One such example is Mr Liao, a 61-year-old Chinese gentleman who was admitted to the TTSH ICU following a motorcycle accident that left him with arm fractures, as well as spine and spleen injuries. He spent 40 days in the ICU and needed the support of a ventilator. With the early mobilisation programme, he was able to get out of bed to sit up in a chair while in the ICU. As his strength gradually returned, he was able to walk assisted even when he was on ventilator support. Even with the fluctuations in his medical condition, which is very much expected in an ICU environment, he was able to walk with the help of a walking aid when he was being transferred to a normal ward after his long ICU stay. This may not have been possible without the early mobilisation programme. In an interview with the Chinese local newspaper *Zaobao*, Mr Liao said, "I was very worried about my mobility after the accident but after receiving early physiotherapy in the ICU, it has enabled me to move! I feel better and am slowly regaining my muscle strength and coordination to go back to walking and doing the things I love!".

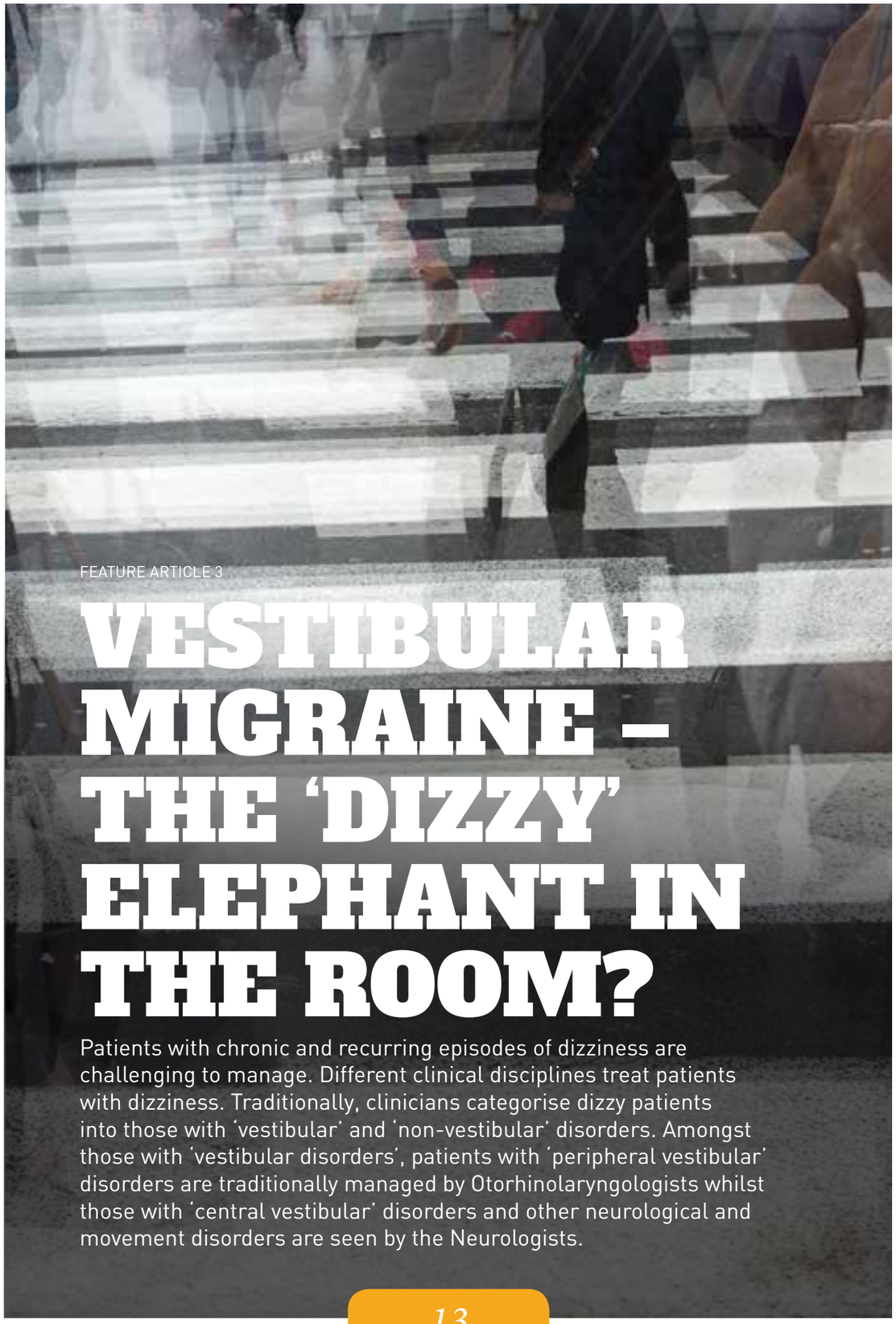
There are some challenges in attempting early mobilisation. For example, there will be days when the patient has to rest in bed due to medical reasons. When this happens, the mobilisation exercises will have to be re-started. Overall however, early mobilisation has been shown to confer expressed benefits towards patients within the ICU setting. With the effective implementation of these protocols with strong interdisciplinary teamwork, we can expect to see enhanced critical care and improved patient outcomes.

