çiğdem Mahallesi Karakusunlar, 06520 Ankara, Turkey. Tel: +90 312 - 284 55 88 Fax (Faks): +90 312 - 311 35 00 e-mail (e-posta): lsennar@tr.net*

One of the most important developments in the field of otolaryngology in the last ten years is the understanding of the pathogenesis and therapy of benign paroxysmal positional vertigo (BPPV). According to Hughes and Proctor,[1] Barany was the first to describe the symptomatology of this disorder in 1921. Later, Dix and Hallpike[2] introduced the term "benign positional paroxysmal vertigo" and described the maneuver and criteria necessary for its diagnosis.

The etiology of this disorder is still unknown in 50% of the cases.[3] In the remaining 50%, BPPV is preceded by head trauma, viral infections, long term immobilization, or ear operations. It was also found in four (6%) out of 63 patients after stapedectomy.[4]

Crawl swimming involves rapid head movements around the vertical axis of the body. These rapid head movements are not present in other forms of swimming (backstroke, breaststroke, and butterfly). We believe that this type of swimming may cause dislocation of the otoconia from the macula. Our literature search of English based scientific databases for BPPV in swimmers yielded negative results. Therefore, we decided to investigate the relationship of BPPV in swimmers.

PATIENTS AND METHODS

The study was carried out in 30 swimmers and 20 controls. There were 15 female and 15 male swimmers, within an age range of 11 to 23 years (mean age 15.6 years) (Table I).

Subjects having complaints of dizziness, vertigo or inner ear involvement were excluded from the study. None of the subjects had a history of head trauma or migraine. Twenty subjects (9 females, 11 males; mean age 15.4 years; range 11 to 21 years) with no otolaryngological or vestibular complaints comprised the control group.

The swimmers in the study group were evaluated for vertigo and given a questionnaire about the presence and characteristics of vertigo. Each subject was asked to fill out an evaluation form prior to the study. After an ENT examination, a complete audiological evaluation was made. Those with the findings of Meniere's disease (low frequency sensorineural hearing loss, a positive Metz recruitment test, and tolerance problem in uncomfortable loudness test) were excluded from the study. The Dix-Hallpike maneuver[5] was used for all patients and controls to diagnose BPPV. Patients who exhibited vertigo, torsional nystagmus (which reverses its direction upon return to sitting position) preceded by a latent period, and fatigability of the nystagmus were considered to have BPPV. Nystagmus was investigated by Frenzel glasses. All subjects had head shake and Romberg tests to check if there was unilateral vestibular weakness.

Patients with BPPV were given the Epley maneuver[5] for therapy, and their progress was followed every week for one month. In their control examination the Dix-Hallpike maneuver was repeated to see if vertigo and nystagmus still persisted.

The statistical analyses was carried out by "the significance test between two mean values".

RESULTS

Four of the swimmers (13%) exhibited characteristic findings of BPPV, being unilateral in three, and bilateral in one. The characteristics of nystagmus were typical of posterior semicircular canal BPPV in all the cases. The latency and duration of nystagmus are given in Table II. The latent period before the appearance of nystagmus and the duration of nystagmus were less than typical in BPPV patients. It was also interesting that no swimmer had experienced vertigo during swimming. None of the subjects in the control group exhibited symptoms or findings of BPPV, as revealed by the Dix-Hallpike maneuver. None of the subjects demonstrated nystagmus on head shake test, and the Romberg test was normal in all the subjects.

The frequency of swimming ranged from 2 to 7 days a week (mean 4.9 days), for a duration of 2 to 14 years (mean 6.1 years) (Table III). The mean daily swimming was 116 minutes (range 90 to 120 minutes). The patients with BPPV were swimming five days a week for six years. The mean length of daily swimming was 120 minutes. There were no statistically significant differences between the swimmers with and without BPPV in regard to the number of swimming days per week, duration of daily swimming, or the number of swimming years ($p>0.05$).

TABLE I
DEMOGRAPHIC FEATURES OF THE SUBJECTS

	Female		Male		Mean age
	n	%	n	%	
Swimmer	15	50	15	50	15.6
Control	9	45	11	55	15.4

TABLE II
CHARACTERISTICS OF NYSTAGMUS IN SUBJECTS WITH BPPV

Patients	Side	Latency (seconds)	Duration (seconds)
#1	Right	1	3
#2	Left	1	4
#3	Left	1	3
#4	Left	2	5
#4	Right	1	4

TABLE III
CHARACTERISTICS OF SWIMMING IN SUBJECTS WITH AND WITHOUT BPPV

	Swimming period		
	Daily (minutes)	Weekly (days)	Years
Swimmers with BPPV	120	5	6
Swimmers without BPPV	116	4.9	6.1
p	p>0.005	p>0.005	p>0.005

BPPV: Benign paroxysmal positional vertigo.

