How to manage a dizzy patient?

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Relevant vestibular physiology

Orientation of our body in space is the primary function of the vestibular system. This is achieved by the integration of signals from vestibular, visual and proprioceptive receptors at the level of brain stem. The information regarding the movement of head relative to the body is largely provided by the paired vestibular sensory endorgans (Fig. 1). The semicircular canals (SCC's) detect angular acceleration and the otolithic organs linear acceleration (i.e. gravity, deceleration in a car). Within the ampulated end of the three paired SCC's (superior, posterior and lateral SCC's) lie the endorgans of cristae. These contain specialized hair cells which transduce mechanical shearing forces into neural impulses. The cilia of these hair cells extend into a gelatinous matrix called cupula (Fig. 2).
The otolithic organs of utricle and the saccule are found in the vestibule. Their cilia project into a gelatinous matrix which contains blanket of calcium carbonate crystals better known as the otolithic macula (Fig. 3).
The impulses from the receptors relay information along the superior and inferior vestibular nerve which in turn synapses with second order neurons in the central vestibular nuclei to form three tracts namely the vestibulo-ocular reflex (VOR), the vestibulospinal tracts (VST) and the vestibulocerebellar tracts (VCT). Of all these, the VOR is the fastest and the most important for initiating a corrective reflex. The VOR is required to maintain a stable image on the retina following head movement. As the head moves in one direction there should be an equal but opposite conjugate movement of the eyes (the doll's eye maneuver). When the VOR is affected bilaterally (i.e. as a result of systemic aminoglycoside toxicity) the patient will complain of blurring of vision with head movement better known as oscillopsia. Should a unilateral defect in the VOR happen acutely then the eyes will be driven slowly towards the side of lesion by the greater vestibular tone of the unaffected side requiring repetitive central corrective saccades better known as nystagmus. Nystagmus therefore is the cardinal sign of both central and peripheral vestibular dysfunction.

History steps

It is possible to diagnose over 90% of dizzy patients by careful history alone[2]. You can use our simplified 4 x 4 method (Fig. 4).
First Step: Allow the patient to describe their symptoms in their own words. Patients with an organic cause for their dizziness tend to give a very crisp description of their spells. Conversely patients with non-organic causes usually have a very vague and prolonged description of their symptoms. There are other points in the history which can help with this distinction[3] (Table 1).
Second step: We should try to distinguish those patients who have true vertigo (i.e. a hallucination of motion relative to oneself) from those with nonspecific dizziness (i.e. lightheadedness, giddiness, floating etc). Patients with non specific dizziness especially those with history of loss of consciousness should be further investigated to exclude cardiovascular, neurologic and iatrogenic causes. Those with true vertigo have a vestibular cause which can be peripheral (localized to the inner ear) or central (localized to the central nervous system).

Third step: We should be able to separate those patients with a peripheral vestibular dysfunction from those with a central cause. In the history it is important to look for associated inner ear (peripheral) symptoms such as loss of hearing, tinnitus and aural fullness. Patients with a central vestibular loss usually have evidence of focal neurological dysfunction such as diplopia, dysphagia, dysarthria, paresis, parasthesia or incontinence.
Fourth step: Patients with peripheral vestibular dysfunction fall into one of four main categories. To help distinguish these categories the most important factor tends to be the duration of the dizzy attacks[3]. Other important distinguishing factors include associated inner ear symptoms such as hearing loss, tinnitus etc.
Benign paroxysmal positional vertigo (BPPV) is the most common inner ear disorder. The attacks in BPPV typically last for few seconds at a time and are associated with certain head movements. It is most commonly provoked by turning in bed to the affected side or by suddenly extending the neck to look up. There is usually more than one attack of dizziness in a 24 hour period. Patients with BPPV do not usually have other symptoms of inner ear dysfunction such as hearing loss, tinnitus or aural fullness. BPPV has been demonstrated to be primarily due to free floating particles (presumed otoconia) in the semicircular canals (canalolithiasis) or from those particles attached to the cupula of the canal (cupulolithiasis) [1]. The vast majority of these particles are found in the posterior canal being the most gravity dependent part of the labyrinth. BPPV can be either primary or occur secondary to head trauma, vestibular neuronitis, Meniere's disease or following inner ear surgery[1].
Dizziness in Meniere's disease is usually associated with fluctuant hearing loss, tinnitus and a sense of aural fullness. The episodes of dizziness last for minutes to hours. It would be very unusual to have more than one attack in a 24 hour period.
Dizzy attacks similar to Meniere's disease without cochlear symptomatology are described as being compatible with a recurrent vestibulopathy (RV).
Patients with vestibular neuronitis (VN) have attacks which last for days to weeks with no cochlear symptoms.