"I was very worried about my mobility after the accident but after receiving early physiotherapy in the ICU, it has enabled me to move! I feel better and am slowly regaining my muscle strength and coordination to go back to walking and doing the things I love!".

FURTHER READING

1. *Adler J, Malone D. Early mobilization in the intensive care unit: a systematic review. Cardiopulmonary physical therapy journal. 2012;23(1):5-13.*
2. *Bailey P, Thomsen GE, Spuhler VJ, Blair R, Jewkes J, Bezdjian L, et al. Early activity is feasible and safe in respiratory failure patients. Crit Care Med. 2007;35(1):139-45.*
3. *Hanekom S, Gosselink R, Dean E, van Aswegen H, Roos R, Ambrosino N, et al. The development of a clinical management algorithm for early physical activity and mobilization of critically ill patients: synthesis of evidence and expert opinion and its translation into practice. Clinical rehabilitation. 2011;25(9):771-87.*
4. *Morris PE, Goad A, Thompson C, Taylor K, Harry B, Passmore L, et al. Early intensive care unit mobility therapy in the treatment of acute respiratory failure. Crit Care Med. 2008;36(8):2238-43.*
5. *Needham DM. Mobilizing patients in the intensive care unit: improving neuromuscular weakness and physical function. Jama. 2008;300(14):1685-90.*
6. *Needham DM, Korupolu R, Zanni JM, Pradhan P, Colantuoni E, Palmer JB, et al. Early physical medicine and rehabilitation for patients with acute respiratory failure: a quality improvement project. Archives of physical medicine and rehabilitation. 2010;91(4):536-42.*
7. *Schweickert WD, Pohlman MC, Pohlman AS, Nigos C, Pawlik AJ, Esbrook CL, et al. Early physical and occupational therapy in mechanically ventilated, critically ill patients: a randomised controlled trial. Lancet (London, England). 2009;373(9678):1874-82.*
8. *Thomsen GE, Snow GL, Rodriguez L, Hopkins RO. Patients with respiratory failure increase ambulation after transfer to an intensive care unit where early activity is a priority. Crit Care Med. 2008;36(4):1119-24.*

MR JAYACHANDRAN BALACHANDRAN
is a principal physiotherapist in the Department of Physiotherapy, Tan Tock Seng Hospital.



FEATURE ARTICLE 3

VESTIBULAR MIGRAINE – THE 'DIZZY' ELEPHANT IN THE ROOM?

Patients with chronic and recurring episodes of dizziness are challenging to manage. Different clinical disciplines treat patients with dizziness. Traditionally, clinicians categorise dizzy patients into those with 'vestibular' and 'non-vestibular' disorders. Amongst those with 'vestibular disorders', patients with 'peripheral vestibular' disorders are traditionally managed by Otorhinolaryngologists whilst those with 'central vestibular' disorders and other neurological and movement disorders are seen by the Neurologists.

