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Guest feature

Benign Positional Vertigo

by Robert Slater, M.D.

Benign Positional Vertigo is a common disorder, especially in the elderly. Unfortunately the diagnosis is frequently missed and patients may be subjected to unnecessary expensive neurologic or even invasive vascular studies. Much of the difficulty lies in the confusion of positional vertigo with "Benign Positional Vertigo", the former being a symptom with diverse etiologies and the latter a diagnosis suggesting benign peripheral labyrinthine pathology.

The characteristic symptom is that of intense, true vertigo with a rotational feeling that occurs with change in position. The symptoms will subside if the patient maintains the position, however most patients will not keep their head in a position that causes vertigo. Symptoms usually last for only seconds, tend to recur throughout the day associated with movement and also frequently remit, disappearing for months or years at a time.

The diagnosis can be made by examining the patient during Hallpike testing. This testing starts with the patient seated. The body is moved to the supine position and the head extended another 45 degrees and rotated 45 degrees to either side. It is best to start with the movement that is most likely to produce symptoms. Patients can often provide this information. If the patient is not sure which position causes symptoms each laboratory should use a consistent pattern of turning the head to a given side first to provide consistency between tests. Labeling the record correctly is essential. Our laboratory uses the abbreviations HHL and HHR for head hanging left and right respectively. The test is performed turning the head to one side and repeating the test if it is "positive" to determine if the nystagmus decreases with repeat testing. Recording should continue until the symptoms or nystagmus ceases or at least 30 seconds and recorded while the patient is returned to the upright position. (Symptoms may be worse on sitting up and the nystagmus may reverse direction.)

The testing can be done in the office with direct observation however a significant number of false negative studies will result because the nystagmus is usually suppressed if the patient's eyes are opened. This obstacle can be overcome by either using special lenses (Frenzel lenses) which allow the examiner to see the eyes but prevent the patient from fixating his eyes, or recording the nystagmus during ENG study allowing detection of motion of the eyes with the lids closed.

If the nystagmus is "classical" it will start after a several second delay, decay over 10 - 15 seconds and decrease with repeat testing. It must also beat toward the uppermost ear and the vertical component should be upward. The nystagmus should also have a strong rotary component with the direction counter clockwise with the right ear down and clockwise with the left ear down. This classical appearance is very rarely associated with central nervous system disease and is reassuring to the physician managing the patient. Nystagmus that varies from this pattern is "atypical" and can be caused by a wide variety of pathologies including benign peripheral disease.

The most accepted explanation for the classical response is that loose otoconia fall back and stimulate the posterior semicircular canal. Stimulation of the posterior canal produces excitation of the ipsilateral superior oblique and the contralateral inferior rectus. This will produce a slow component (in the direction opposite the fast) in agreement with the clinical findings. It should be noted however that the action of these muscles depends upon the position of the eye in the orbit and various degrees of horizontal, vertical and rotary components may be present. The vertical component however will be greatest in the uppermost eye and the recording technique should be adjusted accordingly. This again emphasizes the preference for two channel recording.

There are several pitfalls in examining these patients in the ENG lab that physicians should be aware of. The first is that the diagnosis may be completely missed if ENG data is used alone. This may result because of the rotational nature of the induced nystagmus which cannot be recorded by ENG unless there is a significant horizontal or vertical component. ENG labs using one channel bitemporal recording have an increased chance of missing the nystagmus if the vertical component is the only recordable movement. Overdiagnosis can also be a problem. A brief induced nystagmus may be recorded at the onset of the test. This nystagmus is physiologic and caused by the rotational acceleration of the head. Frequent eye blinks in nervous patients can also be a source of confusion. This problem is lessened considerably by the routine use of a vertical channel that shows eye blinks quite clearly.

It is also important for the test to be repeated to look for fatigue. The nystagmus velocity must be measured if there is any doubt since many patients will have the symp-

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toms and the nystagmus recur but the velocity decrease will provide the clue to the fact that the positional nystagmus is probably of the benign type.

Patients with typical classical benign positional vertigo can most likely be safely followed with a minimum of additional testing. Patients with atypical features may also prove to have benign peripheral disease but further study to rule out progressive central nervous system or peripheral labyrinthine pathology is indicated. Atypical features include nystagmus beating toward the lower most ear, nystagmus which fails to fatigue on repeat testing, rotary nystagmus in the direction opposite to that expected, down beating nystagmus, and nystagmus that begins without a latent period of a few seconds.

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T.N.O.G. Delaware County Medical Center, Broomall, Pa. 19008 215-353-4183

Robert Slater is Chief of the Neurology Division of the Neuro-Otology Group in Broomall, Pennsylvania.