Otoneurological Examination steps

To more precisely identify the localization for an underlying vestibular disorder a formal otoneurological examination is required. There are four steps in this examination (Fig. 5).
First step: Perform an otological examination which includes otoscopy looking for treatable causes of vertigo such as middle ear pathology (e.g. cholesteatoma). The fistula test should be performed by pressing in the tragus repeatedly or by pneumatic otoscopy. Using a 512-hz tuning fork perform Weber and Rinne tests which should give you a general idea about the patient's hearing.
Second step: Do a neurological examination. This should include examination of all the cranial nerves, cerebellar tests, oculomotor tests and general balance tests.
Cranial nerves and cerebellar examinations are needed to exclude a cerebellopontine angle lesion such as an acoustic neuroma. Oculomotor testing should look at smooth pursuit, saccades, visual fixation and vergence (convergence and accommodation) eye movements. If smooth pursuit is normal then it is unlikely a central disorder will be present[4]. In peripheral vestibular dysfunction the nystagmus has clear slow and fast (saw tooth) phases. While in central and congenital forms nystagmus can have complex wave patterns such as pendular, exponentially increasing and decreasing nystagmus etc

General balance tests should include proprioception, the Romberg's, the Unterberger's stepping test and tandem gait tests (eyes open and closed).
Third step: Perform special tests of vestibular function which should include the Halmagyi high frequency head thrust maneuver, the head shake test, the oscillopsia test and the VOR suppression (VORS) test. All of which can be easily performed in a family practice setting.

The **Halmagyi manoeuvre** consists of rapid, passive head movements from each side to the midline while the patient fixates on a central object such as examiner's nose. In normal rapid head movements there should be an equal but opposite conjugate movement of the eyes (in a normal individual the gain (i.e. head movement / corresponding eye movement) should be unity). If there is a defect in the VOR then the eyes lag behind the head movement and several corrective saccades will be required to keep focus on the examiner's nose. These saccades will be evident to the examiner[5].
The **head shake test** is performed by asking the patient to close his or her eyes and then passively shaking the patient's head in a horizontal plane for 20 seconds. The presence of post head shake nystagmus when the patient opens his / her eyes is suggestive for a peripheral vestibular dysfunction. The direction of nystagmus is usually away from the affected side but not always[6]. The presence of vertical or rotatory nystagmus following horizontal head shaking is called "cross coupling" and is suggestive of a CNS disorder.
The **oscillopsia test** is easily performed by asking the patient to read the lowest line they can at rest on a Snellen chart. The patient's head is then shaken from side to side and the patient is asked to read the lowest line they can during the head shaking. Missing more than five lines on the chart is generally indicative of a bilateral vestibular loss[7].
The **VOR suppression** test is best performed by having the patient sit on a rotating chair while fixating on a pen held in their outstretched arms. By rotating the chair while the patient is looking at the pen, the examiner observes the patients eyes. Normally one should be able to fixate and suppress signals from the VOR. However in the case of CNS pathology the identification of the breakthrough nystagmus from the VOR will be visible to the examiner as the patient is unable to fixate their eyes while the chair is rotating[7].
Fourth: The final step in the examination is to do few additional diagnostic tests. Perform the Dix-Hallpike's manoeuvre. Have the patient sit on the bed with the head turned to the either left or right. While looking at the patients eyes for nystagmus place the patient's head down in the supine head hanging position (Fig. 6). The head should be held in this position at least for 10 seconds before any nystagmus would be expected to occur in classic posterior canal BPPV (There is usually a latency of 1-5 seconds). A torsional nystagmus with the fast phase beating towards the affected (dependent) ear should be observed. The direction of nystagmus reverses as the patient sits up. If classic posterior canal BPPV is detected then a particle repositioning manoeuvre (PRM) should be performed. This is done by sitting the patient on a bed and moving to the Dix-Hallpike's position on the affected side. The patient should be kept in this position until the vertigo and nystagmus stops. The patient is then rolled onto the opposite lateral side with the head held in extension. The patient is kept in this position for 30-60 seconds and then asked to sit up. After a PRM, patients are asked to sleep in the semi sitting position for the next two nights. The success rate of the PRM has been reported as being between 30 to 100 percent[1].
Lastly ask the patient to hyperventilate for about one minute. If this reproduces the patient's dizzy symptoms then it is certainly probable that the cause for their dizziness is psychogenic (I.e. anxiety related)[2].

Conclusion

The dizzy patient can be easily handled by the family physician with this simple method. Remembering the four steps involved in the history and the physical examination, most patients can be readily diagnosed. We believe this approach will also prevent any serious pathology being missed and should help family physicians in directing their investigations and referrals more accurately. The four by four step method is comprehensive yet simple. It can be used by all physicians to deal with different forms of dizziness.

References

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