For the 'non-vestibular' group of patients, physicians from various specialties like Internal Medicine, Cardiology and Geriatric Medicine are involved if the underlying diagnosis is syncope or pre-syncope (iatrogenic, vaso-vagal, cardiac arrhythmias, etc.) or general frailty from multiple medical problems. This list is far from exhaustive. For example, the Endocrinologist may be involved if there are diabetic patients with peripheral or autonomic neuropathy or the Ophthalmologist for patients with visual impairment.

Role of our balance system

Our balance system is vital to our orientation and survival within earth's environment that has a gravitational field. It allows us to maintain an upright posture and facilitates bipedal gait. Our balance system also stabilises our visual perception for us to interact with our environment, allowing us to feel steady despite the repetitive and fast natural movements of our head or movements in our surroundings. Our balance system, like our breathing and heart contraction, works automatically and is taken for granted until it malfunctions.

Anatomy and physiology of balance

To understand balance disorders, it is useful to remind ourselves of the normal anatomy and physiology of our balance system. There are three components in our balance system; the afferent component, the central nervous system (CNS) and the efferent component. Our balance **afferent component** has three main sensory inputs: vestibular, from the three semi-circular canals and the two otolithic organs located within each vestibule of our inner ear; visual from our eyes; and somatosensory from our proprioceptive and sensory receptors within our lower limbs, neck and other joints. The **CNS component** including the brain stem and cerebellum helps integrate and modulate the balance sensory inputs. Two vital reflexes work with the efferent balance components to complete the system. The vestibular-ocular-reflex has the six extra-ocular muscles as its **efferent component** and this important reflex helps us maintain visual stability. The vestibular-spinal-reflex has the anti-gravity and peripheral muscles as its **efferent component** and is vital for us to maintain postural and gait stability.

For both reflexes, the vestibule provides the afferent signals and hence any dysfunction with this organ

The pattern of the vertigo will help distinguish the different conditions. For BPPV, the vertigo is positionally induced, has a latency, lasts less than a minute, is fatigueable and is recurrent as long as the disease is active.

gives rise to specific dizziness symptoms. At rest, in a normal person, there is no net difference between both vestibules and hence, there should be no involuntary eye movement. Any acute loss (common) or over-activity (less common) of one vestibule can lead to a unilateral afferent vestibular hypofunction or hyperfunction that results in the acute asymmetry of the vestibular-ocular-reflex. This results in the 'temporary' involuntary repetitive movement or deviation in the patients' eyes, clinically observed as 'jerky nystagmus' (clinical sign elicited by clinicians) and perceived as vertigo; defined as the 'perception or hallucination of movement where there is none' (symptom as described by patients). Vertigo as per its strict definition has been the cardinal symptom in the management of peripheral vestibular disorders by Otorhinolaryngologists.

Jerky nystagmus has a slow and fast phase and with the direction of the fast phase conventionally defined as the direction for the nystagmus. The clinical finding of a jerky nystagmus is a hallmark of an acute peripheral vestibular asymmetry and follows Alexander's laws. The direction of the nystagmus should be away from the hypofunctioning side and the intensity of the nystagmus should also be

accentuated (with no change in nystagmus direction) when gaze is directed away from the hypofunctioning side. And, the intensity of the nystagmus should be increased when visual fixation is removed with the use of very high dioptic glasses. In contrast, central type nystagmus tends to be pendular (i.e. no fast and slow phase) and direction-changing depending on gaze direction. Patients with central type nystagmus frequently do not experience concurrent vertigo.

Peripheral vestibular disorders

The 'big' three diagnoses for the Otorhinolaryngologists are benign paroxysmal positional vertigo (BPPV), Meniere's disease, and acute vestibular hypofunction (from vestibular neuronitis, labyrinthitis or a thromboembolic event within the inner ear). In all three conditions, patients report vertigo as a symptom and jerky nystagmus should be elicited during clinical examination, when patients are symptomatic. Hearing loss and tinnitus will be present if the cochlear is involved (e.g. labyrinthitis) and for Meniere's disease, the presence of aural fullness will complete the clinical picture.