Initially, five swimmers demonstrated only vertigo during the Dix-Hallpike maneuver. Of these, three had unilateral, two had bilateral vertigo. None of these were found to have characteristic nystagmus. The duration of vertigo in these patients ranged from 1 to 2 seconds. However, in follow-up evaluations, none of them had vertigo during the Dix-Hallpike maneuver, so no treatment was considered in these patients.

Four patients with BPPV underwent Epley maneuvers weekly for a period of one month. None of them complained of vertigo and none demonstrated vertigo or nystagmus during follow-up Dix-Hallpike tests.

DISCUSSION

The most important finding of this study is that swimming is among the etiologic factors of BPPV. As pointed out by Baloh,[3] the etiology in almost half of the patients with BPPV is idiopathic. We believe that, by identification of other situations associated with BPPV, the percentage of idiopathic cases will decrease considerably.

There have been dramatic advances in our understanding of the pathophysiology of BPPV. Schuknecht[6] originally put forward the hypothesis of cupulolithiasis. He made this observation during histopathological investigation of temporal bones. Later, Hall et al.[7] suggested the canalolithiasis theory, where free-floating otoliths in the semicircular canals were implicated for this kind of vertigo. Today, this theory is more commonly accepted than that of cupulolithiasis. The main reason for this is that, with the cupulolithiasis theory, it is very difficult to explain the brief duration of nystagmus and vertigo during the Dix-Hallpike maneuver. Debris adhering to the cupula would cause the cupula to be deflected for a duration as long as the head remains in the provoking position. In addition, the cessation of vertigo after the Epley maneuver also suggests the canalolithiasis as a possible underlying mechanism.[8,9]

It is generally accepted that otoconia dislocated from the macula enter the long arm of the semicircular canals, particularly the posterior semicircular canal. What is the etiology of canalolithiasis? Head trauma, viral infections, stapedectomy, swimming, and long-term immobilization such as that after lumbar hernia operations may cause BPPV. They cause dislocation of otoconia from the macula. After dislocation, they may easily enter the posterior semicircular canal. It is very difficult for a single mechanism to cover the pathophysiology in all these etiologic factors. After head trauma, stapedectomy, or swimming, either large pieces of otoconia break away from the macula or they are released in great quantity to cause the symptoms of BPPV. Viral infections may also cause dislocation of a great quantity of otoconia from the macula. However, it is very difficult to link BPPV to long-term immobilization. The only explanation that we can think of is that during long-term immobilization otoconia may aggregate to form large pieces. When these large pieces enter the long arm of the semicircular canal they cause the characteristic symptoms. We think that a certain amount of otoconia must enter the semicircular canal in order to be symptomatic.

What may be the pathophysiological mechanism in crawl swimming? Swimming definitely does not cause head trauma, but during swimming, the head is rapidly turned from one side to the other. This is a strong head movement which, we think, may be responsible for the otoconia to be dislodged from the macula. Normally in this position (during swim-

ming) we do not expect the otoconia to enter the long arm of the semicircular canal. However, when the swimmers lie supine, they may enter the long arm of the posterior semicircular canal. The positive response of these patients to the Epley maneuver also supports the theory of canalolithiasis versus cupulolithiasis. It appears that the relationship of swimming with BPPV does not depend on the number of swimming days per week, duration of swimming within the day, and the number of years of swimming.

It is also important to differentiate nystagmus-positive subjects from patients with vertebral artery compression (VAC). First of all, the swimmers in our study group were adolescents and at this age VAC is expected to be rare. In addition, patients with VAC experience episodes of transient neurological symptoms and signs that are localized to the posterior circulation. None of the subjects in this study had such complaints.

It is difficult to comment on patients with vertigo unaccompanied by nystagmus during the Dix-Hallpike maneuver. It is our impression that this combination may also be a form of BPPV and after the Epley maneuver the patients reported relief from their symptoms. The reason for the symptoms in these patients may be the insufficient amount of otoconia entering the semicircular canals to produce characteristic symptoms. However, these patients may also need canalith repositioning maneuver to return to normal. The authors of the present study are aware that this finding contradicts the fact that vertigo originating from the inner ear must also produce nystagmus. Recently, Haynes et al.[10] mentioned this subgroup in their study and called this "subjective BPPV". They proposed two explanations for this: either the nystagmus showed fatigue with repeated testing until the patient came to their department or this patient group might represent a less noxious form of BPPV. Whatever the pathophysiologic explanation is, they also advocated therapeutic maneuvers for the treatment of BPPV.

In conclusion, crawl swimming may be one of the etiological factors of BPPV. It appears that rapid head movements cause otoconia to be dislodged from the macula, giving way to the semicircular canals. The relationship between swimming and BPPV does not depend on the frequency and duration of swimming. The posterior semicircular canal was involved in all the affected subjects and they all responded very well to the Epley maneuver.

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