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Sir,

A 62-year-old female presented to our emergency room with a three days history of positional vertigo on lying down, rising from and turning in the bed. Neurological examination was normal; there was no spontaneous or gaze-evoked nystagmus. In the right Dix-Hallpike test (DHT), upbeat nystagmus with torsional component was observed, with latency and habituation and the patient was diagnosed with benign paroxysmal positional vertigo (BPPV) of the right posterior semicircular canal (PC). The Epley maneuver was performed and the patient recovered completely. Four days later she again presented with positional vertigo. Again neurological examination was normal. This time in the left DHT, down beat nystagmus (DBN) with slight torsional component towards the right ear (Video 1) was observed, without latency, habituation or adaptation. The patient complained of severe vertigo and nausea. Upon returning to the sitting position a short burst of upbeat nystagmus was observed and the patient complained of severe vertigo and nausea. In the head hanging position DBN with same characteristics was observed. Right DHT was negative. Brain MRI, caloric testing, pure tone audiogram and brainstem auditory evoked potentials were normal. The patient was treated with a repositional maneuver by Yacovino, and after two maneuvers she recovered completely.

The patient was diagnosed with BPPV of the right anterior canal (AC). Positional down beat nystagmus is a rare clinical condition which, in ¾ of patients, implies pathology of the brainstem. [1] However, in the rest of the cases the diagnosis is AC BPPV is usually made. There are several problems in making this diagnosis. First one is to identify the side of the lesion. The direction of the torsional component of the nystagmus and the side that triggers

the vertigo and nystagmus are the key elements to the identification of the affected ear. The torsional component is a key in identifying the affected side, however in AC BPPV the torsional component is very subtle or can be absent. Purely downbeating nystagmus with a lack of torsional component has been previously reported and it is explained by anatomical orientation of the anterior canal. [2] The second one is which test to use to make a diagnosis. The DHT can provoke nystagmus when performed bilaterally, contralaterally and ipsilaterally to the affected AC.[3] The straight head-hanging maneuver is performed if DHT is negative and usually is enough to make the diagnosis. The third problem is determining the mechanism (canalolithiasis versus cupulolithiasis). In cupulolithiasis the debris becomes fixed to the cupula. As the orientation of the ampullary segment of the anterior canal is almost vertical, this could result in extensive contact between debris and the cupula thus facilitating cupulolithiasis as the AC BPPV mechanism. [1] Clinically cupulolithiasis is favored by the absence of adaptation and habituation.[4] Based on all of the above our patient had cupulolithiasis of the right AC, based on the absence of habituation, adaptation and right torsional component of the nystagmus; probably as a result of the right PC BPPV treatment four days earlier.

There are several treatment procedures for AC BPPV. Several non-controlled studies using reverse PC-BPPV methods as their basic methodology and two procedures using prolonged extreme backward head positioning have shown efficacy in treatment of AC-BPPV.[5] Our patient fully recovered after two maneuvers described by Yacovino and colleagues.

This case shows that absence of latency, habituation and adaptation in positional DBN does not necessarily imply CNS damage. The presence of concurrent or successive BPPV of other canals can indirectly support the diagnosis of AC BPPV.

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Videos

Video1. The patient is in left DHT, down beat nystagmus (DBN) with slight torsional component towards the right ear is seen, without latency or adaptation.