The pattern of the vertigo will help distinguish the different conditions. For BPPV, the vertigo is positionally induced, has a latency, lasts less than a minute, is fatigueable and is recurrent as long as the disease is active. For Meniere's disease, the vertigo usually lasts minutes to several hours, but can be recurrent during the active phase. Patients with either of these two conditions can get recurrent vertigo episodes but they tend to be fairly asymptomatic in between the active periods.

However, for acute vestibular hypofunction, the vertigo is usually non-recurrent. The vertigo is often severe, with associated nausea and vomiting that lasts up to a day but rarely beyond. However, the patient can remain dizzy for days to weeks afterwards, pending central compensation for the asymmetrical vestibular input. They are rarely vertiginous beyond the first 24 hours, but suffer from dizziness in the form of imbalance and disequilibrium, head-motion-provoked dizziness and visual-provoked dizziness i.e. induced by excessive movements of their surroundings or being in a visually complex or stimulating environment (such as when watching action movies, or walking in a visually stimulating environment like a supermarket aisle or corridors and passages with repetitive patterns or lines).

Most of these symptoms often diminish once the patients centrally compensate for the acute vestibular hypofunction. For young patients, this tends to happen within days to weeks so long as they are not on a prolonged course of vestibular sedatives like prochlorperazine or cinnarizine that hamper the brain's natural balance compensatory mechanism. Older patients who are slow to compensate centrally and those who have developed an overdependence on their visual inputs for their balance will benefit from vestibular rehabilitation exercises. These are provided by specially trained physiotherapists who are able to provide 'substitution' and 'adaptation' type exercises to compensate for the 'lost' vestibular function and also 'habituation' type exercises for head motion and visual sensitivity.

There are rarer conditions that affect the inner ear, e.g. superior semi-circular canal dehiscence and auto-immune inner ear disease or AIED, that result in recurrent vertigo attacks in patients, but often with other otological symptoms. Patients with AIED usually have symptoms in both ears. It is unwise and unsafe to follow the outdated adage that *'it is not a vestibular problem if there is no vertigo'*. Patients with bilateral vestibular hypofunction do not experience vertigo if there is no asymmetry in their peripheral vestibular function. Instead, they are highly intolerant to any head movement and they actually perceive their entire surroundings bouncing up and down each time they try to walk. This is termed ossilopsia and can be highly disabling. One of the commonest causes of bilateral vestibular hypofunction is ototoxicity due to aminoglycoside antibiotics and platinum chemotherapy agents.

Additionally not all patients with unilateral vestibular hypofunction experience vertigo. As mentioned earlier, most patients with acute vestibular hypofunction experience dizziness symptoms rather

than vertigo beyond the first 24 hours. Another example is the patient with chronic progressive vestibular hypofunction. Patients with vestibular schwannoma will often experience slow, progressive loss of vestibular function unilaterally. The slow pace of vestibular decline allows progressive central compensation and hence most of these patients have neither vertigo nor imbalance.

The Multi-Disciplinary Balance Clinic at Tan Tock Seng Hospital

There is no substitution for a comprehensive history and examination to arrive at the correct diagnosis. The Tan Tock Seng Hospital Multi-Disciplinary Balance Clinic (MBC) was started in the first half of 2012 to manage patients with complex balance and dizziness symptoms. Patients in this clinic are seen concurrently by an Otorhinolaryngologist, Vestibular Therapist and Audiologist. The focus of this clinic is to offer an accurate diagnosis followed by a comprehensive, holistic, personalised and actionable treatment plan. There is also an ongoing engagement with cross-referrals to other clinical teams for example, the Neurologists, the Psychologists and the 'Falls Clinic' which is run by Geriatricians. Over time we will develop an 'Interdisciplinary Balance Network' to better manage patients with complex dizziness disorders.

The 'chronic vestibulopathy' patients

Within the Multi-Disciplinary Balance Clinic, it soon became clear that there is a large cohort of patients who do not fit nicely into any of the common or rarer peripheral vestibular dysfunction diagnoses. Many of these patients have had chronic recurrent dizziness episodes over many years or even decades. Some of these patients needed multiple in-patient admissions with each stay lasting longer than two nights. Many more had needed multiple trips to the Emergency Department or Family Physicians. These recurrent attacks of dizziness can severely impact the patients' quality of life. Many had to give up all forms of exercise and some have lost the confidence to leave the confines of their homes. A few patients have had to give up working or struggle in their primary role as a parent or caregiver. Some have developed secondary anxiety and depression from their unremitting symptoms.

Many of these patients had acquired diagnoses like 'chronic vestibulopathy', 'chronic vertigo', 'non-specific dizziness' and 'multi-factorial dizziness' prior to being seen in the MBC. These diagnoses are deeply unsatisfying and unhelpful to both physicians and patients. Whilst they are descriptive of the patients' symptoms, they do not provide any aetiological nor pathophysiological explanation into their problems. Some patients are put onto long-term vestibular sedatives like cinnarizine which often make things worse.

Many more are on long-term betahistine which is a histamine agonist with variable results. A large proportion of patients are also referred to the Vestibular Therapists. Whilst they may be able to improve patients' functional status without a firm clinical diagnosis, some symptoms like head-motion- and visual-provoked giddiness remain challenging to address. More importantly, without an accurate diagnosis, patients will not receive the relevant advice on the prevention and management of future episodes of dizziness. Many patients state that the unpredictability and the lack of understanding of their diagnosis or the lack of diagnosis, for that matter, can result in loss of confidence, anxiety and excessive worrying. This impacts their quality of life massively.

The elephant in the room

One should be alert to the possible diagnosis of vestibular migraine for patients where no firm balance diagnosis can be made especially after multiple encounters with various health care professionals. Vestibular migraine has maintained a rather enigmatic status despite the many published papers on this condition. Vestibular migraine is a diagnosis given to a subset of patients with migraine who suffer from dizziness which is aetiologically related to their migraine. The publication of a Consensus Diagnostic Criteria by the Barany Society and International Headache Society in 2012 has helped to provide clarity in the diagnosis and management of this condition. In fact, there has been a multitude of terminology used for this diagnosis previously including 'migraine-associated dizziness' or MAD and 'migrainous vertigo' or MV. One wonders if the choice of the former acronym reflects scepticism on the part of the clinicians.

Clinical history

Patients with vestibular migraine suffer from recurrent episodes of dizziness that last between 5 minutes to 72 hours. These episodes of dizziness include vertigo which may be spontaneous, brought on by head motion or head position changes, or induced by visual stimulus. Some patients with non-vertiginous dizziness induced by head motion are described as having 'disturbed spatial orientation'. These symptoms are usually severe enough to interfere with or stop daily activities. However, to make this diagnosis, patients also need to have either a current or previous history of migraine with or without aura. Also, at least half of the dizziness episodes should be associated with at least one of the following: migraine-pattern headaches, photophobia and phonophobia, or visual aura. Last but not least, other dizziness diagnoses must be excluded. A diagnosis of probable vestibular migraine can also be made if the spectrum of symptoms is less than complete.

From my personal experience, the commonest symptoms amongst patients with vestibular migraine are head-motion-provoked and visual-provoked dizziness which may or may not be vertiginous. A strong sense of nausea or vomiting are both common. These are often brought about by any activity that involves head movement including something as benign as running or rushing about. Patients are very reluctant to move their heads and avoid bright, crowded and noisy places during the attacks. Like traditional migraine patients, they will seek a dark, quiet place to rest during attacks. Most patients do not get concurrent headaches although they may get headaches or head heaviness in between the dizziness attacks. A smaller number of patients experience recurrent dizziness attacks without any in-between headaches.

However, for this group of patients who are usually older, a careful history will elicit a previous history of recurrent headaches, with or without dizziness. These headaches could have started as early as their primary school days but with resolution or reduction in their 40s and 50s. This is something for them to celebrate. Unfortunately, after a period of minimal symptoms, they start to suffer from recurring episodes of dizziness. Some patients do complain of head heaviness, which can be at the front, top of head, back of head and sometimes all over the head, rather than the classical unilateral throbbing pattern which they are likely to have suffered when younger.

Many patients also suffer from motion sickness, which can precede their headaches and dizziness for many years. They report difficulty in reading on the bus, going up spiral carpark ramps, or riding

roller coasters when young. There is frequently a strong family history of recurrent headaches and/or dizziness.

Another useful aspect of the history to explore is the relationship between migraine triggers and dizziness attacks. The commonest lifestyle triggers for vestibular migraine are tiredness and lack of sleep, stress, negative emotion like anxiety, worry or depression. Unfortunately, these lifestyle triggers are often inter-related and many patients enter a vicious cycle, culminating in clinical depression and anxiety. Dietary triggers like monosodium glutamate, salt, caffeine, chocolate and alcohol are frequently reported as well. Two other important triggers are dehydration and peri-menstrual periods.

Examination findings

Patients with vestibular migraine do not have spontaneous or gaze-evoked nystagmus. A complete examination includes checking the ears for any acute or chronic ear infective or inflammatory condition. Central examination includes checking for smooth pursuit and saccadic eye movements and cerebellar function. It is vital to exclude vestibular hypofunction as some of the clinical signs may actually overlap. For example, the Halmagyi Head Thrust can sometimes be positive as some patients find it extremely difficult to maintain their gaze. However, interestingly, some vestibular migraine patients are found to have this test positive bilaterally. The differential diagnosis then is bilateral vestibular hypofunction.

For the Dynamic Visual Acuity or DVA test, both vestibular migraine and vestibular hypofunction patients will have a drop of four lines or more. There are three tests that can help differentiate between these two conditions. Post-head-shake nystagmus tends to be positive only in patients with vestibular hypofunction, even in those who had it in the past and have centrally compensated. The Foam Romberg's test is also useful. Patients with vestibular migraine tend to be able to maintain their balance even with their eyes shut. Patients with vestibular hypofunction will almost always fall down, frequently to their hypofunctioning side. The last test is the Unterberger's test where patients with true vestibular hypofunction tend to deviate that side during the test.

Investigations

Vestibular migraine is a clinical diagnosis and the role for investigations is limited. In fact, there is no single investigation that will confirm its diagnosis. However, investigations can be useful to rule out other diagnoses like vestibular hypofunction or Meniere's disease. For most patients, an audiogram to confirm normal hearing is helpful. A Video Head Impulse Test (VHIT) can help exclude vestibular hypofunction, whilst an MRI scan of the cerebello-pontine angle can exclude a vestibular schwannoma.

Vestibular migraine is a clinical diagnosis and the role for investigations is limited. In fact, there is no single investigation that will confirm its diagnosis. However, investigations can be useful to rule out other diagnoses like vestibular hypofunction or Meniere's disease.

Management

It has been immensely useful and rewarding to explain the diagnosis, its aetiology and natural history to most patients. Many of them are desperate for a good explanation for their chronic symptoms. It is often necessary to spend time explaining the lifestyle and dietary trigger factors. But not all patients are able to change longstanding behavioural and dietary habits. And it is certainly not fair to tell the insomniac to just try and sleep more when they have difficulties falling asleep or suffer from early wakening. Pharmacotherapy options should always be explored, and from my experience, more than 90% of patients are willing to try medication, especially if you assure them that the aim is to avoid long term medication.

Using asthma medication as an analogy is very helpful to the patients. They need to understand the two main types of medication which are the prophylaxis and relieving or abortive. For the latter, many patients who continue to get headaches will respond to either paracetamol or NSAIDs. Second-line medication like the triptans and caffeine plus ergotamine tend to be effective as well. Interestingly, most patients who get concurrent dizziness with their headaches also report relief of their dizziness with these abortive medications. However, their role in relieving dizziness that does not occur with headaches is less clear.

For migraine prophylaxis, there are five or six different classes of medication. There is no clear-cut 'most effective medication' on reviewing the literature.

There is a high variability in different centres, let alone different countries, in clinicians' first and second choices. If one is uncomfortable with using anti-convulsants like topiramate or sodium valproate, there are alternatives. The main three medications that I use as migraine prophylaxis are propanolol (a beta-blocker), nortriptyline or amitriptyline (tricyclic anti-depressant) and flunarizine (calcium channel blocker). It is vital to understand the side effects and contra-indications for these medications.

The commonest medication I use for younger patients is propranolol, after excluding the main contra-indication of asthma, symptomatic hypotension and impaired cardiac function. For older patients, low-dose nortriptyline and amitriptyline are my first choices. Side effects include daytime somnolence and mouth dryness. Contra-indications include bladder outlet obstruction and constipation. Flunarazine tends to work for patients who do not tolerate the other two medications. Side effects include insomnia and weight gain.

A large proportion of vestibular migraine patients have chronic insomnia. It is useful to observe that most of them experience a significant improvement in their sleep pattern after starting low-dose nortriptyline or amitriptyline. This often coincides with a significant improvement in their dizziness and/or headache symptoms. However, many complain about the daytime somnolence. Taking the medication 12 hours before their intended wake-up time seems to lessen this side effect. The dosage that maximises the positive effect and minimises side effects is highly variable between patients. Patients are advised to titrate their dosage between 5 and 30 mg to find their ideal balance. Most patients are on 5 or 10 mg of either medication daily.

Patients are often encouraged to take their prophylactic medication for 2 to 3 months initially to confirm the efficacy of the medication. Resolution or improvement in dizziness symptoms helps to confirm the diagnosis. Most patients are reluctant to be on long-term medication and hence, they are advised to stop the prophylaxis after they have addressed their lifestyle and dietary migraine trigger factors.

Many patients go through periods where their lifestyle and/or dietary factors are not conducive, resulting in a flare-up of their migraine symptoms. Fortunately, these are usually easy to predict. For students, these would be around examinations and for working people, around deadlines and other work surges. Many of them are willing to take their migraine prophylaxis medications only during these stressful periods.

There is a small group of patients with significant anxiety and/or depression due to their chronic and unremitting dizziness symptoms. Besides all the treatment modalities, they are also offered a consultation with the Clinical Psychologist. Many of these patients will fail to achieve full control of their migraine symptoms if their psychological well-being is not addressed. Many of these patients fall into the spectrum of persistent-postural-perceptual-dizziness or PPPD disorder that was originally described by Jeffrey Stabbs, a Psychiatrist from the Mayo Clinic.

Conclusion

Chronic recurrent dizziness is common and clinicians should try to avoid leaving patients to be stuck with the 'chronic vestibulopathy' or similar label without making efforts to further characterise the condition. Chronic recurrent dizziness can profoundly impact patients' quality of life. Vestibular migraine should be excluded in this group of patients. As with any other chronic disorders, a careful thorough history and examination is necessary for making the correct diagnosis. The symptoms of vestibular migraine can be successfully controlled in the vast majority of patients.

FURTHER READING

1. *Calhoun AH, Ford S, Pruitt AP, Fisher KG. The point prevalence of dizziness or vertigo in migraine--and factors that influence presentation. Headache. 2011;51(9):1388-92.*
2. *Lempert T, Olesen J, Furman J, Waterston J, Seemungal B, Carey J, et al. Vestibular migraine: diagnostic criteria. J Vestib Res. 2012;22(4):167-72.*
3. *Vukovic V, Plavec D, Galinovic I, Lovrencic-Huzjan A, Budisic M, Demarin V. Prevalence of vertigo, dizziness, and migrainous vertigo in patients with migraine. Headache. 2007;47(10):1427-35.*
4. *Eggers SD, Staab JP, Neff BA, Goulson AM, Carlson ML, Shepard NT. Investigation of the coherence of definite and probable vestibular migraine as distinct clinical entities. Otol Neurotol. 2011;32(7):1144-51.*
5. *Zhao JG, Piccirillo JF, Spitznagel EL, Jr., Kallogjeri D, Goebel JA. Predictive capability of historical data for diagnosis of dizziness. Otol Neurotol. 2011;32(2):284-90.*

DR HO EU CHIN
is a consultant in the Department of Otorhinolaryngology, Tan Tock Seng Hospital.